

CONTRIBUTORS TO VOLUME II.

ALLEN, RAYMOND B , M D , P H D (IN UROLOGY)

BARRINGER, BENJAMIN S , M D , F A C S

BELT, A ELMER, M D , F A C S

CABOT, HUGH, M D , C M G , F A C S

CAMPBELL, EDWARD W , B S , M D , F A C S

CAULK, JOHN R , M D , F A C S

FERGUSON, RUSSELL S , A B , M D

GUTIERREZ, ROBERT, A B , D P H A R M , M D , F A C S

HABEIN, H C , M D

HAGER, BENJAMIN H , B S , M S , (IN UROLOGY), M D

HAGNER, FRANCIS R , M D , F A C S

KEYES, EDWARD L , P H D , M D , F A C S , H O N F R C S
(LONDON)

LOWER, WILLIAM E , M D , F A C S

MacKENZIE, DAVID W , M D , F A C S

RANDALL, ALEXANDER, M A , M D , F A C S

SCHOLL, A J , M D , F A C S

MODERN UROLOGY

IN ORIGINAL CONTRIBUTIONS BY
AMERICAN AUTHORS

EDITED BY

HUGH CABOT, MD LL.D. CMG FACS

PROFESSOR OF SURGERY THE MAYO FOUNDATION GRADUATE SCHOOL OF THE UNIVERSITY
OF MINNESOTA AND CONSULTING SURGEON TO THE MAYO CLINIC ROCHSTER MINNE-
APOTA FORMERLY DEAN AND PROFESSOR OF SURGERY IN THE MEDICAL SCHOOL
OF THE UNIVERSITY OF MICHIGAN ANN ARBOR, MICHIGAN

VOLUME II

DISEASES OF THE BLADDER—DISEASES OF THE URETER
—DISEASES OF THE KIDNEY—RADIATION THERAPY
OF TUMORS OF THE GENITO URINARY TRACT

THIRD EDITION THOROUGHLY REVISED

ILLUSTRATED WITH 374 ENGRAVINGS AND 9 PLATES



LEA & FEBIGER
PHILADELPHIA

1936

COPYRIGHT
LEA & FEBIGER
1936

PRINTED IN U S A

CONTRIBUTORS

RAYMOND B. ALLEN, M.D., Ph.D. (IN UROLOGY)

Dean of the Wayne University College of Medicine, Detroit, Mich. Formerly
Dean in Charge of Graduate Studies in the College of Physicians and
Surgeons and Associate Director of the New York Post-Graduate Medical
School, Columbia University, New York City

BENJAMIN S. BARRINGER, M.D., F.A.C.S.

Surgeon, Urological Department, Memorial Hospital, New York City

A. ELMER BEIT, M.D., F.A.C.S.

Los Angeles, Calif. Associate Professor of Urology, College of Medical
Evangelists

HUGH CABOT, M.D., C.M.C., F.A.C.S.,

Professor of Surgery, The Mayo Foundation, Graduate School of the Uni-
versity of Minnesota and Consulting Surgeon to the Mayo Clinic, Roches-
ter, Minn.

EDWARD W. CAMPBELL, B.S., M.D., F.A.C.S.

Instructor in Urology in the Hahnemann Medical College, Philadelphia.
Assistant Urologist at the Abington Memorial Hospital, Abington, Pa.
Consultant to the McKinley Hospital, Trenton, N. J., West Jersey Homeo-
pathic Hospital, Camden, N. J., Riverview Hospital, Norristown, Pa. and
West Chester Homeopathic Hospital, West Chester, Pa.

JOHN R. CAULK, M.D., F.A.C.S.

Professor of Clinical Genito-Urinary Surgery, Washington University, St.
Louis, Mo.

RUSSELL S. FERGUSON, A.B., M.D.

Assistant Professor, Clinic Surgery (Urology), Cornell University Medical
College, Associate Surgeon, New York Hospital, Assistant Surgeon, Memori-
al Hospital, New York City

ROBERT GUTIERREZ, A.B., D.HARM., M.D., F.A.C.S.

New York University of Havana, Faculty of Medicine and Pharmacy, 1918.
Attending Urologist, New York Hospital and Hospital for the Ruptured
and Crippled, Assistant Attending Urologist, New York Skin and Cancer
Hospital, Chief Female Clinic, Department of Urology, James Buchanan
Brady Foundation of the New York Hospital, Urologist, Broad Street and
Pan American Hospitals, Consulting Genito-Urinary Surgeon, Chambers-
burg General Hospital, Trenton, N. J.

H. C. HABEIN, M.D.,

Assistant Professor of Medicine in the Mayo Foundation, Graduate School
of the University of Minnesota, Rochester, Minn. Consultant in Medicine
at the Mayo Clinic, Rochester, Minn.

BENJAMIN H. HAGER, B.S., M.S. (IN UROLOGY), M.D.,

Associate Clinical Professor of Surgery (Urology), University of Southern
California, School of Medicine, Member of Senior Attending Staff, Los
Angeles General Hospital and California Hospital, Los Angeles, Calif.
Formerly Associate Clinical Professor of Surgery (Urology) and Head of
Department of Urology, University of Wisconsin Medical School

FRANCIS R HAGNER, M D , F A C S ,

Professor of Genito-Urinary Surgery in the George Washington University,
Washington, D C , Attending Genito-Urinary Surgeon, George Washing-
ton University Hospital and Garfield Memorial Hospital, Washington,
D C , Consulting Genito-Urinary Surgeon, Children's Hospital, Gallinger
Hospital and Mt Alto Diagnostic Center, Washington, D C

EDWARD L KEYES, Ph D , M D , F A C S , HON F R C S (LONDON),

Professor of Clinical Surgery (Urology), Cornell University Medical College,
Attending Surgeon, New York Hospital, Consulting Surgeon, Memorial
Hospital, New York City

WILLIAM E LOWER, M D , F A C S ,

A Founder of the Cleveland Clinic Foundation, Surgeon to the Cleveland
Clinic Hospital, Chief of Staff at the Lutheran Hospital of Cleveland, Ohio

DAVID W MacKENZIE, M D , F A C S ,

Urologist-in-Chief to the Royal Victoria Hospital, Montreal, Canada, Clinical
Professor of Surgery, Department of Urology in the McGill University,
Montreal, Canada

ALEXANDER RANDALL, M A , M D , F A C S ,

Professor of Urology in the University of Pennsylvania, Philadelphia, Urolo-
gist to the Chestnut Hill and Germantown Hospitals, Philadelphia, and
Abington Memorial Hospital, Abington, Pa

A J SCHOLL, M D , F A C S ,

Urologist to St Vincent's Hospital and the Santa Fe Hospital, Los Angeles,
Calif

CONTENTS

SECTION I

THE BLADDER.

CHAPTER I

THE ANATOMY AND PHYSIOLOGY OF THE URINARY BLADDER	9
By RAYMOND B. ALLEN, M.D., F.R.D. (In Urology)	

CHAPTER II

MAJOR MALIGNANCIES AND DIVERTICULA OF THE BLADDER	25
By WILLIAM L. LOWE, M.D., F.A.C.S.	

CHAPTER III

INJURIES OF THE BLADDER	31
By FRANK R. HAGNER, M.D., F.A.C.S.	

CHAPTER IV

INFECTIONS OF THE BLADDER	60
By BENJAMIN H. HAGER, B.S., M.S. (In Urology), M.D.	

CHAPTER V

STONE IN THE BLADDER	126
By HUGH CABOT, M.D., C.M.C., F.A.C.S.	

CHAPTER VI

FOREIGN BODIES IN THE BLADDER	171
By HUGH CABOT, M.D., C.M.C., F.A.C.S.	

CHAPTER VII

TUMORS OF THE BLADDER	180
By EDWARD L. KEEFE, M.D., F.A.C.S., Hon. F.R.C.S. and RUSSELL S. FERGUSON, M.D.	

SECTION II

THE URETER.

CHAPTER VIII

THE ANATOMY, ABNORMALITIES, INJURIES, DISEASES AND SURGERY OF THE URETER	213
By ALEXANDER RANDALL, M.D., F.A.C.S. and EDWARD W. CAMPBELL, M.D., F.A.C.S.	

SECTION III.

THE KIDNEY

CHAPTER IX

THE EMBRYOLOGY, ANATOMY, PHYSIOLOGY AND FUNCTION OF THE KIDNEY	323
By H C HABEIN, M D	

CHAPTER X

MODERN TESTS OF RENAL FUNCTION	352
By A ELMER BELT, B A , M S , M D , F A C S	

CHAPTER XI

ANOMALIES OF THE KIDNEY HYDRONEPHROSIS MOVABLE KIDNEY INJURIES OF THE KIDNEY	374
By ROBERT GUTIERREZ, A B , M D , F A C S	

CHAPTER XII

INFECTIONS OF THE KIDNEY	510
By HUGH CABOT, M D , C M G , F A C S	

CHAPTER XIII

SCHISTOSOMIASIS HEMATOBIA (VESICAL SCHISTOSOMIASIS)	570
By BENJAMIN H HAGER, B S , M S (In Urology), M D	

CHAPTER XIV

RENAL HYDATID (RENAL ECHINOCOCCOSIS)	583
By BENJAMIN H HAGER, B S , M S (In Urology), M D	

CHAPTER XV

STONES IN THE KIDNEY AND URETER	598
By A J SCHOLL, M D , F A C S	

CHAPTER XVI

TUMORS OF THE KIDNEY	682
By DAVID W MacKENZIE, M D , F A C S	

CHAPTER XVII

TUBERCULOSIS OF THE KIDNEY AND URETERS	754
By JOHN R CAULK, M D , F A C S	

SECTION IV

CHAPTER XVIII

THE RADIATION THERAPY OF TUMORS OF THE GENITO-URIN- ARY TRACT	805
By BENJAMIN S BARRINGER, M D , F A C S	

MODERN UROLOGY

SECTION I THE BLADDER

CHAPTER I

THE ANATOMY AND PHYSIOLOGY OF THE URINARY BLADDER

By RAYMOND B. ALLEN, M.D., F.R.D.

THE ANATOMY OF THE URINARY BLADDER

THE urinary bladder is a sacular musculo-membranous pelvic organ which serves as a temporary reservoir for the urine. Into it the urine is discharged from the ureters in spurts at somewhat variable but short intervals. The quantity thus delivered varies widely with the several factors which determine the output of the kidneys *viz.*, with the water, liquid and solid food intake and the time of day or night in relation to meals, work and rest (*i. e.* metabolic activity) and with the physical environment of the body (in so far as it affects the activity of the skin). At longer intervals which depend on the output of the kidneys, the capacity of the bladder, the habit of the individual and after early childhood subject to the will, the urine is discharged to the exterior through the urethra. One has only to observe the handicapped condition of the patient with the incontinence of *extrophis vesicalis* to fully appreciate the good fortune of persons who have normal bladders. It should be superfluous to state that a thorough knowledge of the structure and function of the bladder is essential to one who must deal with its abnormal and diseased states.

At birth the bladder differs markedly from the adult structure. It is mainly an abdominal organ, ovoid to pear shape, lying just beneath the anterior abdominal wall covered posteriorly by peritoneum. Its base rests behind the symphysis pubis and its apex extends half way to the umbilicus. The allantois and upper one-third of the primitive bladder are obliterated in fetal life, persisting only as a fibrous cord, the urachus. During infancy and early childhood the bladder migrates with some rapidity downward and somewhat backward into

the pelvis but it fails to reach its definitive position until much later. After a stationary period in later childhood the final, slow phase, of migration begins, persisting throughout puberty and adolescence—a phase of descent which occurs at the same time as a period of relatively rapid growth of the bony pelvis. Because of the concurrent character of these changes, as Testut has pointed out, this stage of migration may be more apparent than real. One must remember, therefore, that throughout childhood and early adolescence a large

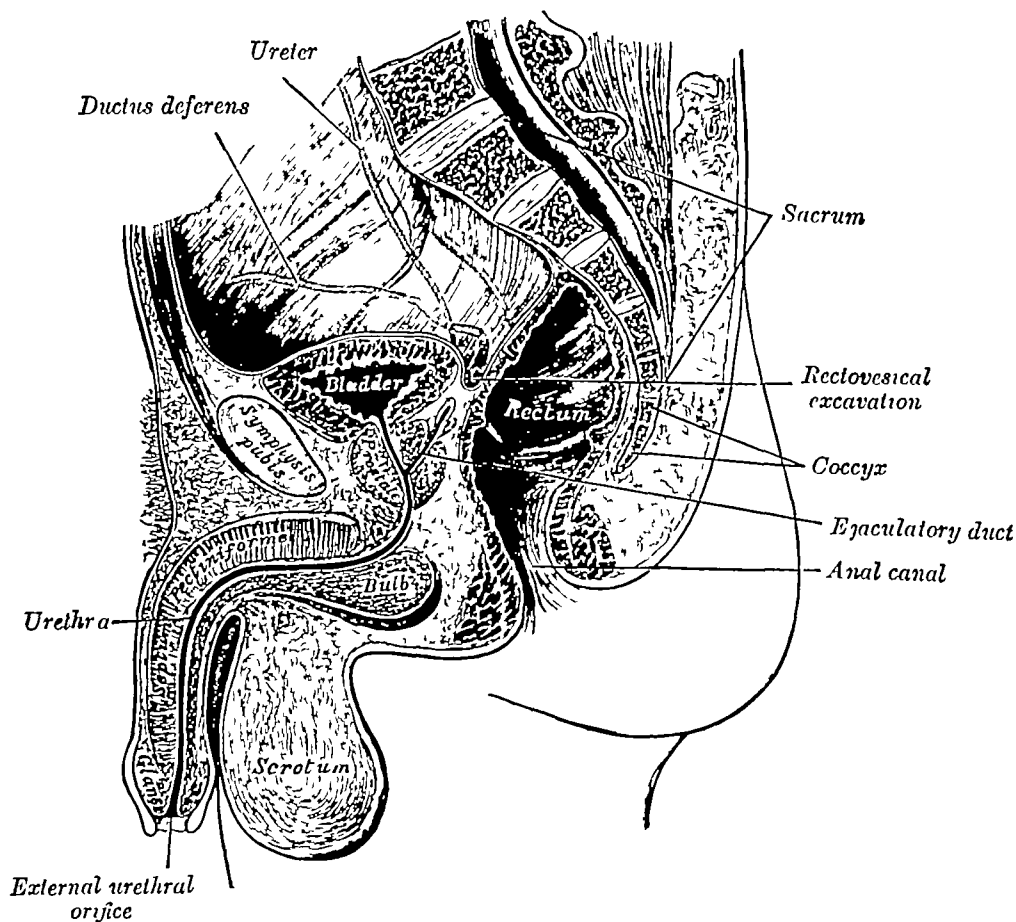


FIG. 1.—Sagittal section showing relations of the bladder to surrounding organs and peritoneum in the male (Grays' Anatomy)

fraction of the bladder when full lies above the symphysis pubis. Unwary surgeons have discovered this fact to their discomfiture and enlightenment.

The bladder of an adult when fully distended has a capacity of approximately 500 cc or 1 pint. In this state it is ovoid, slightly broader at the base, with its long axis directed upward. In front of the bladder are the symphysis and body of the pubic bone, separated only by the loose areolar connective tissue of the prevesical space (space

of Retzius). A portion of the bladder in full distention extends above the level of the symphysis and thus comes to lie behind the anterior abdominal wall. Below are the prostate and the urethra at the neck of the bladder and somewhat laterally and behind are the seminal vesicles and portions of the vasa deferentia and ureters. Behind are the lower sigmoid and rectum. The superior surface and about one-half of the posterior surface of the bladder are covered by parietal peritoneum which separates the bladder from the coils of small intestine.

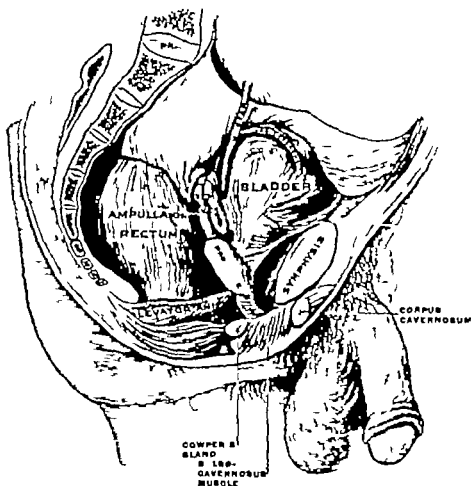


FIG. 2 — Male pelvic organs *in situ* seen from right side. Bladder and rectum distended. The arrow points to the rectovesical pouch. (Gray's Anatomy.)

the sigmoid and in the female from the uterus. The upper one-half of the vagina and the cervix lie behind and somewhat below the bladder to which they are quite firmly but not inseparably attached by connective tissue. The body of the uterus is separated from the bladder only by the vesico-uterine fossa of the peritoneal cavity. With the individual in the standing posture the uterus may rest on the bladder and distort the contour of the bladder wall. The bladder of the female, due chiefly to the absence of the prostate, lies somewhat lower in the

pelvis, which accounts for the fact that very little of it shows above the symphysis

The bladder is fixed anteriorly and above by the urachus and below at its base by the true ligaments (two) of the bladder. The urachus is taut when the bladder is empty, holding the anterior wall in position. As the bladder fills, the upper wall rises, relaxing the pull on the urachus. The true ligaments arise from the pelvic fascia on which the bladder rests. Anteriorly these are attached to the lower border of the body of the pubic bone and to the prostate and the fibrous outer coat of the bladder. Laterally the ligaments are reflections from the pelvic fascia covering the levator ani muscles and are attached

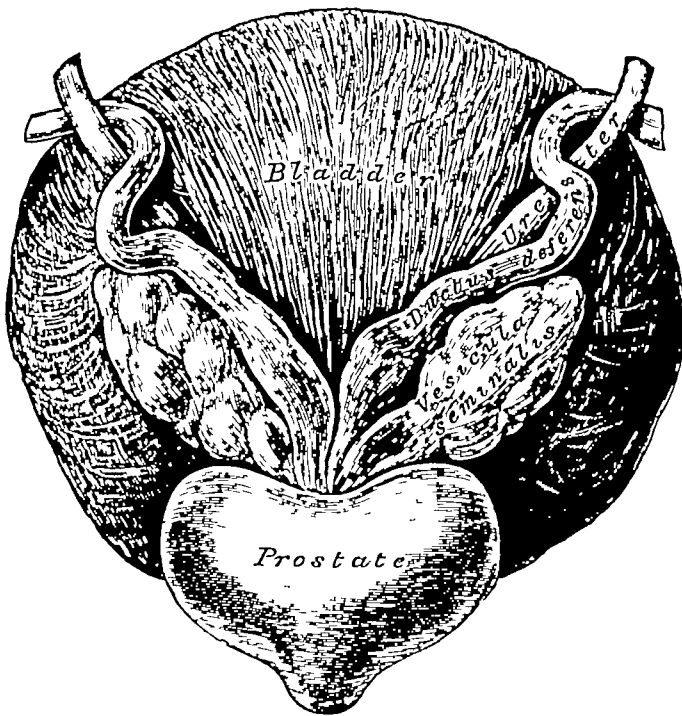


FIG. 3.—Posterior view of bladder showing relations of vasa, seminal vesicles, prostate and ureters (Gray's Anatomy)

to the fibrosa of the bladder. As the bladder contracts during micturition the upper wall is displaced downward and forward against the anterior wall and floor. The shape of the bladder changes from the ovoid to that of an inverted cone with the apex represented by the internal urethral orifice or bladder neck and the base represented by the upper wall, extending from just above the symphysis at the point of attachment of the urachus to the line of attachment of the posterior bladder wall with the rectum where the peritoneum of the recto-vesical fossa (pouch of Douglas) is reflected from the rectum onto the bladder. The lines of reflection of peritoneum onto the bladder are known as the false ligaments. Anteriorly parietal peritoneum is reflected from the

anterior abdominal wall over the urachus onto the anterior wall of the bladder. When the bladder is fully distended the line of this reflection (in the male) may lie as much as 2 inches above the symphysis but it is subject to great variation. During a suprapubic operative approach to the bladder one must therefore exercise great care in identifying the peritoneum to avoid opening the peritoneal cavity. In the collapsed bladder the plane of reflection usually lies at the level of the upper margin of the symphysis. Laterally parietal peritoneum of the

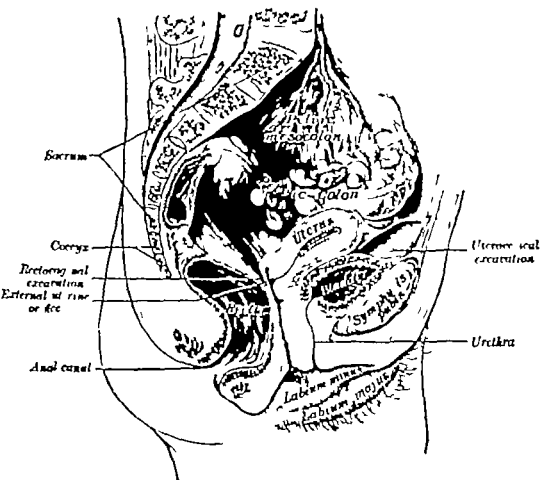


FIG. 4.—Sagittal section showing the relations of the partially filled bladder in the female pelvis (Gray's Anatomy)

pelvic wall is reflected onto the bladder. Posteriorly peritoneum of the recto-vesical pouch is reflected from the rectum onto the bladder in a plane about 2 inches behind the bladder neck and 3 inches from the anus. In the female the posterior false ligament of peritoneum is reflected from the uterus to the floor of the vesico-uterine fossa. While the upper and a portion of the posterior surfaces are invested with peritoneum it should be remembered that the bladder is essentially an extra peritoneal organ. In distending it rounds out and bulges upward and backward into the peritoneal cavity but it remains firmly

attached extraperitoneally. It is rarely, if ever, necessary to make a transperitoneal operative approach to the bladder.

Mucous Membrane.—The appearance of the mucous membrane lining the bladder as seen by means of the cystoscope with artificial light varies with the degree of distention. When the bladder is full the epithelium is smooth and has a salmon-pink color except over the trigone where it has a somewhat darker hue. Venules of the mucous membrane appearing as fine irregular interlacing dark lines can be seen through the epithelium. Occasionally larger vessels near the surface are visible,

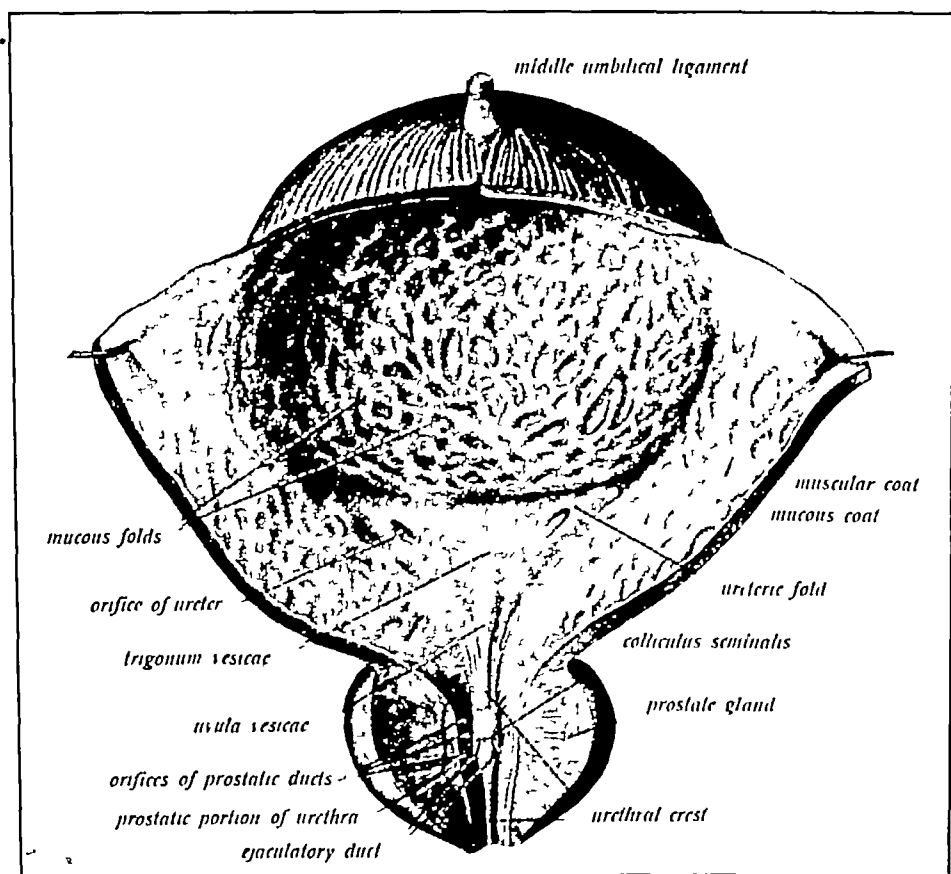


FIG. 5 — Base of the bladder with the anterior wall removed (Sobotta and McMurrich, courtesy of W. B. Saunders Company)

especially when the membrane is relaxed, but it is very rare to see pulsations. Normally the bundles of smooth muscle belonging to the inner layer of the muscular coat are scarcely visible in the full bladder but sometimes they can be seen during contraction as the bladder is emptied. Contrary to some anatomical descriptions, from their mesh-like distribution, it is evident that a purely longitudinal arrangement of the inner layer does not exist.

Contour—The contour of the bladder wall as seen from within is smoothly concave. Sometimes one sees a rounded convexity posteriorly

which may represent the impression of the uterus or a full sigmoid and rectum. The base of the bladder being fixed changes in contour but slightly and then only as a result of the opening and closing of the ureteral and urethral orifices and as a result of full distention. The three apertures make the angles of a roughly equilateral triangular area known as the *trigone* which measures 2.5 to 3 cm on a side. The two ureteral meati lie behind and one to each side of the urethra. The lateral margins of the trigone form a 60 degree angle at the urethra hence the direction of the ureteral opening is 30 degrees from the mid line a fact of practical value when one is searching for the ureteral orifices through a cystoscope. The ureters are connected by a slightly curved ridge convex toward the urethra which is called the interureteral (or Mercier's) bar. It is formed by mucous membrane covering a band of muscle first described by Mercier. The muscle continues past the orifices to merge with the longitudinal muscular layer of the ureter as it passes obliquely through the bladder wall. These muscles produce a slight elevation of the mucous membrane lateral to the orifice marking the course of the ureter through the wall. The lateral boundaries of the trigone also are defined by slight elevations caused by bands of muscle which were described first by Bell (Bell's muscle). The ureteral meatus varies in form from punctate and round to slit like and crescentic. It opens usually at the crests of slight elevations the ureteral papilla. The whole of the trigone appears slightly elevated and the bladder wall falls away behind it forming the retro-trigonal fossa or basofond. The distinctness of these several markings in the base varies widely among normal individuals. In the female they are usually less distinct. The muscular ridges of the trigone become accentuated as an early change in men who develop obstruction at the neck of the bladder. In extreme distention the trigone is stretched the distance between the ureteral and urethral orifices may increase from 2.5 cm to as much as 5 cm. The ureteral and urethral orifices are closed except when urine is being discharged. Each ureter functions quite independently of its mate and it is purely by chance that spurts from them sometimes occur simultaneously. As the urine enters the bladder it causes a noticeable swirl about the meatus which gaps visibly at such times. During micturition when the internal sphincter is relaxed the ureters are closed. At all other times the internal sphincter closes the outlet causing a slight longitudinal furrowing of the bladder neck due to radiating folds of mucous membrane. The line of closure of the urethra is crescentic due to the presence of a slight bulbous elevation the uvula in the mid line posteriorly. This elevation is caused by the fusion of Bell's muscles from either side of the trigone. In pathological enlargement the uvula may produce a ball valve obstruction.

When the bladder is only partially filled the mucous membrane lies in folds except over the trigone where it remains relatively smooth. The submucosa of the trigone is thin the mucous membrane resting

attached extraperitoneally. It is rarely, if ever, necessary to make a transperitoneal operative approach to the bladder.

Mucous Membrane — The appearance of the mucous membrane lining the bladder as seen by means of the cystoscope with artificial light varies with the degree of distention. When the bladder is full the epithelium is smooth and has a salmon-pink color except over the trigone where it has a somewhat darker hue. Venules of the mucous membrane appearing as fine irregular interlacing dark lines can be seen through the epithelium. Occasionally larger vessels near the surface are visible,

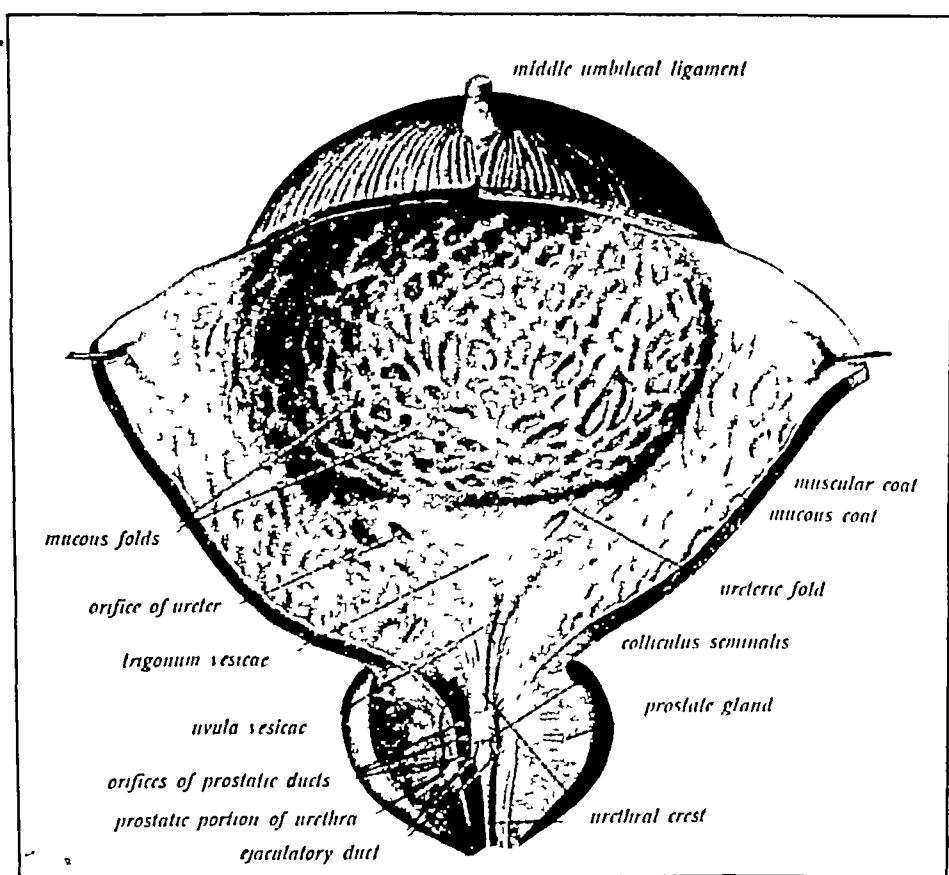


FIG. 5 — Base of the bladder with the anterior wall removed (Sobotta and McMurrich, courtesy of W. B. Saunders Company)

especially when the membrane is relaxed, but it is very rare to see pulsations. Normally the bundles of smooth muscle belonging to the inner layer of the muscular coat are scarcely visible in the full bladder but sometimes they can be seen during contraction as the bladder is emptied. Contrary to some anatomical descriptions, from their mesh-like distribution, it is evident that a purely longitudinal arrangement of the inner layer does not exist.

Contour — The contour of the bladder wall as seen from within is smoothly concave. Sometimes one sees a rounded convexity posteriorly

embryologically is of ureteral origin. They are particularly numerous and thick along the upper margin of the trigone (Meckel's bar) where they run transversely and along the lateral margins (Bell's muscle) where they take an oblique course. In the center of the trigone the muscle is therefore relatively thin.

The **internal urethral sphincter** is derived in part from the middle or circular muscle coat of the bladder and in part from the external longitudinal coat fibers from which continue posteriorly looping around the upper portion of the urethra. A few fibers from the same layer run longitudinally along the roof of the urethra. Bell's muscles from either side of the trigone join at the urethra (uvula) thinning out along the floor of the urethra.

The outermost coat, the **fibrosa**, consists of loosely arranged mixed fibrous elastic fatty areolar connective tissue. It blends with the connective tissue of the muscularis and at the base with surrounding organs and the deep pelvic fascia forming the *true ligaments* previously described. The fibrosa is thinnest above where it is covered by peritonium and contains considerable fat (the true serosa of the bladder) and thickest below and laterally where the ureters large vessels and nerves enter the bladder wall.

The Arteries—Blood reaches the bladder chiefly through the superior and inferior vesical arteries on each side. These vessels are terminal branches of the anterior division of the internal iliac arteries. The superior vesical artery sometimes divides to form a middle vesical artery. Smaller inconstant arteries to the bladder are branches of the middle hemorrhoid obturator internal pudic and inferior gluteal arteries. Small branches of the uterine and vaginal arteries reach the bladder in the female. The superior vesical arteries are functional rudiments of the fetal hypogastric arteries. All of these vessels contribute to the formation of a freely anastomosing arterial plexus in the fibrosa. A secondary arterial plexus is formed in the submucosa. Capillary beds in the muscularis and mucosa are formed from arterioles derived from the two plexuses.

The **venous vessels** do not accompany the arterioles and arteries but take separate courses to the periphery where a large external plexus is formed over the fibrosa. This plexus is best developed over the anterior and posterior walls. The anterior veins are easily identified in a supra pubic exposure of the bladder.

The Lymphatics.—In the opinion of most anatomists definite lymphatics are not found in the mucosa scattered vessels are present in the submucosa. A rich network is formed in the muscular layers where the muscle fibers are closely enmeshed in lymphatics. This plexus is drained by numerous channels leading into the network of lymphatics in the perivesical tissue lymphatics which are continuous with those of the ureters vasa vesicles prostate (uterus vagina) and rectum. Anterior vessels drain into the few small lymph nodes of the prevesical space. These in turn are drained by vessels communicating with the

on the muscularis (a fact which probably accounts for the darker hue of the trigone) Elsewhere in the bladder the submucosa is thick and loose permitting the mucosa to move freely over the underlying muscle

The Epithelium —The epithelium itself also varies with the degree of distention. It is of the transitional variety and can readily adapt itself to larger surfaces by thinning out In the collapsed state of the bladder the epithelium may be five to six cells thick The superficial cells are usually cuboidal or columnar and the underlying cells are polygonal A single layer of cuboidal to columnar germinal cells rests on the basement membrane As the bladder distends, the superficial cells become flatter and the epithelium thinner, the thickness measured in numbers of cells may be reduced to only two or three There are *no glands* in the bladder mucous membrane Rudimentary glandlike epithelial crypts have been described in the base particularly in the region of the trigone They may be of practical importance in accounting for the glandular character of certain carcinomas of this region

It should be recalled in this connection that the epithelium of the trigone is mesodermal arising in the embryo from the mesonephric and ureteric ducts, whereas the epithelium elsewhere in the bladder is entodermal developing as part of the fetal urogenital sinus It is a fact probably not without significance that most carcinomas of the bladder arise in the base in the region of the union of these germ layers, a region which probably sustains the most stress and strain in bladder function

The Stroma —The stroma of the mucosa consists of loose areolar connective tissue containing numerous lymphoid cells and sometimes lymph nodules It gradually blends with the submucosa which is less cellular, very loose, areolar connective tissue and is relatively thick containing numerous elastic tissue fibers It is penetrated by blood and lymphatic vessels and nerves

In addition to the mucosa and submucosa the bladder wall comprises the **muscularis**, the thickest layer, and the **fibrosa** or outer layer The thickness of the muscularis varies from a few millimeters when thin, to as much as 1 cm when fully contracted The muscularis consists of well formed fasciculi of involuntary smooth muscle which interlace freely, forming a veritable meshwork pattern The trabeculation of an hypertrophied bladder wall, as seen cystoscopically, is an exaggeration of this characteristic The outermost fasciculi in general, however, course longitudinally, forming a well-defined layer which has been called the *detrusor urinæ* This is a misnomer, because the whole of the muscularis acts simultaneously and the middle layer which is the best developed probably does most of the work The fibers of the middle layer tend to course in a circular transverse plane near the base and in an oblique direction near the summit The innermost fibers cannot be described as a well-defined layer except in the region of the trigone where they are fine, closely packed, and well differentiated as a separate layer Essentially, these fibers are continuations of external longitudinal fibers of the ureters which is not a surprising fact since the trigone

fibers. Terminal filamentous branches are given off to individual muscle cells.

Like the blood supply to the bladder the nerves are bilateral. They are derived from both the sympathetic and parasympathetic divisions of the autonomic system.¹

The derivation of the bladder nerves is clearly shown schematically in Fig. 6 after Learmonth. The importance of the pre-sacral nerve is plainly evident. It is the main pathway by which sympathetic fibers reach all of the pelvic organs. It is formed by contributions from the first, second, third and fourth (chiefly the first and second) lumbar paravertebral sympathetic ganglia on each side and from the pre-aortic nervous plexus above its middle root by which it is connected with the celiac, semilunar and aorticorenal ganglia. At or near the level of the promontory of the sacrum it divides into the two hypogastric nerves which terminate in the hypogastric ganglia. At operation or necropsy the pre-sacral nerve is found in or near the mid line just beneath the posterior parietal peritoneum lying with its branches between the left common iliac vein and the right common iliac artery unbedded in loose fatty areolar connective tissue.

The hypogastric ganglia lie on the lateral aspects of the rectum. They form firm networks of nerve fibers, ganglion cells and connective tissue. They serve as synaptic relay stations for all efferent sympathetic fibers except those which join ganglion cells in the bladder. Afferent (sensory) fibers of both the sympathetic and the parasympathetic divisions pass uninterrupted through the ganglia. The latter fibers are derived from the second, third and fourth sacral nerves. They are called the pelvic nerves or *nerri erigentes*.

THE PHYSIOLOGY OF THE URINARY BLADDER.

Knowledge of the physiology of the bladder until recently was based chiefly on observations in experimental animals. While helpful these studies have not fully explained certain of the phenomena of bladder activity in man. Direct observations in man (cystoscopically) of the mechanism of filling and emptying of the bladder (Learmonth) and observations on intravesical pressures (Craves, Rose and others) have yielded important information without which a complete understanding of bladder physiology was impossible.

Learmonth has demonstrated that stimulation of the *sympathetic (pre-sacral) nerve* when exposed for an operation causes (1) closure of the uretero-vesical orifices, (2) contraction of the trigone, (3) closure of the internal sphincter, (4) conveyance of sensations of pain from the bladder and (5) inhibition of the expulsive mechanism of the bladder.

Somatic fibers do not reach the bladder itself, but branches from the anterior primary divisions of the third and fourth sacral nerves supply the external sphincter of the bladder fibers which form a part of the pudic nerve. This nerve, according to Learmonth, may contain also some afferent sensory fibers from the posterior urethra.

chains of nodes along the external iliac veins. Efferent channels from the lateral perivesical plexus reach the nodes of the external iliac and hypogastric chains. Some of the posterior lymphatics also connect with this system, the remainder course along and around the rectum to the anterior surface of the sacrum ascending to the nodes at the bifurcation of the aorta. As in other organs, the flow of lymph away from the bladder is influenced by the muscular activity of the organ, during contraction of the muscularis lymph is forced into the perivesical plexus. Flow beyond this plexus is determined by the same

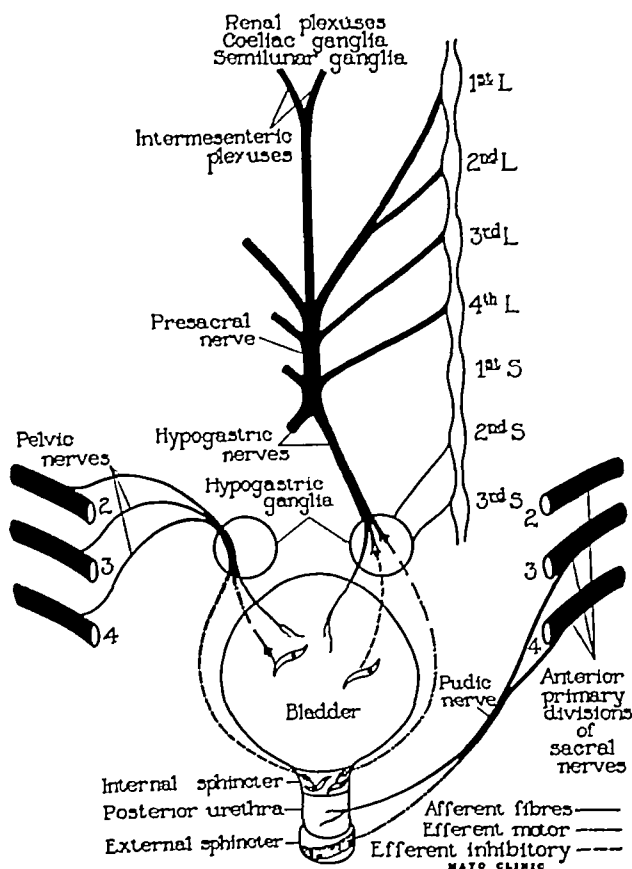


FIG. 6 —Diagrammatic representation of nerve supply of the bladder (Learmonth, *Am Jour Surg*, courtesy of Paul B Hoeber, Inc)

forces which affect the flow of lymph through main lymph channels of the abdomen

Nerves of the bladder are both medullated (sensory) and non-medullated (autonomic). Sensory fibers pierce the muscularis, branch many times in the stroma losing their medullary sheaths, and terminate as fine filaments among the cells in the epithelium. The autonomic fibers form a plexus in the fibrous coat, a plexus which contains also numerous small nests of ganglion cells. Axon branches of this plexus reach the muscularis forming a secondary plexus among the muscle

insufficient time in which to accommodate themselves to rapid stretching. The desire to void therefore comes early and the quantity voided may be small whereas with slower filling the quantity may be much larger but the intravesical pressure is the same. Intravesical pressure and not volume of content alone therefore initiates the desire to micturate.

An important extravesical factor in micturition is intra abdominal pressure. While the bladder muscularis exerts a squeezing action on the bladder content the normal abdominal pressure aids in the expulsion of urine particularly when the bladder is fully distended.

Other factors also are known to influence the frequency of urination by influencing bladder irritability. Exposure to cold increases irritability a fact which is known from common experience in cold climates. Warmth depresses bladder irritability. Apprehension, fright, emotional excitement and suggestion may increase irritability. Sudden unusual stimulation of any peripheral somatic nerve may be reflected in increased bladder irritability indicating that the micturition reflex center is very sensitive indeed. Diseased states of the bladder (inflammation, stone, etc.) cause marked alterations in muscular tone.

In urological practice rapid introduction of irrigating solutions through the cystoscope or catheter increases irritability and prevents full distention. Normal salt solutions, boric acid, potassium permanganate and argyrol solutions in the usual concentrations are non-irritating. Hypertonic sodium bromide and iodide solutions (2 per cent) are depressing to bladder tones and injure epithelial cells as well. The more concentrated solutions of mercuriochrome and silver nitrate usually are very irritating. Hot solutions depress bladder tone. Cold solutions stimulate contraction.

In temperate climates frequency of urination varies between four to seven times in twenty four hours, the total output in the same period is 500 to 1500 cc. As previously stated fluid intake, metabolic activity and the physical environment of the body as it affects the activity of the skin are factors which influence the urinary output. Heavy beer drinkers notoriously have large volume outputs as do patients with diabetes insipidus and mellitus. At the other extreme Begg reports that in a British possession in the tropics where the temperature often reaches 125° F. the natives have to be reminded from day to day to micturate¹.

Normal intravesical pressures do not cause regurgitation of urine up the ureters. This is prevented by the sphincteric-like action of the muscularis at the ureteral orifice and it is generally believed by the fact the ureter takes an oblique course through the bladder wall. In order for the ureter to discharge urine into the bladder it must overcome the intra-ureteric pressure which normally may be as high as 15 mm. of mercury. Under normal conditions the uretero-vesical valves

¹ Vesical calculus is very common among these people.

An extravescical response, (6) contraction of the musculature of the prostate, seminal vesicles and ejaculatory ducts, also was observed

Parasympathetic nerves, on the other hand, are concerned with the initiation and augmentation of the muscular contractions of the bladder. Paresis or paralysis of the bladder is known to result from lesions in any part of the parasympathetic pathway, from the sacral cord to the hypogastric ganglia. From the fact that the internal sphincter opens and closes when required after division of the presacral (sympathetic) nerve, it can be deduced that the parasympathetics inhibit the sphincter. Afferent fibers in the micturition reflex arc must be carried by parasympathetic pathways because normal micturition can occur after division of the presacral nerve. Sensation also is unaffected by presacral section indicating that afferent (sensory) fibers are present in parasympathetic nerves.

Learmonth concludes from a study of these functions that the sympathetic nerves are "filling" or "storing" in function (dilatation of bladder and closure of its sphincter) whereas the parasympathetic nerves are "emptying" in function (contraction of bladder and opening of its sphincter).

Sherrington, working with experimental animals, and more recently Graves and Rose (independently) making their observations on man, have studied intravesical pressures during the various stages of filling and emptying of the bladder. During gradual filling the muscle fibers respond by slight rhythmic wave-like contractions. These contractions increase in vigor as the bladder fills but they cease when the maximum intravesical pressure of 8 to 15 mm. of mercury is reached, at which point a sustained tonic contraction of the muscularis occurs. The content of the bladder at this point varies widely between individuals but is fairly constant from time to time in the same subject. It averages about 250 cc., the usual amount voided at one time. As previously stated, however, the fully distended bladder may contain 500 cc.

In the denervated bladder of experimental animals during the later stages of filling, the rhythmic contractions become greatly augmented until finally the increased intracystic pressure overcomes the sphincters at the outlet, thus bringing on automatic urination, which, at least partially, empties the bladder. This phenomenon suggests that the ganglion cells of the bladder fibrosa act as local reflex nerve centers for automatic bladder control (von Zeissl). The automatic bladder of the tabetic or of the patient with transverse myelitis of the sacral cord are comparable phenomena in man.

From these studies it is clear that the desire to void is not initiated by a given quantity of urine distending the bladder but rather by the intracystic pressure induced by the tonic contraction of the muscularis. Under conditions of rapid filling (diuresis) the intravesical pressure may reach a maximum quite early because the muscle fibers have

CLINICAL CONSIDERATIONS

When empty the bladder cannot be felt suprapubically. Its base, however, can be felt by rectum in the male, and by vagina in the female. When fully distended, with the abdomen relaxed, the bladder can be felt dome-like above the symphysis. Its outline can be delineated by percussion. The smoothly ovoid symmetrical outline of the bladder can be seen in the cystogram (Fig. 7). Other roentgen-ray studies, urinalysis, catheterization for residual urine and cystoscopic examination complete the diagnostic procedures.



FIG. 7.—Normal cystogram showing contour and relations to bony pelvis. (Mayo Clinic)

There are three operative approaches: suprapubic, perineal, and transurethral, all being extra-peritoneal. If the bladder is filled before a suprapubic operation, the danger of opening the peritoneal cavity is lessened. The bladder wall is distinguished by prominent veins on its anterior wall. It is unnecessary and inadvisable to open the prevesical space. If this precaution is taken, extravescical drains need not be used and prevesical infection, a serious complication, is avoided (Cabot).

The high lying bladder of the infant and child should not be forgotten during laparotomy on the young.

The bladder, especially if dilated, as in cases of bladder neck obstruction in aged men, may be included among the structures in the defect of large inguinal and femoral herniae, a contingency for which one must be alert.

With the advent of transurethral prostatic resection as the operation of choice in many of the cases of prostatic obstruction, a thorough

withstand this pressure and remain competent. Bladder neck obstruction or any other factor which increases bladder irritability and tone favors the incidence of regurgitation (Graves). Urine never regurgitates from an atonic bladder unless the uretero-vesical valve is incompetent. Ureteral hypertrophy and dilatation, when associated with increased intravesical pressures from whatever cause, are due to the excessive work which the ureter must do in order to discharge urine into the bladder. Under these conditions, according to Graves, the uretero-vesical valve may become incompetent and regurgitation thus occur.

The **micturition reflex** functions in its simplest form in untrained infants. According to Starling and to Halliburton the center for reflex control is probably located in the lower lumbar and sacral portion of the spinal cord. This is deduced from the fact that section of the cord above this level in dogs does not interfere with reflex emptying of the bladder. Patients with traumatic transverse myelitis above the lower lumbar level of the cord may be trained to empty their bladders, at least partially, periodically without conscious control being necessary. Spontaneous emptying of the bladder in the infant occurs as a pure reflex act. It is doubtful if the very young infant experiences any sensation from a full bladder. With the development of higher pathways and with training, conscious control is gradually exercised, and the reflex center is conditioned and subordinated to the higher center or centers. That one or more such centers exist there is no doubt, but the exact location is unknown. Czyhlarz and Marburg believe there are three centers: a cortical center in the motor area, a center in the corpus striatum and a third in the optic thalamus. Tigerstedt observed bladder contractions in the cat after stimulation of the anterior part of the sigmoid gyrus, suggesting that the pathway may be through the thalamus, crura cerebri, pons and medulla to the cord. The pathway in the cord is said to be in the posterior portions of the lateral columns and (Wesson) near the pyramidal tracts.

NON-ABSORPTIVE POWER OF THE BLADDER

It is not surprising that experiments uniformly show that the mucous membrane lining the bladder is non-absorptive because, as has been pointed out, the epithelium is stratified (transitional), contains no glands and the underlying stroma contains no lymphatics. This, of course, is a self-protective mechanism to prevent the reabsorption of urine which does not occur even in the overdistended bladder. Five cc. of a 5 per cent solution of potassium cyanide placed in the bladder of a dog has no appreciable effect (Macht). Five per cent cocaine hydrochloride introduced into the urethra produces collapse and convulsions but has no effect when introduced into the bladder, a point of great practical importance.

CHAPTER II

MAIFORMATIONS AND DIVERTICULA OF THE BLADDER

By WILLIAM I. LOWER, M.D., F.A.C.S.

The malformations of the bladder included in this chapter are as follows: patent urachus; absence of the bladder; multilocular bladder; hour-glass bladder; diverticuli of the bladder; and exstrophy of the bladder.

I. PATENT URACHUS

The urachus in man as in other mammals is an impervious cord which is embedded in the subperitoneal tissue and passes from the apex of the urinary bladder to the umbilicus. In the adult it varies in length from 3 to 10 cm. and averages 5 cm. at the bladder, tapering to 1 mm. at the apex. It lies between the transversalis fascia and the peritoneum and is freely movable in its lowest part. It is a musculo-tendinous structure and thus acts as a balancing mechanism during the expansion and contraction of the bladder.

Some difference of opinion exists concerning the origin and development of the urachus. Contrary to the view of many writers that the bladder and urachus persist from the allantois and that this structure in normal adult life has become a mere fibrous cord, Begg¹ after a very comprehensive and careful study has concluded that the urachus is the modified—not obliterated—upper end of the fetal bladder and that the allantois plays no role in the development of the bladder. This fact, he states, has been proved beyond question by the research of Lelievre. At birth the urachus reaches the umbilicus. Shortly after birth the urachus normally descends with the bladder leaving the umbilicus. Lack of closure of the upper end of the bladder at this time interferes with the descent of that organ. In case this occurs the bladder remains attached to the umbilicus and a discharge of urine occurs. Begg believes that this explains all congenital vesico-umbilical fistula and that urinary fistula at the umbilicus are frequently vesico-umbilical and not uracho-umbilical.

Various classifications of patent urachus have been suggested and Monod's² modification of Bousvier has been used most extensively. Begg¹ gives the following classification which is similar to that of Monod. Congenital fistula are of two varieties. The first variety includes the cases in which the urine flows freely or perhaps exclusively from the umbilicus. These cases are the result of complete non-development of the urachus, the cavity of the bladder reaching the umbilicus. They are easily closed but tend to re-open if there is

knowledge of the anatomy of the bladder neck is more than ever important. Care must be exercised to avoid perforating the bladder wall in excising portions of the prostate which bulge upward into the bladder.

BIBLIOGRAPHY

- 1 Arey, Leslie B. *Developmental Anatomy*, Philadelphia, W. B. Saunders Company, 1924
- 2 Braus, Herman. *Anatomie des Menschen*, II, Berlin, Julius Springer, 1924
- 3 Cabot, Hugh. Infection, Central Problem in Treatment of Prostatic Obstruction, *Proc. Staff Meetings, Mayo Clinic*, 6, 163-165, 1931
- 4 Davis, G. G. *Applied Anatomy*, Philadelphia, J. B. Lippincott Company, 1913
- 5 Delbet, Paul. *Traite D'Anatomie Humaine* (Poirer et Charpy) V, Paris, Masson et Cie, 1907
- 6 Disse, J. *Handbuch der Anatomie* (Bardeleben) VII, Jena, Gustav Fischer, 1902
- 7 Graves, R. C., and Davidoff, L. M. Studies on the Ureter and Bladder, *Jour. Urol.*, vol 10, 1923, *Ibid.*, vol 12, 1924, *Ibid.*, vol 14, 1925
- 8 Gray, Henry. *Anatomy of the Human Body* (Lewis), Philadelphia, Lea & Febiger, 1936
- 9 Howell, William H. *A Textbook of Physiology*, Philadelphia, W. B. Saunders Company, 1915
- 10 Langley, J. W., and Anderson, H. K. Reflex from Sympathetic Ganglia, *Jour. Physiol.*, vol 16, 1894
- 11 ———. The Innervation of the Pelvic and Adjoining Viscera, *Ibid.*, vol 12, 1895-1896, vol 54, 1931
- 12 Learmonth, James R. A Contribution to the Neurophysiology of the Urinary Bladder in Man, *Brain*, 54, 147-176, 1931
- 13 ———. Neurosurgery in Diseases of the Urinary Bladder, *Am. Jour. Surg.*, New Series, vol 16, April, 1932, pp 270, 274, November 1, 1932
- 14 Macht, D. D. Absorption of Drugs and Poisons from the Bladder and Urethra, *Jour. Urology*, vol 2, 1918
- 15 Piersol, G. A. *Human Anatomy*, Philadelphia, J. B. Lippincott Company, 1911
- 16 Rieffel, Descamps et Aubaret. *Encyclopedie Francaise D'Urologie* III, Paris, Octave Doin et Fils, 1914
- 17 Rose, D. K. Various Cystograms and Their Interpretation, *Jour. Urol.*, 27, 207-228, 1932
- 18 Scammon, R. E. *Developmental Anatomy*, Section I, Morris Human Anatomy (Ed. Jackson), Ninth Ed., 1933, Philadelphia, P. Blakiston's Son & Co., Inc.
- 19 Starling, E. H. *Principles of Human Physiology*, Philadelphia, Lea & Febiger, 1936
- 20 Symington, J. *Elements of Anatomy* (Quain) II, London, Longmans, Green & Co., 1914
- 21 Testut, L. *Traite D'Anatomie Humaine* IV, Paris, Octave Doin et Fils, 1912
- 22 Von Zeissl, M. Ueber die Innervation der Blase, *Arch. f. d. ges. Physiol.*, 50, 111, 1893
- 23 Wesson, M. B. Anatomical, Embryological and Physiological Studies of the Trigone and Neck of the Bladder, *Jour. Urol.*, vol 4, 1920

in a young girl whose urethra was occluded by a membrane nearly $\frac{1}{4}$ inch in thickness.

Patent urachus may be observed at any age the ages of cases cited in the literature ranging in general from 7 to seventy nine years. Men are affected more frequently than women.

Begg¹ states "Of 18 cases 31 were males 13 females and the sex was not stated in 11. In Stadfeld's series 12 were males and 2 females. Thus led this author to the conclusion that congenital phimosis was an important causal factor and it is worthy of note that if 31 cases be artificially separated in my series in which the umbilical flow of urine was very copious the proportion of males to females is 3 to 1. Thirty nine of Begg's patients were under six years of age and only 3 were over forty five years of age. Cohn⁶ reports that 90 per cent of all cases occurred in males. In my own cases 4 were boys aged four and a half months four thirteen and fifteen years and 2 were women one aged thirty-eight years and the age of the other was not stated.

Griffith⁷ called attention to those cases in which although the external outlet of the urachus is concealed there will be a constant discharge of mucus from the navel. In such cases the careful introduction of a sterile probe through the small opening which is more or less concealed by the folds of the umbilicus will demonstrate the existence of a fistulous tract which extends downward in the linea alba toward the bladder. Often if the opening in the umbilicus is plugged up with foreign matter a small abscess forms in the navel and when the patient appeals to the surgeon for relief from this condition he is unaware that he has a congenital deformity. Griffith on two occasions found the so-called umbilical stones in this blind external urachus the nucleus of each being extraneous material. Cullen⁸ reported a case in which the urachal cyst filled the entire abdomen and contained 52 liters of fluid.

Diagnosis.—The condition is very easily recognized. If the urine is discharging through the navel or through an umbilical fistula the diagnosis is evident. The urine may ooze out drop by drop or it may flow in a small stream the latter condition being more often noted during micturition. Sometimes the region around and below the navel is inflamed and painful and occasionally a swelling or a tumor mass may be seen in this vicinity. Cystitis is a frequent complication.

Differential Diagnosis.—It should be remembered that urine may escape from the umbilicus in the presence of diseases of the kidney or ureter. Such a urinary fistula is not an umbilicovesical fistula.

In nearly all cases some form of obstruction of the urethra is found. Dailey⁹ emphasizes the importance of careful investigation of every possible source of obstruction. In children vesical stones congenital urethral stricture or phimosis may account for the obstruction and in adults a careful search should be made for stone stricture and prostatic hypertrophy.

backward pressure in later life. The second variety includes the cases in which the urine escapes drop by drop. These are due to retarded closure of the ventral cloaca to form the urachus. When once cured, the bladder tends to descend naturally and the urachus forms, so that once cured there is no tendency for the fistula to re-open.

"It follows that acquired fistulae are of two types. In the first type, through mal-development there is no urachus, and the bladder apex is at the umbilicus. This condition is shown by reported cases to have been frequently present. In the second type the urine escapes through the dilated terminal centimeter of the urachal canal, or through the weak point at the junction of the urachus with the bladder. It creeps up in the confined limits of the space in which it finds itself. The peritoneum and transversalis fascia fuse near, at the umbilicus, preventing its further progress, and it bursts through the weak point formed by the depression in the lowermost quadrant of the umbilicus."

Begg's experiments and studies have shown that the epithelial canal of the urachus is never obliterated by fibrous tissue although it is impervious in parts due to epithelial debris derived from its own cells. Its lumen is in direct communication with that of the bladder in 33.3 per cent of cases. In cases in which it communicates with the bladder, the lower part of the canal frequently is sacculated and, due to the pressure of urine, this sacculus sometimes ruptures and the escaped fluid thus is allowed to pass up between the transversalis fascia and the peritoneum and to discharge at the umbilicus. However, Begg also states that a urachus which has once descended and assumed normal proportions can never convey urine from the bladder to the umbilicus.

White and Holm³ have reported a case of acquired vesico-umbilical fistula which was caused by back pressure of the urine from the bladder due to urethral occlusion by an hypertrophied prostate. They reported 6 cases in the literature in which vesico-umbilical fistula was caused by hypertrophy of the prostate gland. Since White and Holm's report, Colby⁴ has reported a similar case due to hypertrophy of the prostate gland and he also states that patent urachus associated with bladder infection and obstruction, such as enlargement of the prostate, may account for certain cases of pericystitis of obscure origin.

A blind internal urachus may be converted into a completely patent urachus by the bursting open of the closed end as the result of an abscess. The complete patency of the urachus may follow an obstruction of the urinary tract, by urethral stricture, by cystitis (tuberculous or otherwise), by phimosis, by prostatic hypertrophy, by calculi or by any other condition which would cause a back pressure in the bladder sufficient to break through the lower end of the urachus and thus complete the passage between the bladder and the navel. There are cases on record in which, after prolonged retention, urine has suddenly burst forth from the umbilicus. Kirrmission⁵ mentions a case reported by Cabrol, in which patency of the urachus was found

in a young girl whose urethra was occluded by a membrane nearly $\frac{1}{2}$ inch in thickness

Patent urachus may be observed at any age the ages of cases cited in the literature ranging in general from 7 to seventy nine years. Men are affected more frequently than women

Begg¹ states "Of 18 cases 13 were males 15 females and the sex was not stated in 11. In Stadfeld's series 12 were males and 2 females. This led this author to the conclusion that congenital phimosis was an important causal factor and it is worthy of note that if 31 cases be artificially separated in my series in which the umbilical flow of urine was very copious the proportion of males to females is 3 to 1. Thirty nine of Begg's patients were under six years of age and only 3 were over forty five years of age. Colin⁴ reports that 80 per cent of all cases occurred in males. In my own cases 4 were boys, aged four and a half months four thirteen and fifteen years and 2 were women one aged thirty-eight years and the age of the other was not stated

Griffith⁷ called attention to those cases in which although the external outlet of the urachus is concealed there will be a constant discharge of mucus from the navel. In such cases the careful introduction of a sterile probe through the small opening which is more or less concealed by the folds of the umbilicus will demonstrate the existence of a fistulous tract which extends downward in the linea alba toward the bladder. Often if the opening in the umbilicus is plugged up with foreign matter a small abscess forms in the navel and when the patient appeals to the surgeon for relief from this condition he is unaware that he has a congenital deformity. Griffith on two occasions found the so-called umbilical stones in this blind external urachus the nucleus of each being extraneous material. Cullen⁸ reported a case in which the urachal cyst filled the entire abdomen and contained 52 liters of fluid

Diagnosis.—The condition is very easily recognized. If the urine is discharging through the navel or through an umbilical fistula the diagnosis is evident. The urine may ooze out drop by drop or it may flow in a small stream the latter condition being more often noted during micturition. Sometimes the region around and below the navel is inflamed and painful and occasionally a swelling or a tumor mass may be seen in this vicinity. Cystitis is a frequent complication

Differential Diagnosis.—It should be remembered that urine may escape from the umbilicus in the presence of diseases of the kidney or ureter. Such a urinary fistula is not an umbilicovesical fistula

In nearly all cases some form of obstruction of the urethra is found. Dailey⁹ emphasizes the importance of careful investigation of every possible source of obstruction. In children vesical stones, congenital urethral stricture or phimosis may account for the obstruction and in adults a careful search should be made for stone stricture and prostatic hypertrophy

Begg¹ states that "the main points to decide in cases of urinary discharge from the umbilicus are

"1. Does the apex of the bladder itself reach to the umbilicus? And if so, is the upper segment narrowed in the form of a canal representing a partially formed urachus?

"2 Does the urine escape from a normally placed bladder reaching the umbilicus by a fistulous track between the peritoneum and the transversalis fascia?

"A careful study by cystoscopy and radiography after the injection of opaque fluid such as sodium iodide into the bladder and fistula is the only method of exact diagnosis "

Treatment —In determining the treatment for this condition, it is well to remember that all operative procedures on this vestige of an embryonic condition are attended with danger to life Early operation is advisable because of the danger that the bladder and kidneys may become infected In cases of congenital fistula, most writers agree that it is best to wait until the child is a year old before operation is performed After that age, if the fistula has not healed spontaneously, there should be no delay unless there are complications which contraindicate operation Of course, in the old or in patients who suffer from prostatic hypertrophy, the condition of the patient must decide whether operation is advisable The first step is to search for and remove anything that may interfere with the free normal egress of urine from the bladder In male children, one should look especially for phimosis, for in the great majority of cases the cause is some obstruction to the lower urinary tract, and if this is removed, the fistula often will heal spontaneously

Mast, Streamer and Unfug¹⁰ state that if an obstruction is found and corrected, moderate pressure by pads and adhesive straps over the umbilicus and linea alba occasionally has effected a cure Some surgeons recommend that the urachal tract be opened with thorough cauterization and successful results have been reported in a few cases In cases in which radical extirpation of the entire urachus is necessary, great caution must be exercised to protect the peritoneal cavity from infection, and allowance must be made for bringing the edges together The sections of mucous membrane should be turned in carefully and the bladder wall should be closed by interrupted catgut sutures The question of whether to close or drain must be decided by the surgeon

II ABSENCE OF THE BLADDER

Absence of the bladder is an abnormal condition which usually is accompanied by other deformities, particularly by the termination of the ureters in some unusual position As a rule, nothing can or need be done for a case of absence of the bladder unless the ureters open into the vagina externally when they may be transplanted into the rectum

III MULTIOCLULAR BLADDER.

According to Edwards,¹¹ there are now about 20 cases of true multilocular bladder reported in the literature but he points out that this figure is indicative of a more apparent than real degree of rarity because of lack of figures on the number of abnormalities which occur in still-born infants and the infrequency of necropsies which are performed on children.

Diverticula of the bladder must be differentiated from multilocular bladder and Edwards points out that rarely does a ureter open into a diverticulum but each loculus usually has at least one ureter. bladder loculi usually have a more defined muscular coat than is found in the walls of the diverticulum. Invariably a diverticulum opens into the bladder and this orifice usually is small however in multilocular bladder such a communication may be of any size or it may be absent entirely.

Most multilocular bladders are double and although this anomaly apparently occurs more frequently than absence of the bladder many cases of so-called double bladder in reality are doubtless cases of diverticula of the bladder. The occurrence of triple and even of quintuple bladder has also been noted.

The indication for treatment of these cases depends upon the inconvenience caused by the deformity, and the technique is governed by the individual case.

IV HOUR-GLASS BLADDER.

True hour-glass bladder is an exceedingly rare anomaly and great confusion exists regarding its true incidence due to the application of the term hour-glass to various deformities of the bladder such as diverticulum hypertrophic transverse ridges or cicatricial formation following injury operation or ulceration and patent urachus.

Kearns and Turkeltaub¹² have defined true hour-glass bladder as "a congenital malformation of the urinary vesicle caused by a partial transverse septum which divides the cavity into an upper or cephalad compartment and a lower or caudal compartment. The communicating aperture varies in diameter from 1 to 5 cm. The relative size of the upper and the lower compartments is variable. The ureteral orifices may open into the upper compartment or the orifices may be located in the lower compartment."

Because of the arrangement of the bladder into two compartments the one emptying into the other residual urine ultimately develops and with the onset of infection patients are seen with the usual symptoms of cystitis.

Kearns and Turkeltaub¹² state that differentiation between patent urachus and hour-glass deformity of the bladder is not difficult but that differentiation between acquired diverticulum of the bladder vault and true hour-glass anomaly is more difficult and in some

instances may be impossible. As diverticula frequently develop subsequent to an obstruction, the history may be elucidating. The wall of the diverticulum is quite consistently lacking in the muscular elements and their arrangement as seen in the bladder. Moreover, the division in the hour-glass conformation is apt to be thick and shelf-like and the mucosa over it smooth in contradistinction to the wrinkled appearance of the mucous membrane which is being invaginated into a diverticular cavity."

Treatment—Caulk¹³ recommends complete resection of the hour-glass bladder and he emphasizes the importance of free mobilization of the bladder before resection, the value of ureteral catheter drainage and the necessity for complete removal of the transverse partitions. This method is particularly applicable when the ureters empty into the lower portion of the bladder, but when the ureters empty into the upper cavity, the resection of the lower compartment is complicated, and it is possible that plastic operations would be more feasible in certain types of hour-glass deformity.

V DIVERTICULUM OF THE BLADDER

Diverticulum or sacculation of the bladder are names which sometimes are used interchangeably to designate a condition which has not received much clinical recognition until comparatively recent years. Until 1906, only 5 cases had been reported in the United States, although Durrieux¹⁴ had reported 194 cases in 1901 in an extensive review of the foreign literature. Since then, however, the number of cases has increased in proportion to the development of diagnostic measures. Although the etiology of diverticula is still in dispute, much attention has been devoted to the treatment of this condition and various operative procedures have been devised which yield satisfactory results.

Structure—Diverticula of the urinary bladder may be large or small, single or multiple. They may involve the musculature of the bladder wall or merely the mucosa. Englisch¹⁵ makes a distinction between "true" and "false" diverticula, agreeing with Rathbun¹⁶ that "true diverticula comprise those cases in which there can be demonstrated in the wall of the diverticulum all the coats of the bladder," whereas "false" diverticula comprise those "formed by mucous membrane only." The walls vary in thickness, the thickness as a rule increasing with the amount of inflammation of the diverticulum, although there may be a variation in different portions of the wall of a single diverticulum. Hinman¹⁷ notes that there is no epithelial lining of the lumen, and that the wall is not arranged in the definite layers which characterize the normal bladder wall. In all his cases he noted that the superficial layer next to the lumen showed a chronic inflammatory reaction with engorgement of blood-vessels and round-cell infiltration. "Muscle was invariably scattered in bands, inter-

woven with dense connective tissue. In not one case were definite muscle layers, as found in the bladder wall demonstrable. This appearance is indeed very characteristic. Diverticula have a smooth glistening surface which resembles parchment in contrast to the velvety appearance of normal mucosa. Lutz¹⁸ states that diverticula may vary in form and that they may be cone-shaped oval pear-shaped angular round or saccular and that they may number from 1 to 40. Negro and Blane¹⁹ found that the multiplicity usually varied inversely with the size of the diverticula.



FIG. 8.—Roentgenogram of large diverticulum.

Jarosz²⁰ believes that acquired diverticula usually are multiple and that they consist of atrophied mucous membrane with a thinned-out or missing musculature and that they communicate with the bladder through a large opening.

Size.—The size varies from that of an acorn to that of a baseball and may even be larger (Figs 8, 9 and 10). Not infrequently the sac of the diverticulum is larger than the bladder itself and its capacity

vented in the Alexandrian School, for their use was first introduced to Roman surgery by Eulpius of Alexandria, shortly before the beginning of the Christian Era.

Three great encyclopedists and compilers of the first three centuries, A.D., Celsus, Galen and Orribasius, transmitted Greek and Alexandrian medicine to posterity. It was Celsus who gave the first accurate account of the use of the ligature. He lived in Rome about the beginning of the first century. Whether he was a physician is debatable, but he was an able and scholarly compiler. He wrote an encyclopedia of the arts and sciences of his time, with six sections on medicine and two on surgery. In his discussion, "The Proper Manner of Arresting Hemorrhage," he says:

If there is fear that there may be bleeding one should fill the wound with dry lint, place over it a sponge wrung out of cold water and press upon it with the hand. If the bleeding still continues, it is advisable to change the stuffing of lint somewhat frequently and if this step proves ineffective, then lint moistened with vinegar may be tried, for this liquid acts energetically in arresting hemorrhage. Some physicians, indeed, actually pour it into the wound. There is strong objection, however to the use of an agent, which, like vinegar, arrests the bleeding too completely for it is apt to set up afterwards an intense inflammation of the parts. The same reasoning applies with even greater force to the employment of corrosives and caustics which produce an eschar. Despite the effectiveness of most of these in arresting hemorrhage their use should be discontinued.

Finally if the bleeding continues it will be necessary to grasp the vessel, from which the blood is escaping, and to ligate it in two places, close to the wound, and then divide the vessel between the two ligatures, in order that it may retract (both the new orifices having been already closed by the ligatures). If the circumstances are such that the plan just recommended cannot be carried out, it will then be advisable to apply the red hot cautery to the bleeding vessel.

It is interesting to conjecture why the ligature was used almost as a last resort—probably because the technique of applying it was still in the formative period.

Hellodorus who lived in the Greco-Roman period at the beginning of the second century A.D., in discussing the operation for hernia, wrote "We ligature the larger vessels, but as to the smaller ones, we catch them with hooks and twist them many times thus closing their mouths."

Notwithstanding the knowledge at that time of the use of the ligature, it was not employed for amputations, operations which in those early days were not performed until gangrene had set in, when the incision was made distal to healthy tissue. However

Hellodorus, in discussing amputations had this to say "The hand or foot is amputated if gangrene takes place, or necrosis of an extremity from any other cause. The lower portions of an extremity are removed with less danger the parts above the ankle or knee with greater risk, on account of the great danger of hemorrhage in most cases, because of the division of large blood vessels.

Galen (130-200 A.D.) advised the ligation of arteries when hemorrhage could not be controlled otherwise and is said to have used it later for amputations. He told where he got his Celtic linen thread for his ligatures—"at a shop on the Via Sacra, between the Temple of Rome and the Forum," a shop near his house on the Via Sacra, by the Temple of Peace.

Paulus Aeginatus copies Galen in his treatment of hemorrhage, but adds "You may know whether it is a vein or an artery that pours forth the blood from this, that the blood of an artery is brighter and thinner and is evacuated by pulsations whereas that of the vein is blacker and without pulsations."

For the next 1000 years of the Dark and Middle Ages surgery sank to lower and lower levels. Anything which had to do with the shedding of blood was assigned to the barber surgeons, who had no knowledge of the ligature, and the cautery caustics and astringent ointments dominated the treatment of hemorrhage.

Lanfranchi of Milan became a political exile, spending most of his professional life in France. He became associated with the French surgeon Jean Pitard in the College de St. Come, which the latter had founded in Paris in 1306. The better educated surgeons of Paris tried to elevate the art of surgery and separated themselves from the barber surgeons. They wore long robes, whereas the barber-surgeons wore short robes, and therefore were called the surgeons of the long robe." Lanfranchi wrote a notable treatise on surgery the *Chirurgia Magna*, and in it discussed the problem of hemorrhage. He distinguished between venous and arterial bleeding and advised the use of a styptic with digital compression for an hour and for severe cases the use of the ligature.

Henri de Mondeville, whom we have discussed in his role as a protagonist of clean wound repair was a contemporary of Lanfranchi and an advocate of the ligature. He wrote "Distinguish always between oozing hemorrhage, hemorrhage by jets and that which pumps out of an inward wound." He

points out the fault of the cautery in stopping bleeding—that when the eschar separates the hemorrhage may recur and the wound must be disturbed a second time for another application of the cautery. "Let the vessel be isolated from the surrounding parts with the knife, and torsion be used with ligature. Do not, as Galen teaches, allow the wound to bleed with the notion of preventing inflammation for you will only weaken the patient's vitality give him two diseases instead of one, and favor secondary hemorrhage. When your dressings have been carefully made, do not interfere with them for some days. Do not pull your dressings about, Nature works better alone."

Guy de Chauliac, who did so much to reject the teachings of Hugo, Theodoric and Henri in primary wound healing, was no more constructive in his dealing with hemostasis, for he still preached the use of styptics and the cautery.

It was not until Ambroise Paré (1510-1590) the most famous of the French surgeons sometimes, but questionably called the Father of Modern Surgery appeared as the great war surgeon of his time that the use of the ligature was restored and the use of the cautery and boiling oil for the control of hemorrhage was discarded. He was at first an apprentice in a barber shop but later came to Paris and served as a dresser in the Hôtel Dieu, a hospital in that city. In 1537 he became an army surgeon and in the many campaigns in which he served he had ample opportunity to study gunshot and other war wounds and wound healing. His modesty is epitomized in his very famous, old French saying, "Je l'ai pansé Dieu le guerit" (I cared for him, God cured him). He tells of his conversion from the use of the cautery, and boiling oil to the more kindly care of wounds.

In the year of our Lord 1536, Francis, the French King, sent a puissant army beyond the Alps. In the conflict there were many wounded on both sides with all sorts of weapons, but chiefly with bullets. I will tell the truth, I was not very expert at that time in matters of Chirurgery neither was I used to dress wounds made by gunshot. Now I had read in John de Vigo that wounds made by gunshot were venenate or poisoned, and that by reason of the gunpowder, wherefore for their cure it was expedient to burne or cauterize them with oyle of Elders scalding hot, with a little Treacle mixed therewith.

But for that I gave no great credit, neither to the author nor the remedy because I knew that caustics could not be poured into wounds without excessive paine. I, before I would run a hazard, determined to see whether the Chirurgeons, who went with me in the Army used any other manner of dressings to these

wounds. I observed and saw that all of them used that method of dressing which Vigo prescribes and that they filled, as full as they could, the wounds made by gunshot with tents and pledgets dipped in the scalding oyle at the first dressings, which encouraged me to doe the like to those who came to be dressed of me.

It chanced on a time that by the reason of the multitude that were hurt I wanted this oyle. Now because there were some few left to be dressed, I was forced that I might seem to want nothing and that I might not leave them undressed, to apply a digestive made of the yolk of an egg, oyle of Roses and Turpentine. I could not sleep all that night, for I was troubled in minde and the dressing of the precedent day (which I judged unfit) troubled my thoughts; and I feared that the next day I should find them dead, or at the point of death, by the poison of the wound, whom I had not dressed with the scalding oyle. Therefore I rose early in the morning, I visited my patients, and beyond expectation, I found such as I had dressed with a digestive only free from vehemence of paine to have had a good rest, and that their wounds were not inflamed nor tumified but on the contrary the others that were burnt with the scalding oyle were feverish, tormented with much paine and the parts about their wounds were swolne.

When I had many times tryed this on divers others I thought this much that neither I nor any others should ever cauterize any wounded with gunshot.

Why tourniquet control of bleeding was not mentioned earlier in the writings of surgeons is very difficult to understand, for pressure was one of the earliest methods mentioned in Egyptian and Greek texts. But apparently the first reference to its use was the introduction of the tourniquet by Morel and of the screw tourniquet by Petit, in 1674 A.D. It was successfully employed in a thigh amputation at the Hôtel Dieu in 1688. Speaking of pressure, Liston, the giant Scot surgeon, is said to have compressed the femoral vessels with his left hand while he amputated the thigh with his right.

Von Esrmarch, of Kiel University and a great military surgeon, standardized compression for surgical hemostasis when in 1873 he devised the multi fold elastic rubber bandage which has since been called by his name.

There is some uncertainty as to who first invented and used hemostatic forceps or clamps—Eugene Koeberle, an Alsatian surgeon, or Jules Pean, of Paris in the early 1870's. For many years following their introduction, hemostats were blunt-nosed affairs that crushed more than the blood vessels. It was Halsted who introduced the fine-pointed hemostats that caused so much less trauma.

These paragraphs were taken from "The Works of That Famous Chirurgeon, Ambroise Paré" Translated out of the Latin and compared with the French by Th. Johnson, II Folio, London, T. Coates & R. Young, 1634.

in the wounds. The use of the Halsted hemostats in the repair of wounds, with Halsted's silk technique, prevents unnecessary immediate, as well as later bleeding into the wound.

The bleeding tendency which in the past had been so dreaded by the surgeon, has in recent years been better understood and controlled. Blood transfusion in certain patients having prothrombin deficiency and delayed clotting time has been used effectively. In the deeply jaundiced patient, in whom the bleeding tendency after surgery is so catastrophic, the use of vitamin K for several days before operation has very largely removed this hazard and has made radical operations for the repair of common duct obstruction and for pancreatic tumors possible.

ANESTHESIA

The search for a substance or combination of substances which would relieve the pain of the surgical patient, without danger of killing him, harks back to the earliest days of surgery. Many centuries before the Christian Era the wild Scythians used hemp vapor to bring on unconsciousness. Mandragora was described by Pliny in 70 A.D., in the following words: "It has a soporific power on the faculties of those who drink it. Half a cup is the usual dose. It is drunk against serpents, and before cuttings and puncturings lest they be felt."

During the Middle Ages the sleep-producing sponge—*spongia somnifera*—was used. Hugo of Lucca, the great protagonist of primary wound healing, was quoted by his son, Theodorice, in its elaborate preparation as follows:

Take of opium and the juice of the unripe mulberry of hyoscyamus, of the juice of the hemlock, of the juice of the leaves of the mandragora, of the juice of the woody ivy, of the juice of the forest mulberry of the seeds of lettuce, of the seed of the burdock which has large and round apples, and of the water hemlock, each one ounce. Mix the whole of these together in a brazen bowl, and then in it place a new sponge and let the whole boil, and as long as the sun on the dog days, till the sponge consumes it all, and let it be boiled away in it. When there is need of it place the same sponge into warm water for an hour, and let it be applied to the nostrils till he who is to be operated upon has fallen asleep and in this state let the surgery be done. When this is finished, in order to rouse him, place another sponge, dipped in vinegar frequently to his nose or let the juice of fenigreek be squirted into his nostrils. Presently he awakens.

Such an elaborate mixture should have done something to the patient, but the fact

that it fell into disuse was good proof that it was not effective.

Before and after Hugo's time opium and alcohol were used to produce unconsciousness but if given in quantities sufficient to produce anesthesia the risk to the patient undergoing crude and shocking surgery accompanied by great loss of blood was considered too dangerous by most surgeons.

In the Trustees Room in the London Hospital, which the writer visited in 1930 there hangs a large bell, similar to the ones on our old steam locomotives. Under it is a plaque, with the following inscription: "This bell formerly hung in the front hall of the hospital. It was sounded to summon the orderlies to control the patients undergoing operation, before the discovery of anesthesia." Left to the imagination was the terror of those about to be operated upon, and the dreadful memories of the patients who had experienced the ordeal.

In "The Diary of a Surgeon in the Year 1751-1752," John Knyveton tells of his experiences as a medical student in one of the hospitals in London. His first introduction to the hospital took place one morning when he went to register in Anatomy. He says:

Doctor Urquhart was then performing an amputation of the thigh upon a porter brought in that morning from Covent Garden with a compounded fracture of the left femur the result of a kick from a horse.

The incisions of Doctor Urquhart were placed high upon the thigh. There was considerable trouble from the mass of muscle to find the arteries, which the Doctor for his own advancement wished to ligature with cords, though I learn that in such institutions it is quite common to cauterize them only with a hot iron or with boiling tar. Thus from the plunging of the patient, who seemed unable to comprehend that it was done for his own good, and the clumsiness of the Infirmary surgeon, Mr. Jamieson, ten minutes elapsed before the leg lay on the floor and much blood was shed. The patient being a poor man had few friends able to make him drunk, and so he being a well developed specimen many ropes were necessary to control his struggles. I wished to enquire what the chances of recovery were, knowing that in such a place they must surely be very small, if indeed existent at all, but after one attempt gave it up as the screams of the porter made speech impossible. The next morning I heard that the porter whose leg was removed yesterday is dead.

It was not until the beginning of the nineteenth century that any real progress was made and not much of that. In 1800 Sir Humphrey Davy the distinguished English chemist, experimented on himself with nitrous oxide gas and made the following comment: "As nitrous oxide, in its extensive operation, appears capable of destroying

physical pain. It may probably be used with advantage during surgical operations in which no great effusion of blood takes place." Note the recognition of the hazard of hemorrhage in the state of unconsciousness produced by a noxious drug.

But it was not until 1844 that this suggestion was made use of. Whether it was known to Horace Wells, a dentist in Hartford is not certain but in that year he was successful in the use of this gas in extracting teeth. Later in the same year he demonstrated its use, but not successfully, at the Massachusetts General Hospital in Boston. Following the death of one of his dental patients Wells gave up the use of the gas and probably brooding over this catastrophe, committed suicide.

The next anesthetic agent to become known as a successful one was ether. This was first described as a chemical agent in 1540 by Valerius Cordus and was named ether by Frobenius in 1730 in his description of its preparation. It had been used at times to relieve colicky pains and in "ether frolics" by students. But the first surgeon to use it as an anesthetic for an operation was Crawford W. Long, of Danielsville, Georgia. On March 30, 1842, he removed a cystic tumor from the neck of his patient, James Venable, under ether anesthesia. Unfortunately for him and medical history he did not publish a report of this and other cases, but his use of ether as an anesthetic was later verified and vouched for by the doctors in his locality during the controversy that developed over the question of priority. But as Welch said later "We cannot assign to him any influence upon the historical development of our knowledge of surgical anesthesia, or any share in its introduction to the world at large."

William T. Morton, a dentist and a former partner of Horace Wells, had learned something of the use of nitrous oxide. He later began the study of medicine while still practicing dentistry. One of his instructors was Charles T. Jackson, an able chemist, who taught him the properties of chloric ether. Morton used it in filling a tooth in July of 1844. He then learned from Jackson that sulfuric ether was also an anesthetic and he used it in extracting a deeply rooted bicuspid tooth from one of his dental patients. Later that year he called on Dr. John Collins Warren, of the Massachusetts General Hospital, to persuade him to let him give this sulfuric ether to one of Warren's surgical patients but did not disclose the name of the new

drug to be used. The operation took place in the historic Ether Amphitheater of the Massachusetts General Hospital on October 16, 1846 on what is called Ether Day. In a few minutes while the patient was unconscious, Warren removed a vascular tumor located below the jaw on the left side of the neck. As the patient was waking Warren exclaimed "Gentlemen, this is no humbug." The next day Morton administered the anesthetic for Dr. Hayward who removed a large lipoma of the shoulder. Henry J. Bigelow, the other Senior Surgeon of the hospital, improved on the method of administering ether and on November 18, 1846 he published the paper in the Boston Medical and Surgical Journal announcing to the world the successful use of ether as an anesthetic agent, which Weyr Mitchell later called "the death of pain."

Morton tried to patent sulfuric ether under the name of "letheon" and did not announce its true nature until 1847. He got into a bitter controversy with his former preceptor Jackson, over the legal rights of the discovery and later died an embittered and unhappy man.

The high character and reputation of Warren and Bigelow and the hospital where ether was first publicly demonstrated, accounted in part for the immediate acceptance of sulfuric ether as a general anesthetic. Liston in London used the new drug as an anesthetic in doing a thigh amputation in December, 1846. Syme took it up in Edinburgh in 1847 and the great Russian surgeon, Pirogoff published a manual on etherization the same year. Oliver Wendell Holmes coined the terms "anesthesia" and "anesthetic."

In 1847 the Professor of Obstetrics in Edinburgh, Sir James Y. Simpson, tried ether in his practice but on November 4 of that year he used chloroform, Liebig's discovery in delivering a woman and was so impressed with the easier administration of the drug, and the earlier recovery in other patients, that a week later he published his results in a paper entitled, "Account of a New Anesthetic Agent."

Thus with the discoveries of ether and chloroform and their universal acceptance as effective anesthetic agents, a new era in surgery was born. This was doubly significant, for it not only relieved the patient undergoing operation from the torture of pain and relieved the surgeon inflicting it, but it initiated the era of deliberate and careful surgery on human patients and freed the laboratory workers in experimental physiology and

in the wounds. The use of the Halsted hemostats in the repair of wounds, with Halsted's silk technique, prevents unnecessary immediate, as well as later bleeding into the wound.

The bleeding tendency which in the past had been so dreaded by the surgeon, has in recent years been better understood and controlled. Blood transfusion in certain patients having prothrombin deficiency and delayed clotting time has been used effectively. In the deeply jaundiced patient, in whom the bleeding tendency after surgery is so cataplectic, the use of vitamin K for several days before operation has very largely removed this hazard and has made radical operations for the repair of common duct obstruction and for pancreatic tumors possible.

ANESTHESIA

The search for a substance or combination of substances which would relieve the pain of the surgical patient, without danger of killing him, harks back to the earliest days of surgery. Many centuries before the Christian Era the wild Scythians used hemp vapor to bring on unconsciousness. Mandragora was described by Pliny in 70 A.D. in the following words: "It has a soporific power on the faculties of those who drink it. Half a cup is the usual dose. It is drunk against serpents, and before cuttings and puncturings lest they be felt."

During the Middle Ages the sleep-producing sponge—*spongia somnifera*—was used. Hugo of Lucca, the great protagonist of primary wound healing, was quoted by his son, Theodorice, in its elaborate preparation as follows:

Take of opium and the juice of the unripe mulberry of hyocyamus, of the juice of the hemlock, of the juice of the leaves of the mandragora, of the juice of the woody ivy, of the juice of the forest mulberry of the seeds of lettuce, of the seed of the burdock which has large and round apples, and of the water hemlock, each one ounce. Mix the whole of these together in a brazen bowl, and then in it place a new sponge and let the whole boil, and as long as the sun on the dog days, till the sponge consumes it all, and let it be boiled away in it. When there is need of it place the same sponge into warm water for an hour and let it be applied to the nostrils till he who is to be operated upon has fallen asleep, and in this state let the surgery be done. When this is finished, in order to rouse him, place another sponge, dipped in vinegar frequently to his nose or let the juice of fenigreek be squirted into his nostrils. Presently he awakens.

Such an elaborate mixture should have done something to the patient, but the fact

that it fell into disuse was good proof that it was not effective.

Before and after Hugo's time, opium and alcohol were used to produce unconsciousness but if given in quantities sufficient to produce anesthesia the risk to the patient undergoing crude and shocking surgery accompanied by great loss of blood, was considered too dangerous by most surgeons.

In the Trustees Room in the London Hospital, which the writer visited in 1930 there hangs a large bell similar to the ones on our old steam locomotives. Under it is a plaque, with the following inscription: "This bell formerly hung in the front hall of the hospital. It was sounded to summon the orderlies to control the patients undergoing operation, before the discovery of anesthesia." Left to the imagination was the terror of those about to be operated upon, and the dreadful memories of the patients who had experienced the ordeal.

In "The Diary of a Surgeon in the Year 1751-1752," John Knyveton tells of his experiences as a medical student in one of the hospitals in London. His first introduction to the hospital took place one morning when he went to register in Anatomy. He says:

Doctor Urquhart was then performing an amputation of the thigh upon a porter brought in that morning from Covent Garden with a compounded fracture of the left femur the result of a kick from a horse.

The incisions of Doctor Urquhart were placed high upon the thigh. There was considerable trouble from the mass of muscle to find the arteries, which the Doctor for his own advancement wished to ligature with cords, though I learn that in such institutions it is quite common to cauterize them only with a hot iron or with boiling tar. Thus from the plunging of the patient, who seemed unable to comprehend that it was done for his own good, and the clumsiness of the Infirmary surgeon, Mr. Jamie, ten minutes elapsed before the leg lay on the floor and much blood was shed. The patient being a poor man had few friends able to make him drunk, and so he being a well developed specimen many ropes were necessary to control his struggles. I wished to enquire what the chances of recovery were, knowing that in such a place they must surely be very small, if indeed existent at all, but after one attempt gave it up as the screams of the porter made speech impossible. The next morning I heard that the porter whose leg was removed yesterday is dead.

It was not until the beginning of the nineteenth century that any real progress was made, and not much of that. In 1800 Sir Humphrey Davy the distinguished English chemist, experimented on himself with nitrous oxide gas and made the following comment: "As nitrous oxide, in its extensive operation, appears capable of destroying

investigators should be to simplify rather than to elaborate, the number of drugs and methods of administering them to the patients before, during and after the operation. In recent years there has been a tendency to use too many drugs in different routes, with the result that cardiac and respiratory complications during and after operation have increased because of the polypharmacy of anesthesia.

TRAINING OF THE SURGEON

The beginnings of surgical training are indefinite and the vagaries, advances and recessions of it are many and involved but why and when they were involved are closely related to the historical relations of medicine and surgery.

In the hippocratic era of Greek medicine diseases amenable to the simple surgery of that time were few. The physicians trained in the Coan and the Cnidian Schools practiced both medicine and surgery and followed the teachings of the great master Hippocrates, and his predecessors which had evolved from the priests of the aesculapian temples. There was no superiority of physician over surgeon.

Hippocrates gave the first authentic instructions to the surgeons of historical times. He described the best position and the best lighting for the surgeon. In the oath attributed to him, he gave the rules of conduct and ethical behavior that have been the standard for both physician and surgeon ever since his time, and this is sworn to by medical students as they graduate, all over the world.

This same attitude of mutual respect for medicine and surgery was true in the Alexandrian School, where anatomy and physiology made their first advances owing to the fact that vivisection was permitted on criminals condemned to death. This same attitude of respect held true in the Greco-Roman period but rapidly deteriorated during the later centuries of the degenerate and disintegrating Roman Empire. Galen practiced both medicine and surgery but gave up his interest in the latter in favor of his polypharmacy. His studies in anatomy and physiology were done on animals and his incorrect observations as applied to the human subject remained authoritative for centuries during the next 1500 years.

During the Greek, the Greco-Roman, the Alexandrian, the Nestorian and the Arabian periods, both medical and surgical training were based essentially on the apprenticeship system. This had certain advantages, in that

the apprentice followed the master in his daily dealing with his patients, learned first hand by close observation and if the master was interested in teaching, by instruction. But it was faulty in that the apprentice saw the methods and learned the philosophy of one man only and apprenticeship made him a copier devoid of the knowledge of other teachers and discouraged independent thinking or the spirit of research because of imposed authority. So imbued with tradition and the implicit authority of men like Galen were the doctors of the Middle Ages that only rarely did an original thinker like Hugo Theodoric and Henri dare to question the didactic teachings of their predecessors.

After Galen and Soranus, both great figures in the Greco-Roman period (the latter was the first contributor to the knowledge of the diseases of women) the long night of the Dark Ages began. Notwithstanding the industry of such Byzantine compilers as Oribasius and Paul of Aegina, whose work did not reach Europe until later medicine and surgery became separated and both sank into deep desuetude. The disruption of the Roman Empire, the degradation of its morals and morale and the invasion of the wild barbarians of the north resulted in near oblivion of arts and sciences.

The steady spread of Christianity and its final acceptance by Constantine introduced a new attitude toward suffering and disease in the remnants of the Roman Empire. The crime and immorality of the later Romans and the invading northern hordes drove many of the Christians into asceticism and initiated the founding of monastic orders and their monasteries. The belief that disease and deformity were Heaven sent, to be accepted in humility as well as the worsened polypharmacy of Galen with its plague of nauseating drugs and filthy ingredients which only added to the misery of the sick, led to incantations and prayers for the treatment of the sick and, as medical art grew empty, sorcery, demonology and astrology added to the confusion.

As the monasteries increased, many of them established hostels for the sick and maimed where the religious ministrations of the monks were provided them. As time went on, the monastic orders imposed certain defining regulations on those taking orders—three of them striking in nature the long woolen cassock, the tonsure and the prohibition of the beard. The latter two required the regular shaving of the monks by the barbers who became attached to the

surgery from the stigma of inflicting pain and cruelty on vivisectioned animals. But so many surgeons of that period had been accustomed to rapid and sleight-of-hand operating that the advantages of deliberate surgery were slow in developing. However, anesthesia opened up tremendous possibilities in exploring new and hitherto inaccessible fields that previously had been considered too hazardous because of the struggles of the patient.

Since the discoveries of ether and chloroform, the evolution of the science of anesthesia has been a progressive refinement in general inhalation, local and conductive infiltration, spinal, intratracheal and, in recent years, intravenous anesthesia, with an increasing number and variety of agents singly and synergistically administered.

The invention of the hypodermic syringe and its use in administering sedatives and in anesthesia are important. Who first invented it is uncertain, but it was first introduced in Europe as a medical instrument by Francis Rynd in 1845 and in America by Fordyce Barker in 1856. Thus, we see that the decade of 1840-1850 was of the greatest significance in the battle against pain.

Nitrous oxide combined with oxygen, to combat cyanosis in inhalation anesthesia, was first employed by Andrews of Chicago in 1868. Its use was revived by Goldman in 1900. Crile popularized it in 1901 by giving a preliminary injection of scopolamine and morphine. He called this method anocic-catholization.

Following the discovery of cocaine by Anrep in 1879 and Koller's use of it in eye surgery in 1884, Halsted developed all its possibilities in nerve block conduction and local infiltration anesthesia during the next two years while he was working in New York City. Later procaine hydrochloride, discovered by Einhorn in 1905, replaced cocaine because of its less toxic effect and its freedom from the danger of addiction if used repeatedly.

Spinal anesthesia was popularized in this country by Matas in 1899 and in France by Tuffier in 1900. Stovaine, which at one time was used for its induction, has been replaced by procaine as a vehicle for the safer form of continuous, or fractional, spinal anesthesia in providing complete relaxation for operations below the diaphragm.

Rectal ether anesthesia was first successfully induced by Sutton, at the Roosevelt Hospital in New York in 1908, and refined by Gwathmey in 1913. But this form of general

anesthesia has been discontinued for the more efficient forms of inhalation spinal and intravenous anesthesia.

The use of other gases for general anesthesia, after the introduction of nitrous oxide, was started by Luckhardt of Chicago in 1922, with the highly volatile ethylene. This was more recently followed by cyclopropane. But these very volatile gases carry with them the danger of greater explosiveness and should not be used except in operating rooms especially equipped to carry off electric spark.

Intratracheal anesthesia, especially indicated for thoracic and cardiac surgery, has become increasingly useful. It was made possible by the discovery by Meltzer and Auer in 1909 at the Rockefeller Institute, of intratracheal insufflation of air through a tube passed into the trachea of experimental animals which provided "continuous respiration without respiratory movement." It has also been effectively used in operations in which any amount of blood or mucus in the mouth or throat is liable to be aspirated.

Intravenous anesthesia, induced by a variety of drugs, is the most recent development in the field of general anesthesia. Pentothal sodium is the drug most frequently used. The rapid loss of consciousness within a few seconds after the intravenous injection (usually into the median basilic vein) and the quick recovery without nausea and confusion are boons to the patient, who can be put to sleep and will awaken in his own bed and room.

One of the greatest advances in anesthesia in recent years has been the development of the residency training program in anesthesiology. Physicians trained in medicine and physiology entering this field of medical science are given intensive training in the administration of all forms of anesthetics and at the end of three years are qualified to take the examinations of the American Board of Anesthesiology. Thus, qualified physician anesthesiologists are rapidly replacing the less experienced nurses and interns who in the past had given the anesthetics in our hospitals. The many forms of anesthesia, with the many kinds of modalities used to produce them, require a knowledge of medicine and physiology for the greater safety of the patient.

Furthermore, this training stimulates those who have had it to go into the research side of anesthesiology, with the result that new vehicles and new methods of administration are increasing. One of the efforts of these

primary union. Among these were Hugo Theodoric and Henri.

In 1200 Jean Petard founded the College de Saint Come in Paris for the purpose of distinguishing the lay surgeons, wearing long robes, from the short robed barber-surgeons, illiterate in Latin. The fact that the lay surgeons were permitted to marry as compared to the long robed physicians, who were clerics, was an asset, but more important was the fact that the famous French surgeon Lanfranchi, was the leading light in the college at the time. Increased entrance requirements including two years of the study of medicine and philosophy in Latin, were followed by two years of surgery before becoming a lay surgeon of the long robe with the title of *Maitre Chirurgien Juré*.

However the persistent opposition of the physicians and the failure of the College to recognize Ambroise Paré lost them the leadership of French surgery. Furthermore, they still advocated the use of the cautery and suppuration for they followed the teachings of Guy de Chauliac. The surgical teaching was by apprenticeship under the dialectic teachers of the College. It was replaced in 1723 by the *Académie Chirurgique de Paris*.

Renaissance Surgery. Freedom of thought and the escape from dialectic authority came to Europe in the fifteenth century as the result of several factors: the navigational exploits and the discovery of the Americas; the fall of Constantinople, with the escape of Byzantine scholars with their Greek and Arabic manuscripts into Sicily and Italy; the invention of gunpowder; and, most important of all, the invention of printing, which made possible in great amount at a rapid rate the reproduction of the texts of ancient and contemporary scholars.

In the influence of freedom of thought on surgery the greatest factor was the introduction of dissection in Padua, Bologna and Florence. Equally significant was the effect on the artists' portrayal of the human form, which resulted in the transition from the primitive to the normal painting of the figure. An interesting story is told of the common meeting ground of the physicians and the artists in an apothecary shop in Florence, where the former came to have their prescriptions filled, the latter to buy their pigments. When it became known that the physicians were having dissections, their artist friends were given permission to observe the dissections, at times crowding out the doctors around the dissecting table.

Undoubtedly Leonardo da Vinci the first great anatomist of the period who was then living in Florence, was one of the first artists to join the doctors in the dissections. He is said to have made 750 anatomic sketches from the dissecting table. He may justly be called the father of modern anatomy although Vesalius, fifty years later in his epoch-making *Fabrica* had a far greater influence on surgery because of Leonardo's failure to publish his anatomic studies, many of which were not discovered until they were found in the Royal Library in Windsor Castle in 1784.

Vesalius with his *Fabrica* had a profound influence on surgery as did Harvey with his *De Motu Cordis* published in 1628, describing the circulation of the blood. For these two great works freed the surgeons from the ban of galenic doctrines which had misled them for centuries. Increased knowledge of anatomy and recurrent wars on the continent and in England, with the introduction of gunpowder resulting in a particular type of wound, increased the standing of the surgeons and the demand for their services. This was especially illustrated in the career of Ambroise Paré, the great military surgeon of the sixteenth century which has been described.

During the sixteenth and the seventeenth centuries the training of the surgeon in the hospitals and in the armies continued to be by the apprentice method. The lack of anesthesia and asepsis favored speed as the *sine qua non* for the benefit of both patient and surgeon. Little elective surgery was done. Such procedures as herniotomy and lithotomy were still delegated to a few specialists, who kept their techniques secret as well as their whereabouts after they had operated (hence their name "fugitive surgeons"). These so-called specialists had too high a mortality to have permanent offices. Most of the surgery done by the regular surgeons in the hospitals of Europe and Great Britain was of an emergency and traumatic nature. Compound fractures demanded amputation of the limbs involved and even Lister before he developed his antiseptic techniques, had a 45 per cent mortality in his amputations.

An increasing knowledge of anatomy and the recognition of its importance in surgical training resulted in the surgeon teaching the subject. All the great surgeons of the continent and Great Britain during the eighteenth and the early part of the nineteenth centuries had preceded their work in surgery as

monasteries. A later edict requiring the bloodletting of every monk five times a year increased the work of the barbers and their association with blood.

This all inevitably led to the transfer of any surgical procedure to the barbers, who later came to be known as barber surgeons. To add to the divorce of medicine from surgery and to the insistence that therapy for any illness not requiring surgery be limited to long robed cleric physicians, came the famous, infamous Papal Edict of the Council of Tours in 1163, which read "*Ecclēsia abhorret a sanguine*" (the Church shuns blood). Thus was medicine cut off from its more effective and definite form of therapy surgery which separation lasted until the nineteenth century. In England to this day the physician is called Doctor the surgeon, Mister.

Meanwhile, how did Greek medicine and philosophy survive to be reintroduced and rejuvenated into Italy and Europe through the medium of Arabic manuscripts? During the fourth century a small Christian sect in Mesopotamia, known as the Nestorians, became involved in religious controversy. In their desire to get at authentic Biblical sources, they turned to the Greek Septuagint version of the Bible. In doing so the Nestorian scholars became conversant with the Greek language and thus became acquainted with the manuscripts of Greek medicine and philosophy in the Alexandrian School and translated them into their own Syriac language. This led to their establishing a school and hospital in their city of Edessa. With the decline of the school in Alexandria the Greek sources of medicine and science remained with the Nestorians.

Because of the heresy of the Nestorians in their tenet that the Virgin Mary was the Mother of Christ but not the Mother of God, they were expelled from Edessa by the Catholic Church in 489 A.D. and were given asylum in Gondisapor a Sassanian town in southwestern Persia. Here they re-established their medical school and hospital. They translated their Syriac texts of Greek medicine and philosophy into Arabic and for the next 200 years were the repository of hippocratic and galenic medicine. The nascent Arabs, after conquering the Near and Middle East, sought the services of the Nestorian School and Hospital and were tolerant of these Christians because of their views of the Virgin Mary not being the Mother of God.

The Nestorian scholars with their trans-

lations of Greek manuscripts into Arabic assisted the Arabs in establishing medical schools and hospitals in Baghdad, Damascus and Cairo. The Mohammedan conquerors of North Africa and Spain carried Arabic medicine with them, and in Cordova in Spain they established a medical school which did not delegate surgery to menial barbers but produced Arabic and Jewish physicians who practiced surgery as well as medicine. Both medicine and surgery in the Nestorian, Arabic and Cordovan schools were taught by the apprenticeship method and in certain of the well-organized hospitals bedside teaching was conducted, especially in the Nestorian hospital in Gondisapor.

Until the eleventh century monastic medicine, as practiced by the monks and clerics, consisted in the cult of faith healing, an implicit belief in the miraculous power of the saints and their holy relics. Supernatural help came to be accepted because of the failure of medical art in sickness, especially in the great epidemics. In the eleventh century was established the first independent medical school in Italy in the town of Salerno near Naples. Monastic, Jewish, Arabic and Greek influences led to the tradition that the school was founded by the Four Masters. To the Salernitan center came the Carthaginian scholar Constantinus Africanus. He had learned Arabic and had traveled extensively. In Salerno and in the cloisters of Monte Casino he translated into Latin, from Arabic texts works of Hippocrates and Galen and the Cordovan physicians. In this way Arabic medical doctrine was introduced into Italy and France and profoundly influenced medical teaching in these countries as late as the seventeenth century. It did great harm to surgery in the advocacy of the cautery and of laudable pus in the treatment of wounds.

The establishment of universities began in the twelfth century—Paris (1100) Bologna (1158) Oxford (1167) and Padua (1222) followed by many others. But at first these institutions were under the direction and influence of the Church and therefore the medical students were under servile obedience to dogmatic authority and were forbidden to give any treatment with the hands. The fundamental error of medieval medicine was in its divorce of medicine from surgery.

In the following century however a few independent spirits appeared as physicians who did not hesitate to question authority inveighed against the suppurative treatment of wounds and preached the doctrine of

Hunter was forever observing and experimenting. He commanded his pupil Jenner of vaccination fame who was about to make a journey to the Near East, to bring him a fetus of the camel in each of the eleven months of gestation. It is not recorded by what means he secured the intact skeleton of the famous Irish giant. He became the greatest teacher in anatomy and surgery and had many famous surgeons among his pupils from Great Britain, Europe and America. England became the surgical center of the world in 1800 because of the great surgeons who had trained under John Hunter. Among them were Abernathy, Cline, Astley Cooper, Physic and the physician, Jenner.

Astley Cooper succeeded Hunter and Abernathy as the leading surgeon in London in ability and practice. He contributed soundly to anatomy and surgery. In 1804 he published a beautifully illustrated monograph on hernia. In this monumental treatise he described every known type of hernia, except one, that now is so common the postoperative ventral incisional variety.

In Scotland the most famous teachers of anatomy and surgery in the pre-listerian era were Robert Liston (1799-1847), William Fergusson (1803-1877) and James Syme (1799-1870), all three of Edinburgh. Liston was a giant physically and was the boldest and most rapid operator of his day. He was one of the first surgeons to use ether anesthesia, but only in the last year of his life. Fergusson was a great anatomist and surgeon. His dissections were superb and the surgical instruments he devised are still in use today. Syme began his work in anatomy and later became Surgeon to the Royal Infirmary in Edinburgh, where he trained Lister and gave him every opportunity to carry on his researches in inflammation.

The lecture system was the accepted method of surgical teaching. The lectures were given, as has been stated, by leading surgeons in the lecture halls of hospitals. But the instruction in anatomy in both England and Scotland at this time was given in private schools of anatomy. The failure of city authorities to permit the dissection of unclaimed bodies of inmates of hospitals and asylums resulted in grave robbery to provide cadavers for the schools of anatomy. Rival teams of grave robbers, called Resurrectionists, vied with each other to supply dissecting material for the surgeons teaching anatomy. Indeed, some of the medical

students attending the anatomy classes engaged in the hazardous business.

In the "Diary of a Surgeon in the Year 1751-1752," John Knyveton, a medical student in Dr. Urquhart's School of Anatomy, had this to say about his part in body snatching:

Nov 7 Vastly tired this morning as the result of a Hazardous Escapade from which I count myself lucky to have escaped without Grievous Harm to Life and Limb. Mr. Bloomfield did yester eve put to me that we should disinter the body of the hanged woman for the Advancement of Our Art and the Glory of Medicine, and so after some talk I agreed and we approached our worthy teacher who warning us of the Dangers—for hanging is not the least penalty one is likely to be torn to pieces by the mob should they learn of it—did then commend our Diligence and whilst saying that he would have no hand in it and would know nothing of it should it come to light, did call his huge manservant to him and gave instructions that he was to help us. So home to an early supper and so to Dr. Urquhart's to enter it by the small gate to find that the Doctor had gone out but his man and Mr. Bloomfield and Messrs. Pope and Sinclair gathered in the Anatomy room very comfortable before a fire smoking and discussing a flask of wine. So with them to pass the evening in pleasant discourse I growing somewhat drunk on the wine, very potent, and when the clocks had struck the half after twelve to collect spades and grapples and to muffle ourselves in thick cloaks. And so into the lane and to the graveyard where Mr. Pope did Belch so loud this causing Dr. Urquhart's man to swear vilely vowing that he would rather have a School of Apes to help him than such turnip heads. The grave not easy to find there being very many in a small place and the moon did come out from behind the clouds which I did not care for as we were more likely to be seen but with its aid to find where the mould had been newly turned. George Blumenfield very vehement to dig up the coffin only to find this being opened did prove to contain an old woman very foul. Then Mr. Sinclair on sitting down did find the ground gave way beneath him and so we found the hanged wench and dragged her out and put in the sack which Mr. Pope and I did then carry between us and with great haste to the lane and so to the Doctors again, all mired and sweaty. George Bloomfield did brew us a bowl of punch and we in need of such a Specific. Lord, what a business this be, this Quickening of the Awful Dead, at night when the powers of evil be abroad, amongst the tombs and the earth and the dreadful dismal Fit work only for men of Brutish Minds! Did resolve then to have no more of it, but on reflection realize that nothing is gained without labor and so as Medicine be the most noble of the Arts so the Gateway to it is correspondingly difficult and arduous to pass. Slept on a couch at the Doctors, and to home this morning at Mr. Hunt's and with him and Mrs. Hunt to church, where I heard a tolerable sermon aptly enough on the Resurrection, and wonder what his Reverence would say of my night's activities. Shall to bed early this night.

In Edinburgh, in 1827 the town went mad

teachers of anatomy and many of them continued to go from the dissecting room to their operations in the hospital without change of clothes or cleansing of their hands.

France. In France the Revolution played an important role—at first very detrimental, for all the medical faculties were abolished because of their royal affiliations. But the wars in Europe which followed made surgical training mandatory and the National Convention established what were essentially military medical schools in Paris, Montpellier and Strasbourg. Napoleon later organized the Paris School of Medicine and in 1806 he incorporated the medical school into the University of Paris as the medical branch of the institution. When medical education was again reorganized in 1830 there was started the system of the *Concours* by which the candidates for medical and surgical appointments competed against each other by taking oral examinations before elected examining bodies.

During the first half of the nineteenth century surgeons appeared in France famous for their knowledge of anatomy and their surgical skill. Special mention should be made of Larey (1760–1842) the leading military surgeon under Napoleon, one of his accomplishments being the organization of the famous ambulances to rescue and treat wounded soldiers of Dupuytren (1778–1835) who became the Head of the Department of Anatomy in the Paris Academy of Medicine at the age of twenty-three and Surgeon-in-Chief at the Hôtel Dieu when he was thirty-eight. Although hated by all his colleagues because of his haughty bearing and disdain for the work of other surgeons, he was acknowledged the greatest surgeon of his day. Lambert, Dupuytren's pupil devised the intestinal suture which insured the outer approximation of serosa to serosa, and laid the foundation for all modern gastric and intestinal surgery. Velpeau, Malgaigne and Nelaton should also be included among the prominent surgeons of this period.

Great Britain. In the early part of the eighteenth century little, if any systematized instruction in surgery was to be found. Apprenticeship with a practicing surgeon was the only available method. At St. Thomas Hospital in London, William Cheselden (1658–1732) then the leading surgeon in that city began lecturing in anatomy and surgery and in 1763 Percival Pott (1714–1788) began a series of lectures in surgery at St. Bartholomew's. In 1769 the Medical

School of Guy's Hospital was started and soon after united with the school in St. Thomas Hospital. Surgical lectures were given there and medical lectures at Guy's Hospital.

In Scotland a medical school was organized in Edinburgh by John Munro. His son, Alexander Munro (1697–1767) started the famous School of Anatomy in Edinburgh. He was succeeded by his son and grandson, so that this Chair of Anatomy was held by the Munros for 140 years.

The most famous of British surgeons of this period was John Hunter (1728–1793) the founder of experimental surgery and experimental pathology who ranks with Paré and Lister as one of the three most influential surgeons of all time. He was a man of unbounded energy insatiable curiosity and a determination to know the why and the how of surgical lesions as well as natural phenomena. As an uncouth youth he was brought to London from his home in Scotland by his older brother William, then famous for his School of Anatomy in Windmill Street. John became fascinated with the study of anatomy which later culminated in the Hunterian Museum of some 13 000 biologic specimens, most of them prepared by him and many of them still to be seen in the Royal College of Surgeons in London.

John Hunter studied surgery with Cheselden and Pott, and later was appointed Surgeon to St. George's Hospital. He developed surgical pathology into a live and essential discipline. His studies of inflammation, shock, syphilis and teeth were unique. But his most outstanding contribution was in the pathology and treatment of aneurysm. From an experiment which he performed by tying the external carotid artery of a deer he found that the growing antler on the side of the ligated artery which at first had turned cold, later regained its warmth and continued to grow. When he autopsied the buck he found the ligature secure but saw that the blood supply to the antler was by way of small collateral arteries. "Oho," he said in his notes, "I see that under the stimulus of necessity the smaller arterial channels greatly increase in size to do the work of the larger. I must remember that." This he did, for he changed the old Greek Antyllus operation of ligating the artery above and below the aneurysm to the technique of ligating the artery well above the aneurysm and by passing the sac, with the development of compensatory collateral vessels.

only survived but lived to be seventy-eight without any recurrence of her trouble.

During the next hundred years especially after the discovery of anesthesia, elective surgery increased. Attention was concentrated on the development of new techniques and new instruments. In the late 1870's and early 1880's, the treatment of abdominal acute infections was initiated by the attack on appendiceal abscess—then called perityphlitis. Surgery of the gallbladder, the stomach and the intestinal tract was beginning. The radical attack on cancer was initiated by the radical mastectomy of Halsted and Wills Meyer in 1882. Billroth, in Berlin performed the first partial gastrectomy for cancer in 1883.

In the medical schools of Harvard University, Columbia University and the University of Pennsylvania, medicine and surgery were taught by the lecture system and little bedside teaching was given before 1890. In some of the schools, private quizzes to prepare men for the competitive hospital examinations were attended far more regularly than the lectures in the medical schools.

Much of the surgical teaching during the latter part of the last and the early part of the present century was given from the operating amphitheater where the student saw backs and blood but got little worth while instruction. This led to showmanship on the part of the leading surgeons and a tendency to continue the emphasis on speed in operating, which, of course, appealed to the immature and uncritical students. At this time there began a shift from the emphasis on anatomy to the study of surgical pathology in preparation for surgery. This was to be followed in the early years of the present century by the emphasis on physiology as the most important of these three disciplines as the approach to original and constructive surgery.

In or about 1880 listerism as well as the use of anesthesia had so increased the elective surgery in the hospitals of the United States that internship in surgery became well established. Before this time interns served for both medicine and surgery. The surgical internships were from twelve to sixteen months in most hospitals, with a period of four to six months of houseship during which time the intern was given operative work, the amount depending upon the good will of the attending surgeons with whom he had been working. In the larger centers like New York City the competition for appointment to the surgical services was

determined largely by the amount of operative work that was given to the house surgeon. This resulted in a vicious circle, for the hospitals desiring the best men from the medical schools gave an increasing amount of operative work to the relatively inexperienced intern who when he left the hospital went into general practice, doing little if any surgery thereafter unless he later became attached to a surgical service in one of the hospitals.

The change from this short term and wasteful surgical training to the long term and sound residency training was initiated by William S. Halsted. He had studied in the best German clinics before he returned to New York City where for a period of four years he was known as a brilliant young surgeon imbued with the newer aseptic techniques. A most unfortunate, but entirely innocent experience with the study of cocaine as an agent for producing local and nerve block anesthesia resulted in his having to drop out of surgery for a period of four years. But this enforced withdrawal from surgical activity gave him time for contemplation and a reappraisal of his ideas of surgical techniques and of surgical training. William H. Welch, his great friend who rescued him from his innocent acquired addiction and who appreciated his great promise as a surgeon took him to Baltimore to be his co-worker in the newly organized Johns Hopkins Medical School. Welch had been the pathologist at the Bellevue Hospital in New York before he was chosen to be dean of the new school in Baltimore.

Halsted had seen and appreciated the advantages of the long term training in the German clinics but also was aware of their failure to give this training to more qualified young surgeons. When he was chosen to take charge of the surgical service at Johns Hopkins in 1889 he appointed residents from qualified surgical interns, who served from four to six years, with the appointment to the chief residency for a period of one or more years. This resulted in such well-trained surgeons, after such wide experience in operative and experimental surgery that his residents were appointed to chairs of surgery in the medical schools of the country.

However for many years the Halsted residency program was not favorably received in the surgical clinics of the country because of the antagonism of the younger attendings and the surgical interns, who feared that the residency system would deprive them of the operative experience which they had

over the discovery that two of the grave robbers supplying Dr. Knox's School of Anatomy had murdered a number of people by smothering them and had sold them as freshly exhumed bodies to the School. In London, in 1831 two grave robbers were arrested. It developed that they had murdered thirty to sixty people by giving them rum and laudanum and had sold their bodies for dissection. Both were convicted and publicly hanged. Parliament then passed a bill regulating the supply of unclaimed bodies for purposes of anatomizing. Grave robbery thus came to an end.

During this period of the Resurrectionists the surgeons and the medical students came from the dissecting rooms to the hospitals to take part in the operations and deliveries. Antiseptic precautions were unknown and the varying states of putrefaction of the cadavers, together with the failure to change clothes and wash hands, made hospital infections an unbelievable nightmare. In some of these institutions the mortality reached 80 per cent of the surgical and obstetrical admissions.

Germany The best known surgeons in Germany during this period were Conrad Langenbeck (1776-1851) von Graefe (1787-1840) Dieffenbach (1792-1847) Stromeyer (1804-1875) and Bernhard von Langenbeck (1810-1887). The elder Langenbeck was Professor of Anatomy and Surgery at Göttingen and Surgeon General of the Hanoverian Army. He belonged to the sleight-of-hand type of surgeon and is said to have amputated a shoulder while a colleague present was taking a pinch of snuff. Von Graefe was the Professor of Surgery at the University of Berlin and is considered the founder of modern plastic surgery. Dieffenbach succeeded von Graefe. He was the first to cut the eye muscles for strabismus and was one of the first to operate on vesicovaginal fistula. He was a cultured scholar as well as an able surgeon and teacher.

Stromeyer was one of the founders of orthopedic surgery and was considered the father of military surgery in Germany. He was a poet as well as a great surgeon. Bernhard von Langenbeck, the nephew of Conrad, became the greatest surgeon and clinical teacher of this period in Germany. Under his tutelage the most famous surgeons of the past fifty years in Germany appeared. Two of his greatest accomplishments were the founding of the German Society of Surgery

and the editing of the great surgical journal of that day the *Langenbeck Archiv*.

Long term training in surgery was started in Germany in a pattern of its own, and is continued to the present in much the same form. All students on graduating from the medical school are required to take a year of internship, most of them preferring the rotating type. After that they can become assistants in surgical clinics or as at present, they may wait out a year as volunteers. This volunteer system is necessary in Germany at the present time because of the great demand and the relatively small supply of such positions. This assistantship is somewhat comparable to the assistant residency in the United States. The assistant can advance to the position of first assistant in four or five years, but few reach this promotion. After this, the first assistant may be promoted to become an *Oberarzt*, comparable to the chief resident in our clinics. But unlike him he may serve in that capacity for a period of ten to fifteen years, waiting for an appointment as the head of one of the surgical clinics. In the meantime, he assists the professor who as the *Chefchirurg* is the omniscient one not to be questioned in any way and who does practically all the operating.

The same method of surgical training is followed in most of the European clinics as in Germany with individual modifications in each country.

America Surgical training in the United States, until the latter part of the nineteenth century, was by apprenticeship. A few of the ambitious young surgeons of the period under discussion went to Paris, London and Edinburgh for further training. Especially notable were Physic and Dorsey of Philadelphia who studied under John Hunter in London, and Valentine Mott of New York who worked with Astley Cooper and later became famous for his original work in vascular surgery. Ephraim McDowell of Danville, Kentucky studied under John Bell in Edinburgh. In 1809 he performed the first ovariectomy in abdominal surgery on a forty-seven-year old woman, who had come to his office on horseback, a distance of 21 miles. At the end of the ride she had developed a decubitus of the lower abdomen as a result of the pressure of the pommel of the saddle. McDowell operated while a group of the citizens of Danville waited with loaded guns to operate on him if the woman did not survive the ordeal. The woman not

agers, and now the public, that surgery should be done by properly trained and experienced men. This has made a great difference in the caliber of the surgeons appointed to hospital staffs throughout the country.

It will be recalled that the divorce of medicine and surgery in the Middle Ages resulted in chaos to both. In the present era, although practiced by different groups, the two are so closely related that they cannot be separated as in the past. In our medical schools the etiology, pathology and diagnosis of disease are studied by all the students, as well as the indications for the proper therapy. Many of these diseases require medical therapy in some stages of their development, surgery in others.

The close association of the medical and surgical services in our hospitals today and the fact that many of the interns in these hospitals serve on both services break down any of the old barriers between these two disciplines. In many of the courses on the so-called middle ground diseases, teachers from both departments take part, and the teamwork of both groups in the combined clinics on many of these syndromes, and in the follow-up of them makes the physicians and the surgeons speak the same language and there are no miracles among friends.

It is now an accepted fact that internal medicine and surgery are but two branches of therapy in the science of medicine.

been having in the hospitals where the short term training had prevailed. It was not until the 1920's, when the necessity for long term training became mandatory because of the advantages to the patients and to the attending staff in the surgical clinics, that residencies were started and soon became the recognized and sound method of training those who wished to prepare for major and radical surgery.

Another change in the policy of surgical teaching in the undergraduate curriculum in the medical schools was the recognition of the fact that training in the operative techniques is a graduate function that has to be done in the hospitals in the surgical internship and residency periods. Teaching the symptoms and signs of lesions requiring surgery was emphasized rather than the techniques of treating them and replaced the lectures which formerly were given in the courses in the surgical specialties. With the crowded curriculum in the medical schools this was essential and sound.

This establishment of residency training in the university surgical clinics in the United States may be justly considered the greatest advance in surgical training in the history of surgery. For as it is now done in these, and the best municipal hospitals of the country the residents are given the finest operative training and in their last year independent operative work. They are given increasing responsibility as they advance in their residencies, for the conduct of the surgical services. This makes them far more experienced in assisting the attending surgeons as well as in the care of the patients, for they live in the hospital and are on call at all times to prevent complications or to treat them promptly if they should occur. Under this system patients get far more intelligent and constant care than was the case in the old days. Furthermore, under this plan far more surgeons receive adequate training and experience than is the case in European clinics and they are given the opportunity of independent thinking without the domination and dictation of the omniscient *Gehirnrat*.

So able and proficient did this young generation of long term trained residents become, that it became obvious to the leading surgeons of the country that these surgeons should be given recognition. Previously qualifying boards in other specialties had been organized under the American Medical Association. In 1937 at the instigation of the American Surgical Association, the Quali-

fying Board in General Surgery was organized and began giving written and oral examinations to surgeons who had completed their long term residency training. If these surgeons passed the examinations, they were given certificates of proficiency with the understanding that they would limit their work to general surgery.

So outstanding was the ability of these qualified surgeons during World War II, as compared to the work of the medical officers with short term intern training, that the young officers who had had only nine months of internship were convinced that residency training was essential for any man going into surgery. This conviction was strengthened when the Veterans Administration, properly reorganized after the war required such residency training for appointment to the position of attending surgeon in the Veterans Hospitals.

Other developments in the surgical field in this country have added greatly to the education and training of the surgeon. The surgical journals that have been published have become increasingly competent in publishing the original studies of the surgeons and in eliminating much of the useless, repetitious and uncritical compilation of clinical material which in the past filled the pages of the journals.

The annual and semiannual meetings of the national surgical associations and small travel clubs provide the very valuable means for our surgeons to see and to hear reports of the work of leading surgical authorities. This is especially true of the meetings of the Congress of the American College of Surgeons with the presentation of papers and the panel discussions as well as exhibits of laboratory and technical procedures. This is also true of the Surgical Section Meetings of the Annual Meetings of the American Medical Association. Examples of how effective surgical journals and meetings have been may be cited in the astonishing advances which have been made in the field of thoracic and cardiovascular surgery and the spread of the information regarding the techniques of these procedures throughout the clinics of this country. Within a period of two or three years the new methods have come into use in all the leading surgical clinics.

One of the most important results of the residency training program, together with the qualification of the residents who pass the examinations of the qualifying board, is the recognition by hospital boards of man-

hemorrhage retroperitoneal bleeding from multiple fractures damage to great vessels contusions of the heart and lungs or injuries to the cerebrum and cord.

Delays in diagnosis and treatment may lead to disaster. The newspapers term, "death from internal injuries," carries with it an air of helplessness, or hopeless, mystery which draws from the reader an expression of forgiveness. When the victim of an accident reaches the emergency room, it is regrettable and shameful if every effort including surgical exploration is not made to determine the nature and extent of his injuries.

Before considering the local physiologic changes within the wound, one should be familiar with the general physiologic effects of surgical trauma or violence upon the body as a whole, and the significance of the responses resulting from altered endocrine activities stimulated by mechanical chemical and thermal injuries. These are normal metabolic responses and are usually characterized by a temporary elevation of temperature and pulse rate, a loss of nitrogen from the body for three to seven days followed by positive nitrogen balance, and a loss of potassium for the first two to five days after injury followed by retention of potassium. A transient decrease in the excretion of urine occurs and a diminished urinary excretion of sodium develops for two to five days after injury following which urinary excretion of sodium increases. A loss of weight occurs. Resumption of caloric and nitrogen intake and a positive balance are requirements important to recovery. A drop in circulating eosinophils and an increase in the excretion of hormone products are the result of an accompanying endocrine readjustment between the pituitary gland and the adrenal cortex.

The histologists and pathologists have provided additional terms to help us in our understanding of wound healing. If a wound is a break in the continuity of tissue, healing is the restoration of continuity of tissue. The factors which bring it about constitute a reaction to injury and the whole process is called inflammation.

The process of wound healing may be divided into three phases *initial or lag phase the phase of fibroplasia and the phase of contraction of the scar*.

The use of these terms is not essential to the understanding of wound healing. For example the lag phase was noted from observations made upon experimental ani-

mals in the laboratory. In measuring the strength of a wound following its surgical repair there was on the first day a measurable strength provided by the retracted clot which joined the two cut surfaces. The healing process which went on for the next three or four days not only failed to strengthen the wound but caused it to become weaker than on the first day. When the growing strength of wounded tissue is considered, there is a measurable lag which does not start to improve until about the sixth postoperative day. This phase has clinical significance in the management of wounds and it should not be interpreted as one in which the tissues are inactive.

The phase of fibroplasia was also determined from laboratory observations. Histopathologic studies showed fibroplasia to begin soon after the injury and to increase rapidly throughout the lag period until it predominated in its own intermediate phase of fibroplasia.

Its occurrence during the lag period is responsible for termination of that phase of healing. The extracellular ground substance which is produced by the fibroblasts becomes converted into collagen which upon contraction begins to terminate the phase of fibroplasia. Again there is considerable overlap in the phases of healing. While fibroplasia alone contributes some strength to the wound beyond that of the original clot retraction, conditions must be right for the chemical changes to convert the extracellular ground substances into the finished product of collagen before satisfactory strength is achieved or maintained.

Preparations for the phase of contraction of scar go right along with the entire phase of fibroplasia. Under normal conditions, it accelerates at a rapid pace and starts to become a clinical entity between ten and fourteen days. From then on, scar formation and contraction are the major features of wound healing, continuing for many weeks as the fibroblasts steadily disappear from the scar. The phases of wound healing should not be looked upon as separate acts in a play but as scenes which are taking place almost simultaneously.

The process of healing is initiated by the injury to the tissue cells, and it is essentially the same whether the injury is a planned operative procedure or an accidental wound of violence.

The force causing the injury may create a variety of changes in the tissues, sometimes dividing them as evenly as one could wish a

PHYSIOLOGY OF WOUND HEALING

By WILLIAM A. ALTEMEIER M.D
and JEAN M. STEVENSON M.D

WILLIAM ARTHUR ALTEMEIER, a Cincinnati by birth is a product of his home city's educational institutions. He has progressed through the ranks of the department of surgery of the University of Cincinnati College of Medicine to become professor and chairman of the department. He is director of the surgical bacteriologic laboratory and his interests have extended to the detailed study of tissue healing. His contributions to surgical literature have been fundamental.

JEAN MOORHEAD STEVENSON also an Ohioan was educated in medicine and trained in surgery at the University of Cincinnati, where he is Associate Professor of Surgery. His interest in the healing of wounds was stimulated by the late Mont Reid.

Wound healing is an inherent physiologic process of animals for the repair of their injured tissues. A knowledge of the mechanism of wound healing and of the local and general factors which affect it is obviously essential. Unfortunately this process is not the same for all tissues. Highly specialized tissues such as those of the kidney and brain, repair injuries to themselves by the formation of granulation tissue and ultimate replacement by scar tissue. Less specialized tissues may be regenerated in a form almost identical with their normal structure.

A wound is generally defined as the solution, or break in continuity of tissue resulting from mechanical force. Mechanical force denotes the characteristic of motion of the object which produces the wound. Thermal, bacterial and chemical agents are also capa-

ble of producing wounds, but the character of the injuries produced and the policies of management and repair are so different that they demand separate consideration.

Wounds may be external or internal, obvious or obscure. Any tissue in the body may be involved in a wound while the skin itself may remain intact. This concept of a wound is frequently overlooked by those who generally consider wounds to be external manifestations of injuries. The student finds it difficult to examine carefully a patient for an explanation of his complaints when there is no evidence of external blood loss or gross distortion of contour resulting from major breaks in the skeleton. He must be on his guard so as not to miss deep injuries such as rupture of the spleen, liver bladder or kidney, a torn hollow viscus, mesenteric

By phagocytic and enzymatic action, their work precedes the regenerative growth of tissue.

The debris in closed wounds must be carried away by the circulation while in open wounds much of it may escape to the surface in the form of an exudate or pus. Proteolytic enzymes dissolve dead cells either within or outside of the phagocytic cells. Sometimes bacterial enzymes also assist in this work, but help from bacteria is usually not appreciated because their enzymes may also work on living cells.

Proliferation and migration of endothelial capillary buds into the blood clot begin early and can be seen microscopically on the second day. The fibrin trabeculae in the coagulated blood clot form early and contribute the framework for the subsequent in-growth of capillaries and fibroblasts from the wound edges. The capillaries supply the nutrition and reserves for the reparative elements and carry away their by-products of metabolism and catabolism.

The early product of fibroblastic and endothelial cell growth is called *granulation tissue* and this tissue is essential to the healing of every wound. There is a tendency to disregard serious consideration of granulation tissue when it lies in a vertical plane between the edges of a sutured wound. The regeneration accounting for this tissue is exactly alike in open and closed wounds, but the very name which was adopted to describe it was the result of observations made in open flat wounds. The same volume of granulation tissue in a closed, or concealed, wound can give rise to future complications.

The fibroblasts secrete and deposit in the granulation tissue a protein, collagen, which produces strength to the bond between the wound's surfaces. The old collagenous connective tissue at the periphery of the wound becomes swollen and fuses with the new collagenous tissue by continuity of cell growth and re-formation of collagen fibrils. The deposition of fibrils begins in the periphery of a wound which may still contain clot and manifest phagocytic activity but it does not occur in any portion of a wound containing ing debris or necrotic tissue.

Clinical signs of the formation of granulation tissue may remain hidden in wounds filled with clot, but one can detect its presence about the fourth or fifth day in flat wounds containing little clot. Steadily the granulation tissue spreads and thickens until it is no longer possible to recognize the previously exposed tissues, such as muscle, fat or

fascia. The granules which characterize the tissue represent the presence of especially robust capillaries near the surface which sponsor the volcano-like eruption of tufts of fibroblasts and leukocytes.

As a membrane for a surface wound, or as a retaining wall about an inflammatory process granulation tissue is a great protective device. Although this was known for many generations, it was Billroth who pointed out its protective qualities against bacteria and their toxins in 1865. Billroth demonstrated that signs of tissue destruction or systemic toxicity did not occur after the application of contaminants to the granulating surface of wounds when free drainage had been established. The necessity for strict observance of aseptic technique in the management of granulating wounds may be questioned, but the answer is easily found.

Contaminants will grow on granulation tissue and can cause trouble. *Pseudomonas aeruginosa* usually nonpathogenic for man, has a voracious appetite for newly planted skin grafts. The proteolytic enzymes and other toxins of such bacteria as the hemolytic staphylococcus and the hemolytic streptococcus may be absorbed to produce signs of toxemia. These and other organisms are capable of surviving for years in the cicatrix of a wound which has long been covered by epithelium and may become reactivated by another subsequent injury to the same area. It is wise to remember that some granulating wounds which appear to be innocent may harbor organisms potentially pathogenic for those who dress the wound or for patients near by with open wounds.

It is not unusual in the change of dressings covering wounds to find them sticking to the granulation tissue and to see the tissue bleed with removal of the dressings. A temporary rise in the temperature curve may follow.

If removal of the dressings is postponed, they may become putrid and the patient may become toxic. Retained moisture and proteolytic enzymes may macerate and even attack the surrounding normal skin. When the by-products of cell growth and bacterial action cannot escape, some of them are absorbed and can produce a systemic response approaching that of an undrained wound.

The wall of granulation tissue about an undrained abscess may keep the process localized. It does not, however, stop the absorption of toxic products from the abscess as long as it is under pressure, as is shown by the febrile response before and after the drainage of any abscess.

sharp scalpel at other times completely tearing pieces of tissues away from the body or leaving them hanging in shreds along the edges of the wound. A blow may only bruise the skin but mince the deeper fat and muscle or fragment the underlying bone. A glancing blow may do little more than brush away the top layer of skin. When tissues are divided the contraction of muscle and elastic fibers tends to widen the gap and the continuity of the vascular systems for the blood and lymph is affected. Fluids escape through the breaks into the wound's cavity and to the surface, if a connection exists. The quantity and rate of blood loss is determined by the number, character and sizes of the vessels injured, the nature of the hole in the vessel, the clotting properties of the blood, the types of the tissues injured, the activity of the patient, and the patient's blood pressure.

An effective clotting mechanism is essential to the preservation of life. If bleeding into a wound could be prevented, one would observe that lymph and plasma would fill the wound with clot and serve the processes of healing equally as well as blood. Some of the fluid which collects in a fresh wound comes from lymph and plasma but the material is always colored with red blood cells from damaged vessels or an old blood clot. When the fluid which has collected in closed wounds is aspirated repeatedly with a needle and syringe it will become progressively clearer until it resembles pure lymph or plasma.

When a wound becomes coated with coagulum the bleeding stops and further loss of fluid will consist of plasma or lymph whose flow to the surface is essential to the continued growth of the granulation tissue. The coagulum which forms on granulation tissue comes from the clotting of plasma and lymph while the serum becomes a by product of this reaction.

The formation and retraction of the clot stop the bleeding unless the force of the stream is too great. Completely divided vessels will contract and retract, favoring stoppage with clot. Partially divided vessels cannot retract and the lacerated lumen is held open, prolonging hemorrhage or predisposing to secondary bleeding. Severed capillaries retract and contract just like larger vessels. In experimental studies they have never been seen to clot, but they may open up and resume function under more favorable conditions.

A clot which retracts poorly is soft and inefficient. Bleeding may recur in such wounds

after displacement of the clot during dressing. A firmly retracted clot efficiently stops bleeding, supports the wound's edges and gives the wound what little strength it has in the early hours or days of healing. This clot also provides better footing for the cellular invasion beginning at the periphery of the wound.

Excessive bleeding and clotting are undesirable and may become hazardous. A large clot cannot move itself and it has little resistance to bacterial action. Its organization delays wound healing, increases the inflammatory reaction and leaves behind an abundance of troublesome scar. In some instances, the clot's center will become liquefied, or infected before it can be organized and will then require removal by aspiration or incisional drainage. In sutured wounds large clots may create pressure, favor tissue necrosis, separate wound edges, increase morbidity or impair function.

Another effect of injury is the stimulation of an increased blood flow to the area. The resultant vasodilatation causes the skin to turn pink and produces an increase in local heat. This response is immediate and it continues throughout the healing of the wound.

Immediately after trauma, the pH of the tissues at the site of injury is shifted toward the acid side. In the deeper tissues, a pH of approximately 6.4 will develop and continue during a considerable period of the healing of the wound. This is believed to be one of the factors which produces hyperemia, capillary permeability and local swelling due to an increase of intracellular and extracellular fluids. The swelling which develops is related to the type and severity of the trauma, but all soft tissues do not respond similarly. Rather insignificant injuries about the eyes and lips produce much more swelling than do similar injuries of the trunk or extremities.

Marked swelling delays the process of healing because it restricts circulation, retards local metabolism, produces necrosis of cells and favors bacterial growth. Mild localized swelling, however, is considered to be beneficial. Capillary permeability increases, the channels widen, the rate of flow decreases and the leukocytic cellular elements collect along the capillary walls and make their way through the walls into the tissues. Leukocytes, histiocytes and macrophages arrive in the injured area to aid in the removal of debris and the defense against bacteria. The phagocytic cells, instead of circulating through the clot, apparently dissolve their way into it by enzymatic action.

By phagocytic and enzymatic action, their work precedes the regenerative growth of tissue.

The debris in closed wounds must be carried away by the circulation while in open wounds much of it may escape to the surface in the form of an exudate or pus. Proteolytic enzymes dissolve dead cells either within or outside of the phagocytic cells. Sometimes bacterial enzymes also assist in this work, but help from bacteria is usually not appreciated because their enzymes may also work on living cells.

Proliferation and migration of endothelial capillary buds into the blood clot begin early and can be seen microscopically on the second day. The fibrin trabeculae in the coagulated blood clot form early and contribute the framework for the subsequent in-growth of capillaries and fibroblasts from the wound edges. The capillaries supply the nutrition and reserves for the reparative elements and carry away their by-products of metabolism and catabolism.

The early product of fibroblastic and endothelial cell growth is called *granulation tissue* and this tissue is essential to the healing of every wound. There is a tendency to disregard serious consideration of granulation tissue when it lies in a vertical plane between the edges of a sutured wound. The regeneration accounting for this tissue is exactly alike in open and closed wounds, but the very name which was adopted to describe it was the result of observations made in open flat wounds. The same volume of granulation tissue in a closed, or concealed, wound can give rise to future complications.

The fibroblasts secrete and deposit in the granulation tissue a protein collagen, which produces strength to the bond between the wound surfaces. The old collagenous connective tissue at the periphery of the wound becomes swollen and fuses with the new collagenous tissue by continuity of cell growth and re-formation of collagen fibrils. The deposition of fibrils begins in the periphery of a wound, which may still contain clot and manifest phagocytic activity but it does not occur in any portion of a wound containing debris or necrotic tissue.

Clinical signs of the formation of granulation tissue may remain hidden in wounds filled with clot, but one can detect its presence about the fourth or fifth day in flat wounds containing little clot. Steadily the granulation tissue spreads and thickens until it is no longer possible to recognize the previously exposed tissues, such as muscle fat or

fascia. The granules which characterize the tissue represent the presence of especially robust capillaries near the surface which sponsor the volcano-like eruption of tufts of fibroblasts and leukocytes.

As a membrane for a surface wound, or as a retaining wall about an inflammatory process granulation tissue is a great protective device. Although this was known for many generations, it was Billroth who pointed out its protective qualities against bacteria and their toxins in 1865. Billroth demonstrated that signs of tissue destruction or systemic toxicity did not occur after the application of contaminants to the granulating surface of wounds when free drainage had been established. The necessity for strict observance of aseptic technique in the management of granulating wounds may be questioned, but the answer is easily found.

Contaminants will grow on granulation tissue and can cause trouble. *Pseudomonas aeruginosa* usually nonpathogenic for man, has a voracious appetite for newly planted skin grafts. The proteolytic enzymes and other toxins of such bacteria as the hemolytic staphylococcus and the hemolytic streptococcus may be absorbed to produce signs of toxemia. These and other organisms are capable of surviving for years in the cicatrix of a wound which has long been covered by epithelium and may become reactivated by another subsequent injury to the same area. It is wise to remember that some granulating wounds which appear to be innocent may harbor organisms potentially pathogenic for those who dress the wound or for patients near by with open wounds.

It is not unusual in the change of dressings covering wounds to find them sticking to the granulation tissue and to see the tissue bleed with removal of the dressings. A temporary rise in the temperature curve may follow.

If removal of the dressings is postponed, they may become putrid and the patient may become toxic. Retained moisture and proteolytic enzymes may macerate and even attack the surrounding normal skin. When the by-products of cell growth and bacterial action cannot escape, some of them are absorbed and can produce a systemic response approaching that of an undrained wound.

The wall of granulation tissue about an undrained abscess may keep the process localized. It does not, however, stop the absorption of toxic products from the abscess as long as it is under pressure, as is shown by the febrile response before and after the drainage of any abscess.

sharp scalpel at other times completely tearing pieces of tissues away from the body or leaving them hanging in shreds along the edges of the wound. A blow may only bruise the skin but mince the deeper fat and muscle or fragment the underlying bone. A glancing blow may do little more than brush away the top layer of skin. When tissues are divided the contraction of muscle and elastic fibers tends to widen the gap and the continuity of the vascular systems for the blood and lymph is affected. Fluids escape through the breaks into the wound's cavity and to the surface, if a connection exists. The quantity and rate of blood loss is determined by the number, character and sizes of the vessels injured, the nature of the hole in the vessel, the clotting properties of the blood, the types of the tissues injured, the activity of the patient, and the patient's blood pressure.

An effective clotting mechanism is essential to the preservation of life. If bleeding into a wound could be prevented, one would observe that lymph and plasma would fill the wound with clot and serve the processes of healing equally as well as blood. Some of the fluid which collects in a fresh wound comes from lymph and plasma but the material is always colored with red blood cells from damaged vessels or an old blood clot. When the fluid which has collected in closed wounds is aspirated repeatedly with a needle and syringe, it will become progressively clearer until it resembles pure lymph or plasma.

When a wound becomes coated with coagulum the bleeding stops and further loss of fluid will consist of plasma or lymph whose flow to the surface is essential to the continued growth of the granulation tissue. The coagulum which forms on granulation tissue comes from the clotting of plasma and lymph while the serum becomes a by product of this reaction.

The formation and retraction of the clot stop the bleeding unless the force of the stream is too great. Completely divided vessels will contract and retract, favoring stoppage with clot. Partially divided vessels cannot retract and the lacerated lumen is held open, prolonging hemorrhage or predisposing to secondary bleeding. Severed capillaries retract and contract just like larger vessels. In experimental studies, they have never been seen to clot, but they may open up and resume function under more favorable conditions.

A clot which retracts poorly is soft and inefficient. Bleeding may recur in such wounds

after displacement of the clot during dressing. A firmly retracted clot efficiently stops bleeding, supports the wound's edges and gives the wound what little strength it has in the early hours or days of healing. This clot also provides better footing for the cellular invasion beginning at the periphery of the wound.

Excessive bleeding and clotting are undesirable and may become hazardous. A large clot cannot move itself and it has little resistance to bacterial action. Its organization delays wound healing, increases the inflammatory reaction and leaves behind an abundance of troublesome scar. In some instances, the clot's center will become liquefied, or infected before it can be organized and will then require removal by aspiration or incisional drainage. In sutured wounds, large clots may create pressure, favor tissue necrosis, separate wound edges, increase morbidity or impair function.

Another effect of injury is the stimulation of an increased blood flow to the area. The resultant vasodilatation causes the skin to turn pink and produces an increase in local heat. This response is immediate and it continues throughout the healing of the wound.

Immediately after trauma, the pH of the tissues at the site of injury is shifted toward the acid side. In the deeper tissues a pH of approximately 6.4 will develop and continue during a considerable period of the healing of the wound. This is believed to be one of the factors which produces hyperemia, capillary permeability and local swelling due to an increase of intracellular and extracellular fluids. The swelling which develops is related to the type and severity of the trauma, but all soft tissues do not respond similarly. Rather insignificant injuries about the eyes and lips produce much more swelling than do similar injuries of the trunk or extremities.

Marked swelling delays the process of healing because it restricts circulation, retards local metabolism, produces necrosis of cells and favors bacterial growth. Mild localized swelling, however, is considered to be beneficial. Capillary permeability increases, the channels widen, the rate of flow decreases and the leukocytic cellular elements collect along the capillary walls and make their ways through the walls into the tissues. Leukocytes, histiocytes and macrophages arrive in the injured area to aid in the removal of debris and the defense against bacteria. The phagocytic cells, instead of circulating through the clot, apparently dissolve their way into it by enzymatic action.

ritability. Pain is frequently present and the wound may have a putrid odor.

Unhealthy granulation tissue is prone to occur in wounds which are chronically infected or in old wounds having a densely scarred base in which the period for response to normal healing has passed. Any ischemia resulting from scarring of the base or other causes produces gray yellowish pink or cyanotic granulations. They are hard and fixed from fibroblastic growth and the formation of cicatrix. The surrounding tissues are likewise hard and scarred. Pigmentation of the neighboring skin is common. The wound is unusually sensitive owing to nerve regeneration and a lowered pH resulting from the stasis. Epithelization and generation of new granulations are at a standstill.

The aging process of granulation tissue is largely the function of the fibroblasts. They produce the homogeneous ectoplasm and fibrils which have attachments to the cell bodies. Their arrangement seems to be affected by the lines of stress and their appearance, in increasing quantities, coincides with the initial and continued shrinkage of the wound. This is not readily detected in the week-old wound but the development of the collagenous fibrils imparts to the wound a steadily increasing tensile strength for the ensuing several weeks. Because a wound shows no gain in tensile strength until about the sixth day after injury the term *lag period* is applied to this interval in the healing of a wound. It must be remembered that only because the tissues have been teeming with activity is an increase in tensile strength possible in such a short time. After the sixth day the tensile strength increases rapidly.

The end result of this process of fibroplasia is scar or cicatricial tissue which accounts for the tensile strength, hardening and toughening of the wound as well as for occasional undesirable sequelae.

Scar tissue however is seldom as resistant to tension as is normal tissue. When present in excess it may become a point subject to abnormal stretching or herniation. An excessive force may stretch the scar into a state of disruption (Fig. 3).

Whenever possible the surgeon directs the process of healing along lines favoring the minimal amount of scar tissue. The formation and contraction of scar are great aids to the epithelization of an open wound because they reduce the size of the area to be covered by epithelium. The surgeon who is unaware of the part which contraction of scar plays in wound healing will erroneously



Figure 3 Hypertrophic scar. The skin mark on the abdomen was created by an abdominal incision. Tissue tension widened the scar and stimulated a hypertrophic change. The process is regressing as shown by the transverse wrinkles. Abnormal tissue tension produced a hernia for this patient. The fingers mark the edge of the fascial defect.

credit the dramatic change in size during the second and third weeks of healing to epithelial regeneration. This contraction takes place in a concentric manner but the greatest potential for shrinkage is in its longest axis. Scar will first effect its contraction in a line of least resistance and such areas may be the last to show softening and relaxation. Examples of these are readily found about the eyelids, lips, neck and flexor surface of joints.

Fibroplasia may also cause an increase in tensile strength of the wound and shorten the period of morbidity. Tests on experimental animals have shown that healed sutured wounds of the enteric tract may be stronger than the normal tissue. Specimens inflated with air often burst at a point removed from the site of injury.

Unfortunately the contraction of scar is a powerful force which can abnormally produce distortions and contractures (Fig. 4). There is little elasticity in scar tissue. If moderate traction is maintained for a long time contracture can be improved, but when the pull is discontinued, there is a gradual and definite return to the original contracture. Excessive force suddenly exerted upon a scar may disrupt it.

Other effects of scar contracture include a squeezing effect upon all of the elements within the wound. The edema begins to recede. The capillaries become compressed and may be gradually obliterated, resulting in fading of the redness of the wound area. The tissues become stiff and hard. In large deposits of granulation tissue, cicatrization continues over many months; the final conversion of red scars into characteristically white ones may require a year or more.

Although the tensile strength of a wound may be adequate for the resumption of function in two, three or four weeks, depending

Granulation tissue can provide for the patient other kinds of protection, often unnoticed and unappreciated. A coat of fibrin may reduce the sensitivity of the exposed surfaces of a fresh wound. Sensory nerves grow into the granulations, but they do not reach the surface until approximately the second week, depending on how the wound has been managed and whether or not efforts have been made to retard the deposit of excess granulations.

Another variety of protection offered by granulation tissue is the preservation of vitality in tissues such as nerves, tendons, bone, large blood vessels and cartilage. These tissues are easily damaged or killed when continuously exposed to the drying effect of air, repeated dressings or bacterial infection. Preservation of the protective clot, followed by its early organization into granulation tissue, protects such special tissues and permits them to survive.

Much valuable information can be obtained by observing and studying granulation tissues. The use of the word *healthy* or *unhealthy* is more practical than employment of the term *normal* or *abnormal*. Normal granulation tissue need not be the same as healthy granulation tissue. The granulation tissue normally found in a fifteen-year-old leg ulcer would be most unsuitable for the support of a skin graft.

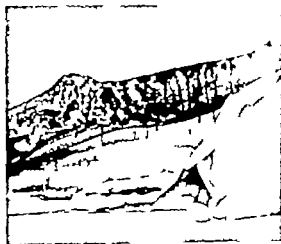
The desired healthy granulation tissue is a thin, firm, bright red membrane with a finely granular surface which does not bleed easily and which has no offensive odor (Fig 1)



Figure 1 Healing by secondary intention. Large deep granulating wound of arm covered with healthy granulation tissue.

The fluid loss from its surface is minimal and it should resemble a transudate rather than an exudate. No foreign or necrotic material should be present in the wound and the surrounding tissues should show no signs of irritation. The growth of epithelium over its peripheral margins is indicative of a healthy state which will support epithelial growth. Healthy granulation tissue may contain bacteria but will not show signs of acute infection.

Unhealthy granulation tissue on the other hand, is usually pale or cyanotic and wet and soft from edema (Fig 2). Its friable surface bleeds easily. The surface is coarse and irregular as a result of overgrowth of tissue in some areas and limitation in others. Excessive exudation, or pus, covers the surface saturates the dressings or irritates the surrounding skin. The patient may show signs of toxicity in the form of fever, edema of the part, regional lymphadenitis, malaise and ir-



A



B

Figure 2. A, Recent wound with unhealthy and infected granulation tissue. Edematous granulations are friable, bleed easily and are covered with bloody purulent exudate. B, Chronic infected granulation tissue which is exuberant, pale, friable and covered with purulent exudate. From the surface of indolent granulations, large amounts of proteins and electrolytes may be lost through continuous exudation. The paleness of the granulation tissues is caused by marked anemia and the edema secondary to infection and hypoproteinemia.



A

B

Figures 5. The stimulation for tissue growth created by an injury becomes abnormal in some patients. A, Keloid has formed in the margin of a burn which was partially covered with skin grafts. B, Keloid formation in donor sites for skin grafts.

The stimulating effects of trauma on scar tissue can readily be observed in many post operative incisions. Patients leaving the hospital during the second week following their operations will be free of skin sutures and will show smooth, soft, hair line approximations of their skin incisions. When observed over the succeeding months, many of the incisions will become hard, raised, deep red in color and will stretch from $\frac{1}{4}$ to $\frac{1}{2}$ inch in diameter. These are hyperplastic scars. They will recede in due time unless the patients are "keloid formers" (Fig. 5). This type of hyperplastic scar is commonly found in areas where the skin closure is under tension resulting from loss of skin stretching from muscular activity increased tension from heavy fat deposits, and stretching of the abdominal wall from intestinal distention, ascites, tumor or pregnancy. However such wounds are sufficiently strong to resist total disruption. Prolonged splinting will prevent the overgrowth of scar but any excess maintained for a long period will stretch the matured scar into an ugly mark. Excessive scar tissue is very susceptible to trauma and small hemorrhages or chronic ulcerations frequently occur beneath, or in the epithelium covering, the scar tissue (Fig. 6).

Thus far it has been necessary to assume that epithelization of the wound has gone on to completion. It must be remembered, however that granulation tissue, starting to mature at the periphery of the wound, can reach an advanced state of cicatricial formation without epithelial coverage. Epithelial growth is of prime interest since the functions of intact skin are so important. It is

protective as a covering of the body surface resisting bacterial invasion, regulating temperature and preventing loss of blood and lymph. It permits muscular contraction and expansion, remains soft and supple yet seldom wears out. It absorbs beneficial effects from light but throws down a barrier of pigment in an effort to prevent overdose. To counteract drying and reduce reaction, it will



Figure 6. Cicatrix on a leg showing its susceptibility to injury and infection. A chronic ulcer has appeared in the dense nonpigment-bearing scar.

on the location and type of tissue injured its actual strength continues to increase during the aging of the cicatrix. As the strength of the wound increases, the stimulation for growth of the tissues involved in the repair will be found to decrease. However repeated injury to a healing wound will prolong the effect of the stimulation for growth. The manner in which this is brought about is not known.

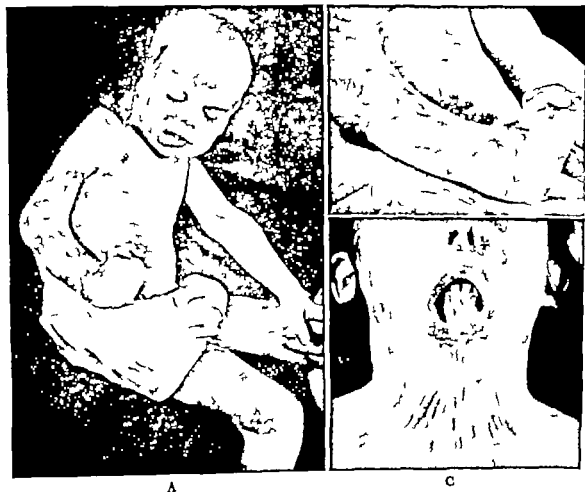
Tissue cultures of fibroblastic tissue show that growth will advance just so far and then come to a halt. It is not cicatrization or circulatory ischemia which stops it because the growing cells at the periphery are bathed in fresh media and could keep right on growing, if so inclined. Obviously it is not the influence of a completed regeneration of an epithelial coverage. Epithelial cells do not take part in the tissue culture experiments and, furthermore, we know that the healing of concealed wounds behaves the same way with the skin remaining intact. It

is fortunate that tissue growth does slow down as healing advances and that it can stop itself at the right time.

If cultures of fibroblastic tissues are fed until the masses stop growing, and if incisions are made in these masses, growth will start again. Products from the injured cells stimulate the regeneration and this can be repeated on the same tissue culture. The effect becomes less each time, however and will not go on indefinitely.

A related effect can be observed in clinical wounds. A granulating wound which is too large for spontaneous regeneration of epithelium may be dressed and carefully tended until growth seems to stop. The placement of pieces of skin graft in the wound will be found to stimulate the marginal epithelium to grow again so that it will have joined the marginal grafts before the grafts themselves have started to send out new epithelium. In this instance, the stimulation must come from the cut epithelium applied as grafts.

B



A

C

Figure 4 The power of scar contractures. A, Extensive areas of granulation tissue following severe burns. Epithelization was assisted by grafts. B, Hypertension contracture of the wrist. C, Flexion contracture of the neck causing eversion of the lower lip.



Figure 5. The stimulation for tissue growth created by an injury becomes abnormal in some patients. A, Keloid has formed in the margin of a burn which was partially covered with skin grafts. B, Keloid formation in donor sites for skin grafts.

The stimulating effects of trauma on scar tissue can readily be observed in many post operative incisions. Patients leaving the hospital during the second week following their operations will be free of skin sutures and will show smooth, soft, hair-line approximations of their skin incisions. When observed over the succeeding months, many of the incisions will become hard, raised, deep red in color and will stretch from $\frac{1}{4}$ to $\frac{1}{2}$ inch in diameter. These are hyperplastic scars. They will recede in due time unless the patients are "keloid formers" (Fig. 5). This type of hyperplastic scar is commonly found in areas where the skin closure is under tension resulting from loss of skin, stretching from muscular activity, increased tension from heavy fat deposits, and stretching of the abdominal wall from intestinal distention, ascites, tumor or pregnancy. However such wounds are sufficiently strong to resist total disruption. Prolonged splinting will prevent the overgrowth of scar but any excess maintained for a long period will stretch the matured scar into an ugly mark. Excessive scar tissue is very susceptible to trauma and small hemorrhages or chronic ulcerations frequently occur beneath, or in the epithelium covering, the scar tissue (Fig. 6).

Thus far it has been necessary to assume that epithelialization of the wound has gone on to completion. It must be remembered, however that granulation tissue, starting to mature at the periphery of the wound, can reach an advanced state of cicatricial formation without epithelial coverage. Epithelial growth is of prime interest since the functions of intact skin are so important. It is

protective as a covering of the body surface resisting bacterial invasion, regulating temperature and preventing loss of blood and lymph. It permits muscular contraction and expansion remains soft and supple, yet seldom wears out. It absorbs beneficial effects from light but throws down a barrier of pigment in an effort to prevent overdose. To counteract drying and reduce reaction, it will



Figure 6. Cicatrix on a leg showing its susceptibility to injury and infection. A chronic ulcer has appeared in the dense pigmented-bearing scar.

on the location and type of tissue injured its actual strength continues to increase during the aging of the cicatrix. As the strength of the wound increases, the stimulation for growth of the tissues involved in the repair will be found to decrease. However repeated injury to a healing wound will prolong the effect of the stimulation for growth. The manner in which this is brought about is not known.

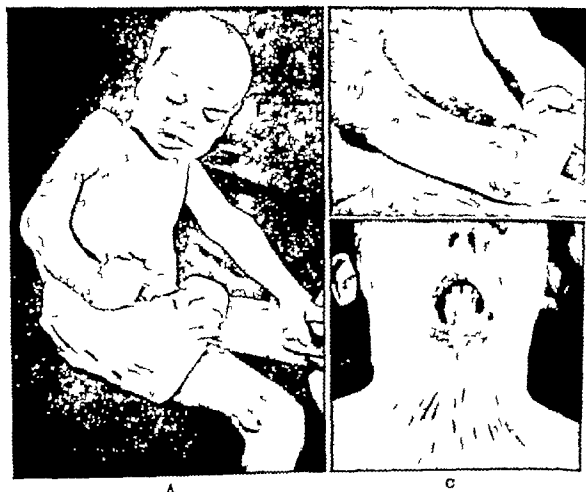
Tissue cultures of fibroblastic tissue show that growth will advance just so far and then come to a halt. It is not cicatrization or circulatory ischemia which stops it because the growing cells at the periphery are bathed in fresh media and could keep right on growing, if so inclined. Obviously it is not the influence of a completed regeneration of an epithelial coverage. Epithelial cells do not take part in the tissue culture experiments and, furthermore, we know that the healing of concealed wounds behaves the same way with the skin remaining intact. It

is fortunate that tissue growth does slow down as healing advances and that it can stop itself at the right time.

If cultures of fibroblastic tissues are fed until the masses stop growing, and if incisions are made in these masses growth will start again. Products from the injured cells stimulate the regeneration and this can be repeated on the same tissue culture. The effect becomes less each time, however and will not go on indefinitely.

A related effect can be observed in clinical wounds. A granulating wound which is too large for spontaneous regeneration of epithelium may be dressed and carefully tended until growth seems to stop. The placement of pieces of skin graft in the wound will be found to stimulate the marginal epithelium to grow again so that it will have joined the marginal grafts before the grafts themselves have started to send out new epithelium. In this instance, the stimulation must come from the cut epithelium applied as grafts.

B



A

C

Figure 4 The power of scar contractures. A, Extensive areas of granulation tissue following severe burns. Epithelization was assisted by grafts. B, Hypertension contracture of the wrist. C, Flexion contracture of the neck causing eversion of the lower lip.

thelium if the area is not wide. The size has not been established because there are many variables which affect the migration. In dark-skinned people, all depths of the epidermis are pigmented, but the deepest cells are the most heavily pigmented. It is interesting to watch pigmentation develop in the larger spontaneously healed burns of the Negro. The deep second degree burns which have regenerated spontaneously show a pigmented honeycomb effect. The comb itself is black and represents the deep valleys between the papillae filled with stratum germinativum which survived the burn. The part of the comb intended for honey is red. It represents the epidermis burned through into the corium at the apices of the papillae with recent regeneration of epithelium and no pigment. In due time the pigmentation becomes confluent. If the burn should go a millimeter or two deeper the pattern is reversed. The red epithelium takes the honey comb design while the pigmentation becomes spotted. This represents the deepest aspects of the papillae toward the subcutis side with preservation of the merest fragment of stratum germinativum. Such a wound may also become pigmented ultimately throughout.

Following the epithelization of granulating wounds, papillae formation is very poor. Glands and hair follicles do not form and elastic fibers appear very slowly if at all. Even then, it may be months or years before they are present in the more favorable locations at the periphery of the wound.

Sensation will return to skin grafts, and to spontaneously epithelized wounds, if the peripheral nerves are intact and if the granulation tissue is not so thick as to be impervious to the growth of nerve fibers. The degree of sensation may be expected to improve up to one or two years after injury.

With the exception of the cellular elements which are carried in the blood, the bulk of the work in the basic processes of wound healing which go on in all types and sizes of wounds is done by the fibroblasts, endothelial and epithelial cells. This holds equally well for injuries to special tissues.

Because of the multitude of variations which may arise as a result of the wound's location, size, shape, duration, causative agent and treatment, some method for sorting and grading wounds in relation to the character of their healing is of practical value. A classification is helpful in the study evaluation and teaching of wound management. Galen, in the second century A.D., is

given credit for devising the terms of primary and secondary healing and, as the terms continue to be in everyday use, it is necessary to sort out certain features of wound healing and group them according to their applicability to these terms.

PRIMARY HEALING

This type of healing is nearly always described as wound healing at its best. It is healing by *first intention* (Fig. 7). It is the healing of a closed, or concealed, wound without the complication of infection, or the secondary opening of the wound for the release of blood, lymph, serum, body secretion or excretion or foreign body. It will include the ideal wounds as well as those which show edema, discoloration or hardness from excess blood, as some granulation tissue forms in the majority of closed wounds. It is wrong to set up a classification which would preclude this fact. Acceptance of the thought that the majority of the wounds which heal per primam do so through the formation of granulation tissue places no restriction on the effort to approach the ideal of wound healing and gives a category for the wounds not belonging in the class of open wounds.

In some classical examples, scarcely any cellular reaction is described in response to the injury which was made so smoothly that practically no cells were destroyed. The wound is aseptic; the parts are approximated accurately, hemostasis is so perfect that no fluid forms within or leaks from the wound, and scar tissue is minimal or absent after healing.



Figure 7 First intention healing. The thin line on the neck represents the healed incision through which a radical neck dissection was done.

cover itself with oil. Cutaneous epithelium is in a constant state of regeneration the deeper cells replenishing the supply of new cells to compensate for the constant shedding of old.

After injury areas of regenerated epithelium will not retain all of these functions. A free graft of skin may carry with it portions of oil glands, sweat glands and hair follicles, but these elements do not form in the new epithelium which grows spontaneously from the margins of the wound or the graft.

Epithelium starts to grow immediately after injury if it has a place to grow. The closely approximated wound and the abrasion will permit prompt growth. The jagged wound with frayed edges of devitalized skin cannot grow epithelium until the dead tissue is removed. A wound with sharp edges and no approximation will not grow epithelium until the clot beside it is organized into granulation tissue. Epithelium will not grow across fibrin unless there is active circulation immediately beneath it. Epithelium does not grow readily downward into a hole or upward over a hill which is at right angles to its surface. It grows slowly in large wounds and often will stop growing before the contraction of the scar is complete thus leaving most of the wound uncovered. When granulation tissue is just right for epithelial growth, the rate of coverage is most rapid.

The corium of normal skin is derived from the mesoderm while the epidermis comes from ectoderm. The sweat glands, oil glands and hairs are ectodermal in origin. The real focus of skin regeneration is from stratum germinativum of the epidermis. In working with split thickness grafts of skin, one finds little trouble in getting any thickness of graft to grow provided the cut surface does not extend deep to the papillae to include a layer of adipose tissue. Free adipose tissue transplants poorly and when left attached to the undersurface of a skin graft, jeopardizes "take" of the graft. A graft taken deeply into the corium will retain good pigment and a sufficient amount of elastic fibers to impart good cosmetic and functional properties to the graft. The preservation of a portion of stratum germinativum of the epidermis is the surest way to obtain epithelial regeneration.

Rather fantastic accounts are given of the capacity of the epithelium to re-establish continuity in brief periods. In these instances the wounds are minor and perfect appositions of the parts are required. When these conditions are provided, it is sometimes

difficult to tell where the injury had occurred after a period of twenty four hours. In the strictest sense, such wounds are not actually healed but are held together by their coagulum.

Crusting, or the formation of a scab, in small wounds is Nature's way of simplifying wound care and wound healing. The drying of the wound's seepage creates a tough, elastic film over the unhealed surface. This film sticks to the dry superficial cells of the epidermis so that the epidermis is securely held in place. Additional bacteria rarely get through and if those beneath do not cause a serious infection the crust will stay on until epithelization is complete and strong. To many surgeons crusting is regarded as something used by Nature for the healing of non-surgical wounds. This is an error because in some degree it is found to cover the skin edges of sutured wounds, even those most carefully approximated. It makes possible the early removal of sutures in wounds of cosmetic importance so that scars are not produced by the sutures themselves. The substitution of some variety of cutaneous adhesive dressing will protect the wound from damage by violence.

To a lesser degree, scab formation occurs at the periphery of the larger open wounds beneath which the growth of epithelium may be studied. Tongues or sheets of cells, coming from the stratum germinativum of the epidermis make their way by enzymatic action through the fibrin under the crust. If mechanical effort is made to remove the crust which is adherent to the epithelium in the early days of healing, some of the fresh epithelium may come away with the crust. Spontaneous separation of the crust will occur in about two weeks.

It is important to have a clear understanding of these principles so that one does not hinder the regeneration of skin by vigorous attempts to cleanse wounds. The use of lubricated dressings on open wounds has come about as a means of protecting the delicate strands of epithelium beneath the peripheral crusts. Any other dressing which would adhere to the crusts cannot be removed without pulling away many of them with their epithelial elements. Dressings designed to be left in place for many days have not been wholly satisfactory because of the retention of the irritating drainage from the granulating wounds. Although crusts never really dry under lubricated dressings they do stay gummy and remain in place.

Pigmented cells will migrate into new epi

detached pieces of fat and muscle which, when freed of blood and dirt can be made to resemble closely the tissues remaining in the wound. Structurally the living and detached tissues may be the same except for the property of bleeding. However these detached tissues are devitalized, that is detached from life. If returned to the wound, it is unlikely that they will survive as grafts. If they fail to live, they lose the color and firmness associated with living tissue. Individually the cells break down and, as a group the tissues soften, fall apart and liquefy. The process is one of necrosis and the tissues become necrotic.

The removal of devitalized tissues in fresh wounds by various techniques of débridement, has become an accepted measure in their operative care. It is customary in some hospitals to regard the surgical treatment of a fresh wound as a "débridement," regardless of the fact that tissue may not have been removed during the operation.

When devitalized tissues of significant quantity are allowed to remain in wounds, they proceed to become necrotic. The number of enzymes, the leukocytic response, the capillary permeability the fluid loss and the absorption of toxins from the wound are increased.

Dead or devitalized tissues are locally irritant, serve as an excellent pabulum for the growth of bacteria and divert energies of living cells from the problem of repair to the problem of elimination of foreign materials. Foreign body reaction occurring about foreign bodies or dead tissue may delay wound healing and foster infection.

Locally the swelling, redness and tenderness will exceed expectations. Open wounds will have profuse drainage and the material will be of exudative nature. Both fibrosis and epithelization are delayed. Not until the necrotic tissue is expelled or removed by the mechanical means of excision or dressings will the drainage subside and healing progress.

It is, therefore, important that devitalized or necrotic tissues resulting from injury should be removed by surgical dissection before active bacterial growth and infection have developed. It is also important that the surgical treatment does not result in any unnecessary trauma to the tissues during or after the operation.

Infection is the greatest enemy of wound healing. The development of any infection, particularly in large wounds, almost certainly increases the period of mor-

bidity after operation since it produces further destruction of tissue and suppresses the process of healing. Tissues destroyed by infection are usually replaced by scar tissue, which may affect function as well as cosmetic appearance.

The surfaces of the human skin and mucous membranes normally are contaminated with bacteria. The microorganisms lie in pits, creases, glands and hair follicles beyond the reach of surface washing. When the body's surface is penetrated by trauma, bacteria cross the cutaneous protective barrier and penetrate the physiologic interior. They may also be carried into a wound by the object producing the trauma or by foreign bodies.

Experience has shown that bacteria may be present in a wound without producing the slightest clinical evidence of infection and that certain factors influence their growth and development. Healthy tissues have a remarkable capacity to kill bacteria or withstand their effects, while unhealthy, irritated or devitalized tissues have limited or little power of resistance to their action. Dead tissue in a wound invites and supports the growth of virulent organisms as well as nonvirulent or saprophytic ones. Therefore it is extremely important to remove any potential pabulum for bacteria and to prevent the development of a similar breeding ground during the postoperative state.

On the other hand, the apposition of live tissue to live tissue after the thorough removal of all foreign, dead or dying tissue promotes healing even in the face of some contamination. The physical condition of the patient is an important predisposing factor to infection. Dehydration, shock, malnutrition, exhaustion, uncontrolled diabetes and anemia may lower his resistance sufficiently to permit bacterial invasion.

Just as the fibroblasts, angioblasts and neutrophils have enzymes to help them to make their way into fibrin, bacteria have toxins and enzymes to take them through the tissue barriers within the wounds. The powers of materials liberated by injured cells are slight when compared with the potencies of those produced by bacteria. It is little wonder that the surgeons prefer to look upon "inflammation" as the handiwork of bacteria and exclude the reactions of uncomplicated repair.

The staphylococcus produces a coagulase which clots small vessels and favors spread by septic emboli and hastens local gangrene. Some staphylococci and streptococci produce hemolysins which break up red blood

A description like this nearly removes primary healing from the realm of surgery and places it out of reach of practicability. Healing is a reaction, and without any reaction, healing would not occur. To eliminate all hyperemia about a wound is an impossibility. The reaction from a pin scratch or from the injection of a local anesthetic may last for days. The mere preparation of skin with its shaving and scrubbing can reduce it to a state of "weeping" with plasma before the incision is made. Every primary dressing that is changed is stained with wet or dried fluid from the wound. No incision can be made through the skin without leaving some scar. Surgical technique cannot be expected to outdo Nature. The ideal wound has to be coddled just as long as any standard wound to insure healing without complication.

The reaction is more quantitative than qualitative throughout the steps of repair in primary healing. When the tissues are in accurate apposition and when surgery has effected most of the desirable requirements for good healing in a closed wound, there will be a minimal reaction and less work for the tissues to do. One cannot speed the healing, but one can control the factors which are capable of delaying it.

SECONDARY HEALING

It is probable that the idea for giving this type of healing the title of *healing by second intention* came from the recognition of second attempts at healing by wounds starting out well but breaking down under the effects of *suppuration*. We still regard the healing of such wounds as secondary (Fig. 1). Generally our minds carry us to the more spectacular examples found in the larger open wounds created by surgery, disease or accident which were not closed. Open wounds which heal without becoming acutely infected do so by secondary intention. They cannot heal by first intention, unless the surgical attention given them at the time of the injury provided for them a skin coverage by flap graft or free skin graft. In this case, the wounds would no longer be "open" and would then qualify for primary healing, if the skin coverage were to remain intact. The detailed description of the organization, aging of granulation tissue and epithelialization is an accurate account for the healing of a wound by second intention. It is not the quantity of the granulation tissue which distinguishes the two, but the absence of epithelial coverage that accounts for secondary healing.

THIRD INTENTION HEALING

An open granulating wound which is assisted in its healing by an operative procedure is changed from healing by *second intention* to healing by *third intention*. Again the implication is that the work was accomplished after a third attempt. This is not always the case, because often no opportunity was given for the wound to heal by first or second intention. It is a little disturbing to be told that a wound upon which a secondary closure has been done actually heals by third intention. If primary and secondary healing include all stages of growth of granulation tissue and epithelial coverage is eventually acquired in both, what new and different process in wound healing takes place in third intention healing?

Third intention healing is a convenient term for surgeons. In the strictest sense, it implies the use of a skin graft, or delayed closure, in the treatment of a granulating wound which is healing satisfactorily by second intention in an effort to obtain earlier healing, less morbidity and a more functional and cosmetic result.

The physiologic process of wound healing is subject to great variations. Optimum wound healing is the normal rate of wound healing under physiologically normal conditions. Numerous experiments of Howes and others, have shown that there is no known substance for topical or systemic use which will accelerate the rate of wound healing beyond the normal physiologic rate. The variation in wound healing is one of retardation or limitation caused by a number of factors both local and systemic.

LOCAL FACTORS IMPAIRING HEALING

Medications which "heal" wounds may be looked upon in the same way that teachers "learn" their pupils. Both assist in the accomplishment of the respective goals but, just as the pupils do their own learning, the tissues do their own healing.

Tissues have their own normal rates for growth in the process of healing. This rate is approached when all of the variables advantageous to the healing rate are present in a normal amount and when all of the variables having the ability to disturb or retard the healing processes are controlled or absent.

Devitalized Tissue and Necrotic Tissue. These terms are not to be used interchangeably. While each is nonliving tissue, necrotic tissue implies greater advancement of tissue degeneration.

In a fresh wound of violence, one may find

which come closest to the ideal are used as foreign bodies intentionally placed in certain wounds to serve a useful purpose. When the reaction is minimal, the tissues tolerate them and encase them in scar tissue.

Many metals not considered desirable for use in surgery are tolerated by tissues which surround them with scar but a delayed injury from their presence may be suffered. Lead bullets may slowly decompose to produce lead poisoning. Iron may corrode. A fragment lodged in an eyeball may cloud the tissues with pigment and cause blindness. Sharp objects, such as needles may travel through the tissues from the effects of pressure endured during exercise and introduce complication by their penetrations. Large and irregular objects resting against pressure areas may produce erosion.

Other physical attributes affect tissue tolerance. Porous materials are tolerated less well than the solid and impervious ones. This is especially true when bacteria are growing in the wound. A monofilament suture will do better than one made with twisted or braided fibers. The fewer foreign bodies left in the wound and the smaller their sizes, the better will be the tissue tolerance.

Foreign bodies may be the primary irritants because of their chemical natures. Strong acids and alkalis may effect their own burns and many salts are locally or systemically poisonous. Insoluble foreign bodies such as glass, gravel and carbon particles can be looked upon as inert substances. If not contaminated with bacteria, they may remain in the tissues with little or no reaction if not removed by operation.

Organic materials such as grass, straw, clothing and wood are not tolerated. The usual bacterial contaminations will cause recurrent inflammations until they are removed.

Sutures are also examples of foreign bodies. Catgut sutures are so chemically constituted that human tissues may act upon them and destroy them. Nonabsorbable sutures must be inert and nonirritative to tissues.

Homologous skin will grow but soon apparently produces antibodies in the host which will destroy the grafts about the third week. Homologous grafts of fascia, cartilage and blood vessels are tolerated much better than are grafts of skin elements. The tissues do not live, but their forms are preserved for structures of fixation by the host's cells provided the grafts are not complicated by infection.

When a foreign body lies in badly damaged tissues, its chances of retention within the wound are greatly reduced. Since the traumatized tissue enveloping it is also a foreign body which will undergo some degeneration, little opportunity is given the living cells to envelop and accept it. Liquefaction with drainage may dislodge it or cause it to be an additional source of irritation. Great care must be observed in surgery so that tissues are not unduly traumatized when one expects to leave foreign bodies within the wound.

One of the greatest problems of foreign bodies comes with their relationship to infection. Being inert and occupying space, they keep living tissues apart. The larger and more numerous they are, the greater is their contribution to complications in healing. If they are of the absorbable kind, the very act of absorption creates edema and free fluid which favor bacterial growth. If they are porous, bacteria will remain alive within the foreign bodies and survive the effects of antibiotics and antibacterial drugs, which may give temporary control to the reaction in the living tissues. At a subsequent date the infection may start again. When a reaction about a foreign body is going on, drainage from a wound will be copious and complete healing will not be accomplished until the foreign material is removed. Tissues which formerly seemed to grasp the material very securely will be found to soften and withdraw from it. Again the only remedy is removal.

It is an accepted fact that foreign bodies left in tissues predispose to infection of the area at any time. A patient who has carried for years a foreign body of one kind or another and has had no difficulty from it may develop an acute reaction and an abscess about the object. Sometimes these are traced to secondary trauma without additional external contamination. At other times the source of the infection is unknown and it is assumed that the organisms are distributed by the blood to an area of lowered resistance.

Local Blood Supply. To interfere with the local blood supply of a wound, even in a healthy individual, is like limiting the raw materials to a good industry. Production will drop off. The process of granulation tissue formation and wound healing is impossible without an adequate blood supply.

The blood supply may be interfered with locally by the injury itself, laceration of regional vessels such as the popliteal artery, or pressure of old or new scar tissue. Swelling

corpuscles to stain the tissues, produce a brownish discoloration to the drainage and contribute to the development of anemia in severe infections.

The streptococcus may also produce a fibrinolysin which makes it difficult for the tissues to wall off a streptococcal infection, since granulation tissue is required for this purpose, and the growth of granulation tissue is dependent upon the deposition of fibrin. This enzyme is known as streptokinase and is commercially available for therapeutic use against heavy deposits of fibrin.

Streptodornase is another enzyme complex produced by streptococci. Its action is on the constituents of nuclei and cytoplasm of cells particularly leukocytes found in pus. Some of the enzymes in the dornase factor are believed to be hyaluronidase, ribonuclease, desoxyribonuclease, nucleotidases and nucleosidases.

Most organisms produce proteinases which assist in the general breakdown of proteins. Special ones such as collagenase, permit them to penetrate fascial barriers. The human bite infections are famous for this and much of the credit goes to the *Bacteroides melanogenicus* and the anaerobic streptococcus. The former produces a black pigment and contributes to the putrid smell of lung abscesses, dirty mouths, appendiceal abscesses and infections of the human bite. Its enzymes readily dissolve fascia, ligaments and tendon sheaths to cripple permanently its victims.

Clostridial organisms may work singly or in combination. Some produce strong proteinases, others saccharolytic enzymes which ferment muscle sugar into gas and acid. It is believed that hyaluronidase acts as a spreading factor and assists in the dissemination of their toxins. Other bacteria also produce hyaluronidase presumably for the same purpose. The proteinases of some clostridia are very potent. The proteinase and collagenase of *Clostridium histolyticum* are capable of completely liquefying the muscles and fascia of the extremity of an animal or a man.

Some bacteria, such as the staphylococci, streptococci and clostridia, can produce necrotizing enzymes capable of producing necrosis of tissue by direct contact. In addition, spontaneous bleeding from infected granulation tissue has been associated with the products of growth of certain bacteria. In active infections combinations of all of these enzymes may assist the microorganisms in their penetration and destruction of the tissues. Edema secondary to infection

may interfere with circulation and foster necrosis as in gas gangrene.

In brief toxins produced by bacteria may account for hyperemia, increased capillary permeability edema, spontaneous bleeding, clotting, clot and tissue liquefaction and fragmentation and death of tissue cells. Moreover combinations of bacterial toxins possess a synergistic effect. Enzymes liberated from dead and dying cells in combination with bacterial enzymes may further tissue reaction.

Foreign Bodies. A mass which is not normal for the tissues in which it is found is a foreign body. In the broad sense, it is not entirely true that a foreign body is something introduced from the outside. Tissues can produce their own foreign bodies such as urinary calculi, salivary calculi, gallstones, calcium deposits in areas of old injury or uric acid crystals of gout.

Foreign bodies produced by the tissues may be liquid. Following injuries to glandular tissues their secretions or excretions escape from normal channels and become foreign for the surrounding structures. Examples of this include the distention with saliva of a closed wound of the cheek which has caused damage to the parotid gland free bile in the abdominal cavity from a ruptured bile duct or a lacerated liver leakage of pancreatic juice following injury to this gland extravasation of urine following trauma to the urinary tract, and leakage of gastric or intestinal secretions into the abdominal cavity after perforation of a hollow viscus.

Abnormal accumulations of blood and lymph in tissues may be classed as foreign bodies. Hematomas and fluid accumulations of serum and lymph in large closed wounds are inert materials abnormal for the tissues and they are treated by the host as foreign bodies. A mass of tissue which dies and is allowed to remain in place also falls into the category of a foreign body.

Obviously the reactions will present great variations. To acquire good clinical judgment about the management of foreign bodies, one should analyze their attributes in relation to tissue responses. Most of the reactions to foreign bodies can be traced to the physical and chemical natures of the bodies themselves, the state of health of the tissues about them and the presence of bacteria.

A continuous search is being conducted for metals which are strong, light, noncorrosive, malleable and nonirritating. The ones

Anything that can be done to control or eliminate stasis will benefit wound healing. Elevation of the part for gravity drainage has already been suggested. Compression dressings have been in vogue for years but they are difficult to apply and their pitfalls are many. During the heat of summer the heavily padded pressure dressings applied to body burns have contributed to the retention of heat and the production of thermal disasters to some patients. Constricting dressings about the thorax and abdomen may cause respiratory embarrassment and pressure dressings on an extremity which do not go all the way to the end will serve to increase the stasis and swelling beyond the bandage.

Hemorrhage. Disregarding the effects on the individual of having a continued loss of blood to deplete his blood volume or his cellular and serum protein reserves, bleeding into wounds does have adverse influences on their healing. Ideally the coagulum should be just enough to stick the parts of the wound together and to eliminate dead spaces. The effects of blood accumulation beyond this amount, resulting from the injury or inadequate stasis during operation, are rather clear since the sides of the wound will be forced apart. The resulting hematoma will increase the tension on the tissues to produce pain, ischemia, necrosis and delayed healing. Sutures subjected to the stretching effect of an expanding hematoma may cut through and become loose.

Bleeding in a closed wound will cause the blood to extravasate along planes of least resistance. Bleeding from severe fractures of the pelvis, for example, can extend in the retroperitoneal tissues to the diaphragm, mediastinum and even to the level of the neck. These areas can hold more blood than the patient can afford to lose and death is common following such accidents. The presence of blood in these areas disturbs the innervations to the gastrointestinal tract and favors the onset of ileus.

Extravasated blood in muscle and fascia stiffens the tissues, increases the edema, occludes capillary flow, impairs the flow of lymph and lessens local resistance to infection. Function of a part may be affected by the pain, swelling, stiffness, diminished blood supply and impaired innervation resulting from marked extravasations of blood.

Since extravasated blood cannot return to the circulation as whole blood, it usually clots and slowly undergoes degeneration and absorption, while a part of it is utilized in

the growth of granulation and scar tissues. This, too, increases the morbidity for the injury and leaves behind an undesirable amount of scar to be reckoned with during the convalescence and rehabilitation.

There is no circulation within a hematoma until it becomes organized. Until that time, bacteria within the hematoma cannot be reached by systemic antibacterial drugs. Large hematomas which are slow to absorb and which have not been aided by therapeutic evacuation sometimes end in rigid wall cavities containing encapsulated fluid.

The effect of blood clots being without circulation can be observed in work with skin grafts. If a hematoma develops beneath a graft it will hold the skin away from its source of nourishment and bring about its death. The bleb may then be opened and the contents evacuated. The surrounding area of graft, having remained viable, may be sufficient to provide epithelial growth to cover the defect or additional grafting may be necessary if the hematoma was large.

General Factors Influencing Wound Healing. *Blood supply.* Since living cells contributing to the process of healing must receive their energy and materials necessary for growth from the circulating blood, any systemic disease which diminishes the blood supply may impair wound healing. Diseases producing anemia, cardiac decompensation, obliterative arterial disease, decreased blood volume and edema may therefore, have marked effects on the process of healing. The surgeon must, therefore, observe carefully the efficiency of the general circulation in order to determine whether or not healing is possible or if it can be improved by treatment which renders the circulation more efficient. Careful attention should be paid to the status of the cardiac function since digitalization may be required to overcome dependent edema in patients with cardiac decompensation. If the arterial supply to the part is greatly diminished by arteriosclerosis, it may be wise to place the injured leg in a slightly dependent position to augment the local blood supply. The presence of anemia and decreased blood volumes should be determined and corrected. There is suggestive evidence that the poor wound healing observed in severely anemic patients is probably secondary to protein and vitamin deficiencies rather than to the anemic state itself.

Nutrition. A normal physiologic status is essential for a normal and most rapid rate of wound healing. Alterations in the physiologic

from contusion, congestion, infection or toxins from bites of poisonous animals serves to cut down the local supply of blood. Whatever the cause, impairment of wound healing occurs.

Signs of retardation in healing appear. The formation of granulation tissue is retarded and the tissues become pale. If blanching is produced by digital pressure, the return of circulation is sluggish. The skin is cool and wounds are slow to epithelize and contract. The line of incision shows little hyperemia and is easily separated. The open wound shows pale, wet, soft granulations which grow sluggishly or fail to grow at all. Purulent exudation is minimal, but bacterial growth is likely to increase tissue destruction. Skin flaps are readily lost and grafts may fail to take or are easily dislodged by activity.

Effective surgery should not be attempted on tissues known to have poor blood supply. In an emergency everything possible should be done to improve the condition accounting for the disorder.

In the care of wounds of violence involving tissues which are otherwise normal the surgical therapeutic effort to remove devitalized tissues from the margins of these wounds is based in part on the appreciation of the contribution of a good blood supply toward prompt healing. Apposition of tissue with an assured supply of blood is most desirable. Flaps with precarious blood supply must not be impaired with constricting bandages and tight or excessive sutures. Excessive scar tissue with deficient circulation which affects the recovery of a part should be excised to a good source of blood and the defect corrected by a suitable graft.

When ischemia develops in tissues which have no impairment of sensation, marked pain develops. Whether it is produced by an arterial embolus, tight dressing, tight cast, tight sutures or a distended wound, the mechanism is similar. This can be appreciated through two simple experiments. Place a blood pressure cuff about the arm and inflate it to a degree well above the systolic pressure. Record the time and see how long it can be tolerated. Second, sit in a chair and obtain the most comfortable position possible. Extend the legs on a foot stool if you wish, and pick up an interesting book. From then on do not change the position. When ischemia begins to develop over the heels, calves, knees, ischial tuberosities, sacral area or over the greater trochanter of the femur because of the continued pressure, the pain

will become disturbing enough to interfere with the reading. Record the time, and do not move. When it becomes most unbearable, think of the infant being given an intravenous injection in a leg which is strapped too tightly to a splint, or the patient who has a lump in his cast, or the bedridden creature who cannot change his position but is turned every four hours by a nurse. What part of four hours can you sit without moving?

Edema and Lymphatic Obstruction When stasis of lymph and venous blood begins to pile up in a wound, the efficiency of healing begins to wane. When lymph flow is obstructed, edema results. When venous flow is obstructed, venous engorgement and edema result. Oxygen tension in the tissues is reduced, carbon dioxide builds up, the pH falls, tissue edema increases, and more stress is placed upon the lymphatic channels. Tissue metabolism slows and the by products of metabolism begin to accumulate in the tissues to produce a sense of fatigue and malaise.

Lymphatic or venous blockage produces swelling which is detrimental to wound healing and favors bacterial growth. The drainage of open wounds increases. Vesicles may appear in the skin which may break down and form open wounds. Tissues boggy from edema, will have a restricted blood supply. Capillaries and venules which do not become occluded with clot will be obstructed by pressure. Fibrosis in tissue is enhanced by stasis but fibrosis is not a desirable reaction when it extends beyond the healing of the wound itself. Fibrosis in tissues about a wound weakens the resistance of those tissues to future injury and retards the repair of those injuries.

Excellent examples of the effects of lymphatic and venous stasis are found in the lower extremity injuries in the lower third of the leg heal more slowly than in any other part of the body. Acute and chronic ulcerations occur frequently and become complicated by fibrosis and cicatrization. While other factors enter into the problems of wound healing for the lower extremities stasis of blood and lymph is a common denominator of all of them.

The part played by stasis in delaying the healing of wounds is evident when one observes an ambulatory patient's leg ulcer of long duration heal rapidly as soon as the patient is placed at bed rest with the extremity elevated. The improvement which comes with bed rest is the result of correcting the stasis accompanying the upright position.

Anything that can be done to control or eliminate stasis will benefit wound healing. Elevation of the part for gravity drainage has already been suggested. Compression dressings have been in vogue for years but they are difficult to apply and their pitfalls are many. During the heat of summer the heavily padded pressure dressings applied to body burns have contributed to the retention of heat and the production of thermal disasters to some patients. Constricting dressings about the thorax and abdomen may cause respiratory embarrassment and pressure dressings on an extremity which do not go all the way to the end will serve to increase the stasis and swelling beyond the bandage.

Hemorrhage. Disregarding the effects on the individual of having a continued loss of blood to deplete his blood volume or his cellular and serum protein reserves, bleeding into wounds does have adverse influences on their healing. Ideally the coagulum should be just enough to stick the parts of the wound together and to eliminate dead spaces. The effects of blood accumulation beyond this amount, resulting from the injury or inadequate stasis during operation, are rather clear since the sides of the wound will be forced apart. The resulting hematoma will increase the tension on the tissues to produce pain, ischemia, necrosis and delayed healing. Sutures subjected to the stretching effect of an expanding hematoma may cut through and become loose.

Bleeding in a closed wound will cause the blood to extravasate along planes of least resistance. Bleeding from severe fractures of the pelvis, for example, can extend in the retroperitoneal tissues to the diaphragm, mediastinum and even to the level of the neck. These areas can hold more blood than the patient can afford to lose and death is common following such accidents. The presence of blood in these areas disturbs the innervations to the gastrointestinal tract and favors the onset of ileus.

Extravasated blood in muscle and fascia stiffens the tissues. Increases the edema, occludes capillary flow. Impairs the flow of lymph and lessens local resistance to infection. Function of a part may be affected by the pain, swelling, stiffness, diminished blood supply and impaired innervation resulting from marked extravasations of blood.

Since extravasated blood cannot return to the circulation as whole blood, it usually clots and slowly undergoes degeneration and absorption, while a part of it is utilized in

the growth of granulation and scar tissues. This, too, increases the morbidity for the injury and leaves behind an undesirable amount of scar to be reckoned with during the convalescence and rehabilitation.

There is no circulation within a hematoma until it becomes organized. Until that time, bacteria within the hematoma cannot be reached by systemic antibacterial drugs. Large hematomas which are slow to absorb and which have not been aided by therapeutic evacuation sometimes end in rigid wall cavities containing encapsulated fluid.

The effect of blood clots being without circulation can be observed in work with skin grafts. If a hematoma develops beneath a graft it will hold the skin away from its source of nourishment and bring about its death. The bleb may then be opened and the contents evacuated. The surrounding area of graft, having remained viable, may be sufficient to provide epithelial growth to cover the defect or additional grafting may be necessary if the hematoma was large.

General Factors Influencing Wound Healing. *Blood supply.* Since living cells contributing to the process of healing must receive their energy and materials necessary for growth from the circulating blood, any systemic disease which diminishes the blood supply may impair wound healing. Diseases producing anemia, cardiac decompensation, obliterative arterial disease, decreased blood volume and edema may therefore, have marked effects on the process of healing. The surgeon must, therefore, observe carefully the efficiency of the general circulation in order to determine whether or not healing is possible or if it can be improved by treatment which renders the circulation more efficient. Careful attention should be paid to the status of the cardiac function since digitalization may be required to overcome dependent edema in patients with cardiac decompensation. If the arterial supply to the part is greatly diminished by arteriosclerosis, it may be wise to place the injured leg in a slightly dependent position to augment the local blood supply. The presence of anemia and decreased blood volumes should be determined and corrected. There is suggestive evidence that the poor wound healing observed in severely anemic patients is probably secondary to protein and vitamin deficiencies rather than to the anemic state itself.

Nutrition. A normal physiologic status is essential for a normal and most rapid rate of wound healing. Alterations in the

from contusion, congestion, infection or toxins from bites of poisonous animals serves to cut down the local supply of blood. Whatever the cause, impairment of wound healing occurs.

Signs of retardation in healing appear. The formation of granulation tissue is retarded and the tissues become pale. If blanching is produced by digital pressure, the return of circulation is sluggish. The skin is cool and wounds are slow to epithelize and contract. The line of incision shows little hyperemia and is easily separated. The open wound shows pale, wet, soft granulations which grow sluggishly or fail to grow at all. Purulent exudation is minimal, but bacterial growth is likely to increase tissue destruction. Skin flaps are readily lost and grafts may fail to take or are easily dislodged by activity.

Elective surgery should not be attempted on tissues known to have poor blood supply. In an emergency everything possible should be done to improve the condition accounting for the disorder.

In the care of wounds of violence involving tissues which are otherwise normal, the surgical therapeutic effort to remove devitalized tissues from the margins of these wounds is based in part on the appreciation of the contribution of a good blood supply toward prompt healing. Apposition of tissue with an assured supply of blood is most desirable. Flaps with precarious blood supply must not be impaired with constricting bandages and tight or excessive sutures. Excessive scar tissue with deficient circulation which affects the recovery of a part should be excised to a good source of blood and the defect corrected by a suitable graft.

When ischemia develops in tissues which have no impairment of sensation marked pain develops. Whether it is produced by an arterial embolus tight dressing, tight cast, tight sutures or a distended wound, the mechanism is similar. This can be appreciated through two simple experiments. Place a blood pressure cuff about the arm and inflate it to a degree well above the systolic pressure. Record the time and see how long it can be tolerated. Second, sit in a chair and obtain the most comfortable position possible. Extend the legs on a foot stool if you wish, and pick up an interesting book. From then on do not change the position. When ischemia begins to develop over the heels, calves, knees, ischial tuberosities, sacral area or over the greater trochanter of the femur because of the continued pressure the pain

will become disturbing enough to interfere with the reading. Record the time, and do not move. When it becomes most unbearable, think of the infant being given an intravenous injection in a leg which is strapped too tightly to a splint, or the patient who has a lump in his cast, or the bedridden creature who cannot change his position but is turned every four hours by a nurse. What part of four hours can you sit without moving?

Edema and Lymphatic Obstruction. When stasis of lymph and venous blood begins to pile up in a wound, the efficiency of healing begins to wane. When lymph flow is obstructed, edema results. When venous flow is obstructed, venous engorgement and edema result. Oxygen tension in the tissues is reduced, carbon dioxide builds up, the pH falls, tissue edema increases and more stress is placed upon the lymphatic channels. Tissue metabolism slows and the by products of metabolism begin to accumulate in the tissues to produce a sense of fatigue and malaise.

Lymphatic or venous blockage produces swelling which is detrimental to wound healing and favors bacterial growth. The drainage of open wounds increases. Vesicles may appear in the skin which may break down and form open wounds. Tissues, boggy from edema, will have a restricted blood supply. Capillaries and venules which do not become occluded with clot will be obstructed by pressure. Fibrosis in tissue is enhanced by stasis but fibrosis is not a desirable reaction when it extends beyond the healing of the wound itself. Fibrosis in tissues about a wound weakens the resistance of those tissues to future injury and retards the repair of those injuries.

Excellent examples of the effects of lymphatic and venous stasis are found in the lower extremity. Injuries in the lower third of the leg heal more slowly than in any other part of the body. Acute and chronic ulcerations occur frequently and become complicated by fibrosis and calcification. While other factors enter into the problems of wound healing for the lower extremities, stasis of blood and lymph is a common denominator of all of them.

The part played by stasis in delaying the healing of wounds is evident when one observes an ambulatory patient's leg ulcer of long duration heal rapidly as soon as the patient is placed at bed rest with the extremity elevated. The improvement which comes with bed rest is the result of correcting the stasis accompanying the upright position.

veloped. In some instances incisional hernias developed. Microscopic examination of the scarred areas showed degenerative changes in the collagen.

Some of the components of vitamin B complex should be added to large doses of vitamin C and given to patients after injury or operation. It is suggested that during a period of acute stress after injury and for two or three days afterwards the patient should receive 1 gm. of vitamin C 50 mg. of nicotinic riboflavin and 500 mg. of nicotinic acid daily. Thereafter smaller doses of the vitamins are recommended until recovery is complete.

One of the fundamental roles of vitamin A in the body is concerned with maintenance of the integrity of epithelial tissue. Lack of vitamin A may lower local resistance to infection. Since this vitamin unites with proteins to become an essential constituent of all specialized epithelial tissues adequate stores are essential for epithelization following operations or injuries. In indolent ulcerations, in which the infection has been completely controlled inhibition of wound healing may persist until large doses of vitamin A are given systemically. On the other hand, the contention that topical applications of vitamins A and D in the form of cod liver oil stimulate wound healing has not been proved.

Vitamin K has an essential role in the control of hemorrhage in relation to a deficiency in prothrombin known as hypoprothrombinemia. It is of great importance in patients who are jaundiced or who have marked hepatic insufficiency. Continued bleeding in wounds of such patients will result in improper or delayed healing. Under such circumstances, the administration of vitamin K is of great help in producing effective clotting of blood and in preventing continuous oozing into the wounds.

Adequate intake of vitamin D is required for the proper absorption of calcium and its deposition in bones. At times it is desirable to pay special attention to the intake of vitamin D in connection with the healing of wounds or infections involving bones. Liver impairment following surgical procedures may interfere with the absorption and utilization of vitamin D. The need for supplementation which exists following ordinary operations becomes greater when bones are involved in the lesion.

Temperature. Temperature has an effect on the growth of cells and therefore, on wound healing. In tissue cultures, this effect

is well known. It is probable that the ideal temperature for the healing of a wound is the normal body temperature and that every effort should be made to approximate it with regard to dressings and atmospheric conditions for a wound. Patients subjected to refrigeration anesthesia or refrigeration treatment for prolonged periods may show a marked tendency toward nonhealing.

Rest. Rest of the part is of considerable importance, particularly in relation to wounds of the extremities in the region of joints. Motion of the area by joint or muscular activity disturbs the medium in which the cells are growing and invites extravasation of blood and fluid. However in abdominal wounds the experience of the past fifteen years has amply demonstrated that the healing of properly sutured wounds is not retarded significantly by early ambulation.

Metabolic diseases. Metabolic diseases such as diabetes and portal cirrhosis may be factors which inhibit wound healing. In the case of uncontrolled diabetes a decreased resistance to bacterial infection may increase bacterial destruction of tissues in wounds and cause protracted delays in wound healing. In addition, obliterative peripheral arterial disease associated with diabetes may affect the local blood supply and the process of healing. In portal cirrhosis hypoproteinemia, hypoprothrombinemia and vitamin deficiency states may influence the rate of healing of wounds.

Uremia. The experience of the Research Team of the U S Army in Korea during the Korean War showed that wound healing was significantly retarded in uremic states which developed in some battle casualties as the result of massive tissue necrosis or infection. Wound healing in civilian patients with uremia likewise appears to be retarded. The reasons for this are not clear.

HEALING OF SPECIAL TISSUES

Epithelium has marked powers of regeneration and injuries involving the skin and mucous membrane are usually followed by rapid repair and more or less complete restoration of the typical cellular arrangement produced by regeneration. Mucous membranes of the squamous type behave much the same as skin.

Tendons heal by regeneration through the process of granulation. A moderately rapid growth of granulation tissue replaces the exudate between the sutured ends of tendons and fibroplastic proliferation occurs within the granulation tissue. New collagen tissue

state may have deleterious effects on the healing process and must, therefore, be corrected. Malnutrition or diet deficiency may be the cause of lowered resistance. In patients Difficulty in maintaining cellular nutrition may result in failure of the body to mobilize the defense mechanism and may even cause local conditions favoring the growth of invading bacteria.

In some patients with infections it has been found that there is a complement deficiency in the blood. This is a nonspecific element in the blood necessary for defense against invading bacteria.

A deficiency in protein, known as *hypoproteinemia* inhibits fibroplasia within the granulation tissue. Hypoproteinemia may be caused by prolonged protein starvation or by loss of protein following severe or repeated hemorrhage, prolonged sepsis continued inflammatory exudate, drainage from fistulae, cirrhosis of the liver or ascites. It is frequently associated with tissue edema produced by a loss of fluid from the capillary bed into the intercellular spaces. In the presence of protein deficiency the edges of a wound appear soggy with edema and they may give no evidence of fibroplasia as late as the eighth to fourteenth day after injury. In wounds of rats suffering from protein depletion it has been shown that the quantity of mucopolysaccharides and collagen per gram of tissue is less and that the time required for their production is increased over that for normal animals. This may result in disruption of abdominal wounds. It is essential to determine the presence of hypoproteinemia by measuring the values for serum protein and to combat any hypoproteinemia by transfusions of plasma or blood and a diet rich in protein before and after operation.

During the period immediately after injury or operation, a state of negative protein balance is common. An increased protein intake may restore a positive balance and provide more adequate nutrition.

Lyons has emphasized that delayed wound healing may be observed in patients with a reduced total serum protein volume and a reduction in blood volume associated with severe wounds or protracted illnesses. Such patients are also in negative protein balance. The observed serum protein and hemoglobin levels may be normal in patients with decreased circulating blood volume. Under such conditions, the administration of liberal amounts of blood to patients who have lost

10 to 20 per cent of their body weight usually is followed by improved wound healing.

Agammaglobulinemia is a state of deficiency of a special protein, *gamma globulin* in the blood which decreases the patient's resistance and favors the development of infections resistant to treatment.

Vitamins. It is well recognized that surgical procedures and injuries diminish hepatic function and that the liver is concerned with the metabolism and storage of most of the vitamins. Replacement therapy is generally considered helpful in maintaining the body at optimum efficiency since some of the vitamins play an important role in the healing of wounds.

Several of the vitamins have been shown to have special significance in the relation to healing of the wounds and infections. *Vitamin C* is required for the production and maintenance of the intercellular cement, especially in the capillary bed and in the collagenous tissues. Lack of or deficiency of, intercellular substance in the granulation tissues and capillary bed results in hemorrhage into the wound space and into the bordering tissues, prolongs the lag period, retards fibroplasia and delays the development of adequate tensile strength in the wound. A study by Dunphy of surgical wounds made in rats living upon diets deficient in vitamin C showed that fibroplasia progressed well and ground substance was present in adequate amounts. The animals were unable to utilize the basic materials for the production of collagen, however except in the presence of vitamin C.

Vitamin C is also involved in protein metabolism through its effect on tyrosine and phenylalanine metabolism. Experimental studies of Lind and Crandon indicate that minor degrees of vitamin C deficiency are not very important from the standpoint of wound healing. However prolonged deficiency is associated with marked interference with the healing process. Like hypoproteinemia, vitamin C deficiency may be a causative factor of wound disruption or dehiscence. Vitamin C is essential to the normal healing of wounds and to the maintenance of their strength. Pirani and Levenson have reported evidence that scar tissue seems to be more sensitive to vitamin C deficiency than is normal connective tissue. In normal guinea pigs who were injured, allowed to heal for six weeks and then started on diets deficient in vitamin C, the previous scar became swollen and hemorrhagic when scurvy de-

- Hartzell, J. B., WinBeld, J. M., and Irvin J. L.: Plasma Vitamin C and Serum Protein Levels in Wound Disruption. *J.A.M.A.* 116:669 1941
- Harvey S. C.: The Healing of the Wound as a Biologic Phenomenon. *Surgery* 25:655 1949
- Howes, E. L., and Harvey S. C.: Tissue Response to Catgut Absorption, Silk and Wound Healing. *Internat. J. Med. & Surg.* 43:225 1930
- Howes, E. L., Plotz, C. M., Blunt, J. W., and Ragan, C.: Retardation of Wound Healing by Cortisone. *Surgery* 28 177 1950
- Koch, S. L.: Injuries of the Parities and Extremities. *Surg., Gynec. & Obst.* 76 1 189 1943
- Lawman, T. H., and Ingalls, T. H.: Vitamin C Deficiency and Wound Healing. *Ann. Surg.* 105:616, 1937
- Mason, M. L., and Shearon, C. G.: The Process of Tendon Repair. *Arch. Surg.* 25:615 1932.
- Menkin, V.: Mechanism of Leukocytosis with Inflammation. *Arch. Path.* 30 363, 1940.
- Moore, F. D., and Ball, M. R.: Metabolic Response to Surgery. Springfield, Ill., Charles C Thomas, 1932.
- Penney J. R., and Balfour B. M.: Effect of Vitamin C on Mucopolysaccharide Production in Wound Healing. *J. Path. & Bact.* 61:171 1949
- Pirani, C. L., and Levenson, S. M.: Effect of Vitamin C Deficiency on Healed Wounds. *Proc. Soc. Exper. Biol. & Med.* 82:95 1953.
- Pohle E. A., Ritchie, G., and Motz W. W.: Studies of the Effect of Roentgen Rays on Healing of Wounds. Histological Changes in Skin Wounds in Rats Following Postoperative Irradiation with Very Small and Moderate Doses. *Radiology* 52:707 1949
- Prudden, J. F., Lane, N., and Meyer K.: Lysozyme Content of Granulation Tissue. *Proc. Soc. Exper. Biol. & Med.* 72:38 1949
- Sandblom, P.: Effect of Injury on Wound Healing. *Ann. Surg.* 129:305 1949
- Stevenson, J., and Reid, M. R.: Treatment of Traumatic Wounds. *Am. J. Surg.* 46:442, 1939
- Ziffren, S. E., and May S. C.: Effect of Various Enzymes on Infected Wounds and Necrotic Tissue. *S. Forum* (1950) pp. 405-410 1951
- Zintel, H. A.: The Healing of Wounds. *S. Clin. North America* 26 1404 1946.

is formed and produces firm union of the severed ends. Studies of Mason have shown that the tendon ends start to proliferate about the fourth or fifth day after suture and to send out bands of cells and fibrils into the adjacent tissue. If the tendon is injured or if it is stripped of its blood supply necrosis is likely to occur and delay the process. After the fourth or fifth day the tendon itself begins to proliferate and to send cells into the coagulum. If the gap is not too great, it may be bridged by proliferating cells of the tendons themselves.

Muscle does not regenerate but heals by granulation and scar tissue formation. Proliferation of fibrous tissue from the muscle sheath and the intermuscular septa forms new fibrous collagenous tissue which unites the separated muscle and restores its function. A minimal amount of scar tissue necessary to fill the wound defect in the muscle is advantageous, since it is apt to produce less interference with the function of that muscle. Healing of muscle injuries will not progress until all necrotic and devitalized muscle is removed.

Bone heals by regeneration. It has remarkable powers of healing after injury. A clot first forms between the two ends of fractured bones. Thereafter typical proliferation of granulation tissue develops from the endosteum, the periosteum and the haversian canals, along with the formation of capillary buds. The osteoblasts in the periosteum and endosteum proliferate and lay down new osteoid tissue. The clot is absorbed and replaced by vascular tissue and new osteoid tissue. Calcium salts are then deposited in the osteoid tissue making it firm and known as *callus*. The superfluous, or excessive external callus is largely or completely absorbed until the normal contour of the bone is restored. Healing occurs more rapidly in long bones than in flat bones. Bones with little or no periosteum are apt to heal slowly. Immobilization of fractured bones is particularly essential for healing. The repair of bone at the site of injury or infection may be so complete that it may be impossible to detect after one year.

Cartilage may or may not heal by regeneration, depending upon its location. Articular cartilage in joints does not regenerate whereas costal cartilages can regenerate provided the perichondrium is present and viable. Only the latter produces parent cells capable of producing adult chondrocytes which reproduce cartilage.

Composed of mesothelium, serous mem-

branes such as the peritoneum, have great powers of regeneration. Denuding of peritoneum from large areas of the wall of the abdominal cavity has been shown to be followed by early regeneration of the peritoneal surface.

As soon as the surfaces of the mucous membrane of the *gastrointestinal tract* are united by suture an exudate forms and seals the union. This exudate is invaded by granulation tissue from the subserous connective tissue. Healing is usually more rapid than in other epithelial tissue. The united mucosa which has been inverted into the lumen by the suture undergoes more or less necrosis along the suture line. As this sloughs off, however granulation tissue forms and is then replaced by new mucous membrane which may in every way resemble the normal tissue. Thus, healing is by granulation of the entire wall except the mucous membrane where it is by epithelial regeneration. Mucous membrane of the *gastrointestinal tract* has exceptional powers of regeneration.

Components of *nervous tissue* do not regenerate unless a neurilemma sheath is present. Nerve cells which have been destroyed do not regenerate. However the various fibers of the cell may regenerate when the cell itself remains viable. Division of a peripheral nerve may be followed by growth of the axon in a peripheral direction and function may be restored. The connective tissue which grows more rapidly than the nerve fibers may surround them and impede their further growth. Wounds of the brain and spinal cord heal by the proliferation of the glial-supporting tissue.

READING REFERENCES

- Akerscoler W. A. Treatment of Fresh Traumatic Wounds. J.A.M.A. 124 406 1944
 Altemeier W. A., and Culbertson, W. R. The Prevention and Control of Surgical Infections. S. Clin. North America 35 1645 1955
 Altemeier W. A., Gunnell, J., and Stevenson, J. Wound Infections, in Bowers, W. F. ed. Surgery of Trauma. Philadelphia, J. B. Lippincott Co 1953, sec. 5 80-102.
 Arey L. B. Wound Healing. Physiol. Rev 16 327 1936.
 Cole, J. W. Shaw D. T., and Fraser P. Cutaneous and Serum Inhibition of Hyaluronidase; Experimental Study. Surg. Gynec. & Obst. 90 289 1950
 Cole, J. W. and others: A Histological Study of the Effect of Cortisone on Wound Healing per Primam. Surg. Gynec. & Obst. 92:321 1951
 Dunphy J. E., Udape, K. N. and Edwards, L. C. Wound Healing. Tr. Am. S. A. 74 16, 1956.
 Findley C. W. Jr. and Howes, E. L. Effect of Edema on Tensile Strength of Incised Wound. Surg., Gynec. & Obst. 90:666 1950

crossed rather than decreased the necessity for precise diagnosis. Specific infections, and not clinical syndromes must be treated

NATURAL MECHANISMS OF NONSPECIFIC RESISTANCE TO INFECTION

The human body is constantly exposed to a variety of microorganisms. Certain varieties are resident normally on the external and internal surfaces of the body. Others are present on inanimate objects in the environment. In health, man lives in equilibrium with the microorganisms to which he is constantly exposed. This tolerance is due to a system of anatomic, physiologic and cellular defenses which afford protection against the microbial hazards of everyday life.

The anatomic (mechanical) barriers to invasion by microorganisms are both external and internal. The external defenses include the epithelial surfaces of the skin, respiratory tree, gastrointestinal tract and urinary tract. Hair follicles and secreting glands in skin and mucous membranes are potential avenues of invasion of bacteria into deeper structures. To ensure protection against infection by external barriers, clean wounds are promptly closed by suture or skin graft. Examples of internal barriers are the fascial planes, the muscle compartments and the serous membranes, such as pleura and peritoneum. These internal barriers govern, to some degree, the direction and spread of infection.

Physiologic defenses against infection which dilute or discharge bacteria are exemplified by the washing and diluting action of tears, saliva, mucous secretions, gastric juices and bile, and by the mechanical emptying of the intestinal and urinary tracts.

The principal cellular defenses are the phagocytes and reticuloendothelial cells which clear bacteria and their products from the tissues and the blood stream. Their action is enhanced by circulating antibodies (globulins) and other nonspecific antimicrobial systems, such as complement. The production of specific circulating antibodies, as in the response to the injection of tetanus toxoid, is the basis for attempting to immunize an individual against a particular disease.

PRINCIPAL MICROORGANISMS OF SURGICAL IMPORTANCE

The great majority of diseases are caused by microorganisms which come from human carriers. Others are caused by microorganisms from sources external to the body. In

most situations the nature of the offending microorganisms is more important in determining specific therapy of infections than is their source. For this reason, knowledge of the principal types of microorganisms and the infective processes associated with their activities is of fundamental importance in diagnosis and treatment.

The three major groups of microorganisms that have a causal relationship to infections are the bacteria, the fungi and the viruses. Fortunately most bacteria and fungi can be stained easily and studied by relatively simple techniques and their sensitivities to chemotherapeutic agents can be estimated in vitro. Bacterial infections are encountered most frequently. Bacteria may be gram-positive, retaining crystal violet or gram-negative, releasing crystal violet and taking on the counterstain. They vary in size and shape and, in this respect, are grouped as cocci, bacilli or spirochetes. They may grow aerobically or anaerobically. In clinical infections they may be present singly as in hemolytic streptococcal cellulitis or they may be present in a combination of several species as in acute secondary peritonitis, bronchiectasis and burn sepsis.

GRAM-POSITIVE BACTERIA

Staphylococci. The most numerous infections are those caused by the ubiquitous staphylococci, such as *Staphylococcus aureus* or *albus*. Staphylococci are known to produce a whole range of metabolites: hemolysins, leukocidin, fibrinolysin, hyaluronidase, coagulase and enterotoxin. In some way these metabolites influence the diverse manifestations and course of staphylococcal inflammations. There is hardly a tissue or system of the body which may not be the site of staphylococcal involvement. These organisms manifest themselves clinically in three principal forms.

Circumscribed purulent necrotic lesions are the most common and may involve any site of the body. They may be localized, locally invasive or associated with bacteremia, with or without metastatic abscess. Response to treatment characteristically is slow and the staphylococci persist until cure is complete. Antibiotic sensitivities vary according to the antibiotic and the duration and intensity of its communal usage. Bacteremia may also arise as a complication of cannulation of a vein and intravenous administration of fluids. The lesions of soft tissues seen most frequently are paronychia, furuncles, carbuncles and postoperative

INFECTIONS

By EDWIN J. PULASKI, M.D.

EDWIN J. PULASKI is at present Chief of the Department of Surgery at the United States Army Hospital, Fort Dix, New Jersey, and holds the rank of Colonel in the Army Medical Corps. Born in Pennsylvania, he graduated from the University of Pennsylvania and its medical school. After his residency training in surgery at Columbia University Presbyterian Hospital, he devoted himself to the study of surgical bacteriology. He was the founder and first chief of the Army Surgical Research Unit and chief of the Army's streptomycin research program. He has continued to contribute to the knowledge of infections and their response to various methods of control.

The term "infection" denotes an inflammatory condition attending the entry and multiplication of microorganisms in the body. There are many factors from which are derived the principles of prevention and treatment of those infections of concern to the surgeon.

The symptoms and characteristics of clinical infections differ greatly with the species and virulence of the causal pathogens, the tissues and anatomic parts of the body affected, the general condition of the individual and the extent and duration of the infection. The presence of microorganisms on the skin and mucous membranes of the body and in fresh traumatic wounds is not "infection" but is "contamination." Patients who require surgical procedures in such situations are referred to as "contaminated cases." The distinction between contamination and infection is sometimes difficult. Adverse changes may be manifest only by delayed healing, excessive scarring or failure of "take of skin grafts.

In the prechemotherapy era, infections were designated as "surgical" when they were treated by operation. Since the avail-

ability of chemotherapy, this designation has excluded many cases that formerly would have been included in the surgical category. At present there are three types of infections important to the surgeon: (1) suppurative inflammations having a course profoundly influenced by surgical intervention, (2) infections complicating operative procedures, and (3) incidental or concurrent infections in body sites remote from the field of operation.

The present knowledge of prevention and treatment of microbial diseases stems from the development of aseptic techniques since Lister and from advances in bacteriology, immunology and antimicrobial therapy. The greatest advance has been the discovery of the antibiotics. As a result, a significant decline has occurred during the last two decades in the once uncontrolled streptococcal, pneumococcal and clostridial infections among patients in surgical wards. Other infections, nevertheless, have not disappeared since the advent of antimicrobial therapy and their threat remains a continued source of anxiety in all fields of surgical endeavor. Furthermore, antibiotic therapy has in-

of animals and man. The germination of implanted spores and multiplication of vegetative bacteria can come about as a result of ischemia, the presence of calcium salts (soils) the deoxygenation of tissue by proliferating aerobic bacteria or by autolysis of dead tissues. Toxigenic clostridial infections are unique since the clinical picture is determined by their toxins and products of breakdown of involved tissues. Prevention and treatment are surgical, immunologic and antibiotic.

Under proper conditions *Cl. tetani* proliferates at the site of its lodgment without any characteristic changes in the local wound. It elaborates a toxin of extreme potency. The incubation period is four days to three weeks or longer depending somewhat on the character, extent and location of the wound. The toxin passes along peripheral nerves to reach the anterior horn cells where its action results in characteristically painful muscular contractions, primarily of the masseter and neck muscles, and secondarily of the trunk. These contractions can be reproduced in experimental animals by injecting purified toxin. Active immunity is produced by tetanus toxoid, passive immunity by tetanus antitoxin.

Clostridial myositis (gas gangrene) is a rapidly spreading edematous myonecrosis developing in severe wounds of muscle and bone. It results from the invasion of ischemic muscle by spore-bearing organisms of the genus *Clostridium*, particularly *Cl. perfringens*, *Cl. novyi* and *Cl. septicum*. *Cl. perfringens* occurs most frequently. Changes in other organs include necrosis in the kidneys and degenerative changes in the liver. A filtrate of *Cl. perfringens* contains a galaxy of enzymes including alpha toxin (lecithinase), leukocidin, fibrinolysin, collagenase and hyaluronidase. Experimentally the principal lethal factor is alpha toxin (lecithinase) affecting capillary endothelium and sarcolemma. This factor plus the modification of the ground substance by the action of hyaluronidase and fibrinolysin is responsible for the development of wet gangrene. Collagenase also plays a part in the local spread of clostridial infection. The widespread metabolic disturbance is probably associated with the peripheral vascular failure that is a late characteristic of the disease. Since the availability of penicillin, enthusiasm for polyvalent gas gangrene antitoxin therapy has waned. Toxoids for active immunity are in the developmental stage.

Clostridial cellulitis is a serous crepitant

process of subcutaneous retroperitoneal or areolar tissues produced by one or more of the clostridia, usually *Cl. perfringens*. In contrast to gas gangrene muscle invasion is absent and systemic effects are less pronounced.

Puerperal clostridial septicemia is an infection which becomes lethal rapidly unless vigorously treated and is characterized by sudden intravascular hemolysis and peripheral circulatory failure.

THE GRAM-NEGATIVE BACILLI

The principal gram negative bacilli of surgical importance are the coli-aerobacter aerogenes groups and the various species of *Proteus* and *Pseudomonas*. They constitute a large part of the normal intestinal flora and ordinarily they are not harmful within the colon. When they reach tissues outside the intestinal tract they cause disease particularly in the lungs, the urinary apparatus, biliary tree, peritoneum, blood stream, meninges and necrotic tissues. Antibiotic sensitivities of gram-negative bacilli vary with the species and individual strains within species. Treatment is directed at correcting underlying pathology, eradicating the focus of infection and adjunctive antimicrobial therapy.

Gram-negative infections of the urinary tract are manifested by inflammation and smooth muscle spasm, dysuria and pyuria.

Infections of the biliary apparatus usually accompany inflammations due to obstruction or biochemical disorders.

Acute bacterial peritonitis is usually polybacterial; the gram-negative bacteria constituting an important part of the total flora. Included are anaerobic forms (*Bacteroides* species) which are present also in mixed putrid infections of wounds and of the perineum. *Escherichia coli* and other coli forms do not produce a foul odor when growing in sterile pus. The putrid odor of appendiceal peritonitis is primarily the result of proliferation of anaerobic bacteria, principally *Clostridia*, *Bacteroides* and anaerobic streptococci.

The usual portal of entry for gram-negative bacilli causing bacteremia is the genitourinary tract, the gastrointestinal tract or an extensive thermal or traumatic wound. Bacteremia from these organisms may also be caused by transfused, contaminated whole blood or unsterile solutions used parenterally. The protein-lipid polysaccharide endotoxins of gram-negative bacilli all have similar effects following intravenous admin-

wound infections. Parotitis and abscesses of dental origin are frequently staphylococcal. The soft tissue infections may be precursors of more serious infections such as bacteremia, osteomyelitis, meningitis or pneumonia. Metastatic staphylococcal abscesses may occur in the brain, lung, kidney perirenal spaces or prostate.

Another form is staphylococcal enterocolitis, a widespread membranous lesion occurring in the intestinal tract as a sequela of overgrowth of staphylococci, as an incident of antibiotic therapy or from some obscure cause following resection of the stomach or colon. Its features are cholera form diarrhea, circulatory collapse and passage of sloughed mucosa.

The third form is the severe shocklike syndrome of staphylococcal food poisoning attributed to preformed enterotoxin elaborated by staphylococci.

Staphylococcus aureus is now classified by bacteriophage typing of individual strains into four broad groups (I II III and IV). Each group is further comprised of a large number of subtypes. No particular group of staphylococci is exclusively associated with any type of staphylococcal lesions. Strains resistant to penicillin and other antibiotics frequently are of the Group III variety but not exclusively. Bacteriophage typing is a valuable tool in the epidemiologic investigation of outbreaks of staphylococcal infection.

Streptococci. Streptococci are classified according to their ability to hemolyze the blood added to agar plates. Beta hemolytic streptococci elaborate an array of substances—erythrogenic toxin streptolysin leukocidin, hyaluronidase, streptokinase and deoxyribonuclease. Erythrogenic toxin is associated with the rash of scarlet fever. Hyaluronidase assists in the penetration of intracellular cement substances by hydrolyzing mucosubstances. Streptokinase and the ribonucleases are capable of dissolving fibrin and liquefying purulent exudates. In purified form, they find therapeutic application for liquefying blood clots and thick pus. These multienzymatic activities explain the spreading character of hemolytic streptococcal cellulitis and lymphangitis. Colonization of granulating wounds, so-called silent infections by beta hemolytic streptococci, is one cause of failure of skin grafts to "take." The organisms produce a lysis of the fibrin "cement" that fixes the graft to the wound surface.

The resistance of hemolytic streptococci to antimicrobial agents is rare. The presence

of a microaerophilic hemolytic streptococcus is associated with a rare, progressive ulceration of the skin and subcutaneous tissues—the chronic, burrowing, undermining ulcer described by Meleney. Sensitivity of this organism to antibiotics varies.

Nonhemolytic and alpha hemolytic (*Streptococcus fecalis* *Streptococcus viridans*) streptococci are usually less virulent than the beta hemolytic variety. Nonhemolytic streptococcal infection does not have characteristic clinical features. For this reason, identification of the organisms and culturing for sensitivity to antibiotics are important as guides to therapy. Singly or in combination with other organisms the nonhemolytic streptococci and *Str. viridans* are associated with the abscesses of dental caries, liver abscesses, subacute bacterial endocarditis and infections of the urinary tract. They are frequently found in the peritoneal exudate following perforations of the duodenum and in empyema of the gallbladder. They are present in the mixed flora of peritonitis following perforation of the appendix or colon.

Anaerobic streptococci, singly or in combination with other forms of bacteria, are associated with streptococcal myositis, puerperal sepsis, perirectal and intra-abdominal abscesses, wound infection from a human bite, chronic synergistic gangrene of the abdominal or chest wall and lung abscess. Their presence should be suspected when streptococci are found microscopically in stained smears of pus but are not recovered bacteriologically in routine aerobic cultures.

Mycobacterium Tuberculosis. The tubercle bacillus is a gram positive and acid fast organism producing the well-known disease of tuberculosis. The surgeon encounters this disease most frequently in the pulmonary lymph node, abdominal, renal and osseous forms.

Bacillus Anthracis. An important aerobic pathogenic gram positive bacillus *B. anthracis* is the causative agent of anthrax (malignant pustule) sometimes confused with pyogenic coccal infection of the skin.

Spirochetes. These are less commonly encountered in surgery than the cocci and bacilli. Those of major surgical importance are the *Treponema pallidum* of syphilis, the *Borrelia cincenti* of Vincent's angina and the *Spirillum minus* of rat-bite fever.

Toxicogenic Clostridia. *Clostridium tetani* and the group of toxigenic clostridia associated with gas gangrene are widely distributed in nature, including soil and feces

bacteria indigenous to a particular area of the body as in acute appendicitis.

Resistance to infection may be decreased by mechanical or functional disturbances or by a combination of these two factors. Examples of mechanical disturbances are wounds of violence, thermal injuries congenital anomalies, and obstructions of organs, such as the bronchus, the ureter, the common bile duct and the appendix. Examples of functional disturbances are biochemical inflammations, such as phlebitis, cholecystitis and allergies in the skin or respiratory tract. In a few instances bacteria may penetrate mucous membranes without trauma, as in typhoid fever, amebic or bacillary dysentery and tuberculosis. Infection (superinfection) may also arise as a complication of antibiotic therapy in the absence of prior tissue damage, e.g., staphylococcal pneumonia, diarrhea or secondary infection in the urinary tract.

Bacteria not only must enter but also must multiply in tissues before they can produce harmful effects. The microorganisms freshly implanted in tissues may be so few in number or of such low pathogenicity that they are destroyed by defensive mechanisms of the body without clinical evidence of inflammation. This accounts for the failure of infection to develop in every open wound. If the number and character of the invaders are sufficient to overpower the resistance of the tissues, the bacteria multiply; they also release toxic metabolites and gain a foothold at the site of lodgment.

Local Effects. The response of the host is to localize and eradicate infection by means of inflammation—a reaction which manifests itself locally and systemically. The proliferating microorganisms and their metabolites act as stimuli to which the tissues respond by a series of interrelated progressive phenomena: (1) localized cellular destruction, (2) increased capillary permeability and formation of edema, (3) stickiness of the endothelium and (4) margination of leukocytes and entrance of various cells into the injured tissue. One or another phase may be inconspicuous or predominant, depending upon the bacterial species.

The reaction of inflammation is an attempt on the part of the body to localize the proliferating bacteria at their portal of entry and thus prevent their general dissemination. Globulins, antibodies and fibrinogen pass into the affected area, creating a fibrin network and thrombi, which prevent the infected material from leaving the area. Cer-

tain species of bacteria may enhance fibrin formation (staphylococci); others may retard its formation or actually destroy it (*Cl. perfringens*, hemolytic streptococci).

The effort at fixation is accompanied by the accumulation of phagocytes and reticulo-endothelial cells. Phagocytosis is retarded by reduced blood flow as in shock and reduced body temperature, and by deprivation of oxygen as in larger accumulations of pus.

As the leukocyte wave deteriorates, macrophages from the local tissues and the blood make their appearance. They increase in number remarkably and actively phagocytose and digest the invading bacteria. Specific antimicrobial therapy, by means of bacteriostatic or bactericidal action, assists phagocytic activity. The development of macrophages into fibroblasts supplies the active elements for regeneration and repair, formation of scar tissue and the walling off of foreign bodies.

The longer a microbial inflammation persists, the more harmful its effect becomes. In addition to necrosis, abscess formation and retardation of absorption of exudate may prevent collapse and apposition of the tissues and may cause formation of a persisting sinus or fistula. In certain organs such as brain, liver or lung, function may be seriously impaired. Crippling adhesions may form on serous membranes such as pleura, joints or bowel.

Exudate may be absorbed by lymphatics; it may spread or be converted to pus or be vascularized, i.e., form granulation tissue. Stated in another way, the infection may evolve by (1) restoration of tissues to normal, as in hemolytic streptococcal lymphangitis; (2) localization, abscess formation, resolution and scar formation, as in carbuncle; (3) granulomatous reaction, as in any chronic inflammation, e.g., actinomycosis, or (4) dissemination of the infection. The route of spread may be by continuity, as in phlebitis by contiguity, as in osteomyelitis from paronychia, by lymphatics, as in streptococcal cellulitis, or via the blood stream through blood vessel erosion.

Systemic Effects. The metabolic alterations and systemic manifestations of infection are reflections of the extent and severity of the disease. Breakdown products of bacteria and tissue proteins act upon the heat-regulating center, altering the thermostatic level at which body temperature is regulated. Chills are associated with vasoconstriction. Failure to lose body heat results in fever. When vasodilation and sweating take place,

istration chills fever prostration and varying degrees of peripheral circulatory failure. Bacteremia is difficult to eradicate and may be lethal.

Gram negative bacillary meningitis produces no specific manifestations and is diagnosed by bacteriologic studies.

Many species of gram-negative bacilli cannot digest total proteins i.e. they cannot invade living tissues, but they can assimilate amino acids and peptides. Proteolytic enzymes in dead or devitalized tissue and in pus provide degraded proteins which act as a nutritional pabulum for the growth of gram negative bacilli. Their enzymes are capable of producing lysis of a blood clot and of exciting a discharge that is persistent and interferes with tissue repair. The discharge may be irritating and cause excoriation of skin. Drainage of pus and removal of dead tissue and debris are necessary for complete eradication. Scar formation, sinuses and fistulae are frequent sequelae of persisting gram-negative infections of soft tissues and bones.

Proteus like other gram negative bacilli, usually produces infection only when found outside its normal habitat—the intestinal tract. *Proteus* microorganisms tend to increase in number in the bowel when coliform bacteria are suppressed, e.g. after broad-spectrum antibiotic therapy. Diarrhea may result.

Pseudomonas species are pathogenic when introduced into areas devoid of natural defenses or in the presence of other pathogenic organisms. They cause infections of wounds and burns, meningitis when introduced by lumbar puncture, and urinary tract infection when introduced by catheters or unsterile solutions. A characteristic green pus is formed.

Gram-negative bacillary infections encountered less frequently are those caused by *Salmonella*, such as typhoid bacilli and paratyphoid bacilli. Their manifestations include intestinal perforation, cholecystitis, peritonitis or abscesses of soft tissues and bone. Other gram-negative bacillary infections include chancre caused by *Haemophilus ducreyi* and granuloma inguinale caused by *Donovania granulomatis*. Tularemia is caused by *Pasteurella tularensis*; the local manifestations being a nonhealing ulcer on the hands or face and enlargement of the regional lymph nodes.

FUNGI

Fungal diseases may be superficial or

deep seated. Epidermophytosis of the feet, an example of a superficial mycosis, is frequently the portal for spreading pyogenic infection. Erysipeloid, commonly known as "fish-handlers disease," is a cutaneous disease of the hands due to *Erysipelothrix rhusiopathiae* which affects those who handle dead animal matter. The deep mycoses, such as actinomycosis, blastomycosis, coccidioidomycosis, histoplasmosis and nocardiosis, are relatively uncommon. Fungal superinfection of the respiratory tract and bowel, e.g., moniliasis may follow protracted courses of antibiotic therapy especially in debilitated individuals.

The most common of the deep mycotic infections, clinical actinomycosis, is caused by two organisms *Actinomyces bovis* and *Nocardia*. Both produce granulomatous supuration and a predominance of abscesses and fistulae. Clinical actinomycosis is usually a mixed infection wherein identification can be made of *Actinomyces bovis*—the anaerobic ray fungus of Wolff Israel. Its clinical recognition lies in the observation of the sulfur granule characteristic of the fungus cluster in pus. *Nocardiosis* is an infection caused by a streptothrix organism, *Nocardia asteroides* being representative. True sulfur granules do not occur but particles composed of mycelial threads and fibrils are recognizable in pus. The organisms yield an abundance of mycelial forms on culture.

VIRUSES

The principal entities due to viruses encountered in surgical practice are herpes zoster, lymphopathia venereum, homologous serum jaundice and cut-scratch disease.

PROTOZOA

The principal surface infection resulting from animal parasites is caused by *Entamoeba histolytica*, the etiologic agent in amebic colitis, amebic hepatitis and abscess.

EFFECTS OF MICROBIAL INVASION

Requisites. The primary requisites for the development of infection in the body are alteration of the cellular environment and the entrance, establishment and multiplication of pathogenic bacteria in the locus. Infection may be associated with the entry of microorganisms into normally sterile tissues, as in a traumatic wound involving muscle or other deep structures, or it may be associated with conditions which support the growth of

gressive spread or beginning of localization and suppuration.

Exhaustion of pabulum, accumulation of metabolites and development of antibodies culminate in a decreased metabolic turnover and a decline in the number of microorganisms present. In pyogenic infection, a walling off and an accumulation of pus and necrotic debris result in abscess formation. At this stage, antibiotic therapy is supplementary to the release of pus and necrotic debris.

These three stages are important in the timing and response to chemotherapy. They also serve as guides for operative intervention and supportive therapy.

Usually laboratory studies are required to confirm the etiologic diagnosis. In addition to their antibiotic sensitivities, etiologic diagnosis implies the isolation and identification of the causal microorganisms in exudate, blood (blood or bone marrow culture) or biopsy material (tuberculosis or fungal granulomas). For quick presumptive diagnosis, as in actinomycosis, infection of the urinary tract, gonorrhea or wound infection, a stained smear of the exudate may reveal, in a matter of minutes, valuable information concerning the microorganisms present. Isolation of the microorganisms and culture sensitivity tests furnish the true criteria for the selection of antimicrobial agents.

In most acute phases, reliance upon laboratory results is obviously not possible before therapy must be initiated. Once these results are obtained, the proper therapeutic measures can be adopted. Culture sensitivity tests make possible correct selection of an antibiotic or a combination of antibiotics, early recognition of mixed infection and an indication of the expected course of the disease by virtue of this knowledge of the invasiveness, toxicity and biochemical properties of the microorganisms. Present knowledge of the action of antibiotics indicates that no effect from systemic antibiotic therapy is to be expected unless the microorganisms are sensitive *in vitro*. Moderate sensitivity suggests that, if an increase in the dosage is feasible and safe, a favorable therapeutic response is possible.

Whenever deviation occurs in the expected response to therapy or when secondary infection by antibiotic-resistant organisms is suspected, antibiotic culture-sensitivity tests should be repeated. Precise laboratory diagnosis is also imperative in all staphylococcal and nonspecific infections, such as those of the urinary tract which do not respond to a

trial of empiric antibiotic therapy within forty-eight to seventy-two hours, in recurrent or metastasizing infections and in acute and chronic infections in which the surgeon is unable to determine the etiologic agent by clinical evaluation.

Errors in Diagnosis. It must be stressed that the growth of bacteria in culture does not necessarily exclude the presence of another etiologic agent or disease. Staphylococci, streptococci or other microorganisms are frequently isolated from foci which actually have as their underlying basis fungal infection, tuberculosis, a neoplasm or other lesions. Positive cultures taken from them may represent contaminants or secondary invaders. Also in polybacterial infections such as acute secondary peritonitis, sensitivity data on aerobic organisms without accompanying anaerobic cultures may be misleading. Conversely the report of "no growth" may or may not be valid. If inoculation on suitable media was delayed, swabs containing exudate may have dried out. If few in number the organisms may have died in the interim. The culture media may not have been suitable and microorganisms, such as anaerobic species, may have been missed because proper culture techniques were not employed. Cultures may have been discarded before sufficient time had elapsed for the development of slow-growing organisms.

If the organisms cannot be demonstrated by repeated smear, biopsy or culture, and if microbial disease is strongly suspected, it may be necessary to resort to agglutination tests as in typhoid fever or to complement fixation and/or skin tests, as in tuberculosis or chancre. Other indirect diagnostic procedures include roentgenographic studies to test for the presence of infiltrating disease, congenital anomalies, acquired obstruction, sinuses or fistulae.

It is especially important to rule out causes of pain and fever other than those due to infection, which are not influenced by antibiotic therapy. Among these are virus infections of the respiratory tract, liver or central nervous system or mononucleosis, nonmicrobial inflammations, such as phlebitis or infarct, cholecystitis, regional enteritis and ulcerative colitis, collagen diseases, metabolic diseases, such as thyrotoxicosis or cirrhosis and neoplasm, blood dyscrasias, lymphomas, serum sickness and drug fever.

Local Therapeutic Measures. These include (1) protection from trauma (2)

the temperature tends to drop. As the body temperature increases, the respiratory rate and, to a lesser extent, the depth of respiration increase. The cardiac rate also increases.

Plasma and electrolyte loss may be reflected in hemocentration and other changes, such as reduced blood volume. The augmented excretion of nitrogenous products in the urine is evidence that protein destruction is increased. If peripheral vascular resistance is reduced to a significant degree, a sharp fall occurs in blood pressure and vasomotor collapse may occur.

The shock syndrome accompanying overwhelming infections may be due to causes other than effects on peripheral vessels. Other mechanisms which may cause shock are reduction in blood volume, direct effect of infection on the heart, adrenal insufficiency, anemia and vitamin deficiency.

COLLATERAL INFLUENCES ON THE COURSE OF INFECTION

Local Factors. The elimination of infection and institution of tissue repair are hampered when (1) the blood supply to the area is impaired, (2) blood clots, accumulations of serum, foreign bodies and devitalized tissue are present, (3) large accumulations of pus are under tension, cutting off the circulation and curtailing phagocytic activity, (4) rigid fibrotic walls are formed which impede collapse of tissue defects and sterilization of the cavity, (5) obstruction is present and impairs physiologic function of an organ such as in the bronchus, biliary tree or lower urinary tract, (6) the exposed surface wound is subject to reinfection, (7) superimposed trauma of any sort increases tissue damage still further, (8) movement delays localization and enhances the absorption of toxic products from the infected area, (9) dependent position of extremities slows resolution of edema, and (10) drainage of accumulated exudate and débridement of necrotic tissues are delayed or inadequate. The rationale of surgical treatment lies in the correction of these factors.

Systemic Factors. Certain systemic factors also influence the course of infection: (1) cardiovascular shock, anemia, agammaglobulinemia or bone marrow suppression from any cause; (2) metabolic starvation, uncontrolled diabetes or neoplastic cachexia; (3) hormonal Cushing's disease, stress of major surgical procedures, and (4) therapeutic prolonged treatment using corticosteroids, nitrogen mustard, irradiation, or

other bone marrow depressants or antibiotic therapy culminating in superinfection from alteration of the indigenous flora of the body. The rationale of supportive treatment lies in the correction of these factors.

PRINCIPLES OF TREATMENT

The basic principles of treatment of bacterial inflammations consist of early clinical and etiologic diagnosis, the institution of appropriate local and general supportive measures and specific antimicrobial therapy. Details of the application of these principles will necessarily vary with the nature and location of the infection. In general, however, some or all of them may be applied to produce a beneficial effect.

Early Accurate Diagnosis. The diagnosis of infection is based upon the history, clinical manifestations and the identification of specific etiologic microorganisms. Little difficulty is encountered when the clinical pattern conforms to that usually associated with classical infection of the body area involved. Obstacles to accurate etiologic diagnosis present themselves when infections are atypical or when they involve deep structures such as the neck and the cavity organs. Under such circumstances it may be necessary to call upon every appropriate diagnostic aid available.

Careful history of the illness and a physical examination may suggest the specific etiology of the infection. Microorganisms in the body develop and reproduce in a pattern roughly following their growth patterns in vitro.

The incubation period, or lag phase, is the interval between contamination of the tissues and the onset of infection. During this period, the reaction of the tissues to the products of bacterial metabolism has not progressed sufficiently to produce the typical local signs and symptoms of infection. Clinical manifestations during the incubation period are vague headache, malaise and fatigue. The incubation period may extend over a period of hours (hemolytic streptococcal sepsis), days (tetanus) or weeks (tuberculosis).

There is a phase of rapid, constant growth—the time of greatest microbial activity—when the organisms can be influenced most effectively by antibiotic therapy. This period is evident by the general and local clinical manifestations of inflammation—chills, fever, leukocytosis, pain, tenderness, discoloration, swelling and limitation of function. The peak of the disease is evidenced by either pro-

set up by bacteria causes destruction of tissue or is sufficiently active to produce a focus resulting in pronounced constitutional symptoms and signs. This is especially true when the active focus is in a structure not essential to the body, e.g., a congenital anomaly, the appendix, gallbladder part of a lung, uterine tube or lymph nodes.

Whenever dead space is present, its elimination is necessary for total eradication of infection, e.g., in sinus tracts, fistulae and cavities with rigid unyielding walls infected thyroglossal, branchiogenic, dermoid or pyloidal cysts and other foci of chronic infection.

Supportive Measures. Supportive therapy is designed to hasten the natural processes of recovery and to combat the effects of infection. The various measures employed are analgesics and antipyretics for relief of pain, fever and restlessness; administration of fluids, electrolytes, vitamins and calories to meet increased metabolic demands; in addition to normal losses, transfusions of whole blood to correct anemia, blood loss or reduced blood volume; gamma globulin to correct hypogammaglobulinemia, and vasopressor drugs to combat peripheral circulatory collapse. Further supportive therapy also includes concurrent treatment of underlying cardiovascular, metabolic or hormonal conditions.

Specific Measures. These include the administration of antitoxins, sera and antimicrobial agents. The purpose of specific therapy is to interrupt the natural course of the disease. Except for tetanus antitoxin in established tetanus, the use of vaccines, sera and antitoxins for the treatment of specific infection has been supplanted largely by specific antibiotic therapy. Vaccines and toxoids continue to be important, however, in inducing specific active immunity in the host.

PRINCIPLES OF ANTIMICROBIAL THERAPY

There are certain basic principles in utilizing antimicrobial therapy to combat infections.

The exact site of the inflammation must be localized from the history, from physical examination and from appropriate diagnostic procedures. The causative agent must be isolated and cultures for antibiotic sensitivities are to be taken whenever possible. In cases in which it is not possible to obtain cultures or in which the cultures cannot be

relied upon, the clinical syndrome must dictate empiric selection of the antibiotic.

The basic principles of management have not changed, nor is antibiotic therapy a substitute for tried and proved indications for surgical intervention. Antimicrobial therapy however has modified the time for application in certain situations.

Antibiotics must be given in doses sufficient to contact the causative organisms at the site of the lesion. This demands knowledge of the drug's pharmacologic and toxicologic properties and of the physiologic and pathologic barriers to its diffusion into the tissues. If no response has been noted in the usually accepted time, the indications for the use of the antibiotic should be reviewed critically.

A singly effective antibiotic is preferred whenever possible. The use of a combination of two or more antibiotics simultaneously administered separately or as a single mixed preparation should be based on bacteriologic studies showing that the infectious agent is resistant to single drugs, but susceptible to a specific combination. The principal indications for combination antimicrobial therapy are: (1) in the topical employment of nonabsorbable drugs of limited spectrum, such as polymyxin, bacitracin and neomycin, for mixed infections of skin and granulating wounds; (2) in the use of drugs with potentialities for rapid emergence of drug-resistant mutants during treatment, e.g., micrococcal infections treated with erythromycin or novobiocin and streptomycin; (3) in the treatment of desperately ill patients with suspected infections of unknown etiology until bacteriologic studies are available; and (4) in the treatment of staphylococcal bacteremia, meningitis or pneumonia, and in gram negative sepsis resistant to single drugs. When drugs are given in combination, full doses of each member of the pair should be given for optimal results.

Supportive therapy to bolster host resistance and correct deficits is a necessary complement to antimicrobial therapy. Steroid therapy is reserved for support of patients receiving these drugs preoperatively and for support of patients with septic bacterial shock until the lesion can be controlled by standard treatment.

The most important limitations and dangers of antimicrobial therapy are diagnostic errors, symptomatic rather than specific treatment, masking of serious infection with-

avoidance of secondary infection and super infection, (3) heat (4) incision and drainage (5) débridement (6) extirpation of infected foci, and (7) elimination of dead spaces.

Protection against trauma is provided by total body rest in serious infections and by local rest, induced by bulky compression dressings, splints, slings and adhesive strap-pings as in the case of extremities. Rest and immobilization also furnish protection of the lesion from additional trauma such as accidental bumping or striking of the area of infection and manipulation of the lesion by the patient or others. Gentleness during examination and changes of dressing is also a requisite as is application of bandages in such a manner that they cannot cause constriction or friction.

Protection against secondary infection implies special care during exposure of the open wound in order to avoid superinfection. It also requires avoidance of ingress of bacteria from dressings soaked through with exudate. Soaked dressings must be changed immediately.

Heat may be applied locally to increase blood flow and for comfort. The application of heat particularly in the form of hot soaks, has been supplanted largely by dry treatment since the advent of antibiotic therapy. Objections to using hot soaks are delay of incision and drainage until skin necrosis has occurred, predisposition to swelling, tendency of sodden tissues to narrow drainage exits, possibility of satellite infections from the wet environment and of superinfections from frequent exposure of the wound and danger of thermal injury. At present the principal use for moist heat is to loosen crusts and minute particles of devitalized tissue from the superficial dirty wound.

The purpose of incision and drainage is to provide an exit for a collection of pus and necrotic debris. Antibody cellular and chemotherapeutic elements from the blood stream may then gain access to residual areas of infection and provide conditions for wound healing. Drainage by aspiration with a sterile needle and syringe may be employed for diagnosis and for removal of pus in situations where secondary infection or disturbance of function by incision and drainage poses a major threat. Examples are early pericarditis, pleural empyema and suppurative arthritis. The aspirations are repeated whenever exudate reaccumulates. Instillation of a solution of an appropriate

antibiotic is frequently employed in conjunction with removal of pus by aspiration. Proteolytic enzymes and irrigating fluids are used occasionally to promote drainage by thinning of exudate in such areas as sinus tracts the pleural cavity joints and a paralyzed urinary bladder.

The incision is made large enough to provide an adequate opening in the cavity. It is placed in such a manner that gravity drainage is obtained and, insofar as possible it follows the lines of tension of the skin to avoid an unsightly scar or a scar that later may interfere with function of the part. In critical areas, such as the hand or the neck, care must be exercised to avoid damage to blood vessels, nerves and tendons. Culture of exudate is always taken as a precaution against any diagnostic or therapeutic error. Drainage is enhanced and maintained by the use of gauze wicks, rubber plastics or catheters. These drains establish a channel from the surface to the center of the cavity. If proper sterile precautions are not taken during a change of dressings, secondary infections are a frequent complication. Drains are shortened or removed as soon as it is apparent that the discharge has subsided. If drains are allowed to remain too long, a rigid walled tract may develop and persist. In addition to the hazard of secondary infection.

A patent bronchus is a requirement in the resolution of pulmonary disease. Free flow of urine and bile and of bowel contents is also a necessity in urinary biliary and enteric infections. Antibiotic therapy therefore, is not fully effective when calculus obstructs the urinary tract or biliary system or when there is a subarachnoid block or a block of a secreting organ. The obstruction must be corrected before antibiotic therapy is maximally effective.

Débridement—often an accompaniment of incision and drainage—has for its purpose the elimination of harmful factors such as foreign bodies, hematomas, loculations, necrotic sloughs and abscesses in granulation tissues. Precise excision when there is a high level of antibiotics in the blood is regarded as the opportunity to eliminate the pabulum necessary for bacterial growth and, at the same time, allow the antibiotic and host defense mechanisms free access to residual bacteria on the wound.

Extirpation of foci or centers of microbial activity is a specialized form of débridement. It is advantageous to extirpate the local focus whenever the inflammatory process

HOST PARASITE RELATIONSHIPS

Antimicrobial drugs may alter these relationships in the tissue response in the immunologic response and in the bacterial flora indigenous to the body.

Alterations of Tissue Response Whereas chemotherapy is aimed at sterilization of the infective process—a goal rarely achieved—recovery can only be obtained by means of the defense mechanisms of the body. Antibiotic action may injure the infecting agent sufficiently to alter its invasiveness or to reduce its ability to produce harmful substances. When the number of organisms is kept to a minimum by an antimicrobial agent, a lower level of resistance of the host may be effective in checking spread of infection and promoting recovery. There is no convincing evidence however that chemotherapy inactivates the toxic metabolites of bacteria after they have formed and entered the general circulation. Consequently, in order to minimize the effects of the infection, large accumulations of pus must still be drained, antitoxin must be administered for clinical tetanus, hopelessly damaged muscle must be removed promptly in gas gangrene and circulation must be supported in vascular collapse due to gram-negative bacillary endotoxemia. When indicated, general measures must also be instituted to bolster the body defenses.

The tissue response may also be modified under circumstances in which the drug suppresses multiplication of microorganisms but does not eradicate them. With bacteriostatic drug therapy, only temporary response may occur in diseases such as acute osteomyelitis, pyelonephritis, staphylococcal empyema and subacute bacterial endocarditis. In this way an acute infection may be converted to a chronic one.

Alterations of Immunologic Response. As the intensity and duration of therapy are modified by antibiotic therapy the immunologic response of the host may also be modified. This alteration is of no serious consequence in many acute bacterial inflammations, but in others it may be. For example, in typhoid fever chloramphenicol may greatly accelerate recovery, but a relapse may occur in some patients as soon as the drug is withdrawn. At times an alteration of the immunologic response may be desirable. For example, infection with beta hemolytic streptococci is often followed by the development of antistreptolysin antibodies and, occasionally, by the development of rheumatic fever. If the infective process can be

interrupted early and completely by a bactericidal drug such as penicillin the development of antibodies and of rheumatic fever can be prevented, presumably by interference with host reactions.

Alteration of Microbial Flora. Blood borne antibiotics have an effect not only on microorganisms of the infectious process, but also on members of the bacterial flora of the body wherever they reside. Drug-susceptible members of the normal flora are suppressed and an imbalance is created which may in itself lead to disease—superinfection. Patients are especially vulnerable to superinfections who have lowered resistance associated with anatomic defects, cachexia from any cause, blood dyscrasias and hormonal or metabolic disturbances.

In debilitated patients the bacterial flora of the nasopharynx is suppressed, allowing resistant staphylococci, coliform bacilli and yeasts to multiply freely these resistant forms occasionally cause thrush, pneumonia or septicemia. In women taking large oral doses of a broad-spectrum antibiotic, the normal vaginal flora may be suppressed permitting marked overgrowth of yeasts this leads to moniliasis. In postoperative patients, in whom alimentation by mouth is interrupted for a few days, suppression of fecal flora may lead to overgrowth of staphylococci and to staphylococcal enteritis.

Disturbances in normal microbial flora can give rise to secondary disorders other than superinfection. Nutritional deficiencies may occur when the normal intestinal flora is suppressed for a long time. Bleeding may develop because of a deficiency of vitamin K that is normally synthesized in part by the intestinal flora.

HOST DRUG RELATIONSHIPS

The condition of a patient governs the route of administration and choice of antibiotic. Coma, paralytic ileus and vomiting are examples of conditions which may preclude the oral route of administration except by gavage. The inability of certain antibiotics, such as bacitracin, polymyxin, kanamycin, neomycin and streptomycin to be absorbed into the general circulation after oral administration precludes oral use of these drugs except for intra-enteric action. Peripheral circulatory collapse or other cause of interference with absorption of a drug from the upper gastrointestinal tract is also a contraindication for oral antibiotic therapy. Under such circumstances, intramuscular or intravenous therapy is necessary to

DRUG-PARASITE RELATIONSHIPS

It has been noted that, *in vitro* we are dealing with rapidly proliferating microorganisms in continuous contact with a known concentration of drug in a culture medium of fixed composition. In the body environmental factors and concentrations of antibiotics vary widely.

Antibiotic therapy is optimally effective in early acute microbial inflammations. Most bacteria responsible for invasive infections are extracellular in location and are readily accessible to phagocyte cells and antimicrobial drugs. As the lesion progresses the bacteria in the periphery of the lesion continue to proliferate rapidly while those in the center of the focus, finding less suitable conditions for growth, multiply less rapidly and are less susceptible to drug action. Dormant, or slowly metabolizing organisms thus may survive high concentrations of antibiotic and, subsequently may produce a clinical relapse of infection.

Diffusion of Antibiotics. The longer the infection persists the less likely is the possibility of a blood borne drug reaching the infected tissues in therapeutic concentrations. The lesion induced by the microorganism actually may "protect" it from the drug. The walls of an abscess for example, are relatively avascular and may form a partially obstructive barrier to drugs and body defense mechanisms. Fibrin deposits if dense enough, may also impede free diffusion of an antibiotic, whereas frank pus and necrotic tissue may adsorb a drug and thus prevent its distribution to bacteria. Once bacteria become located within tissue cells the cell membrane may protect them from drug activity. Some drugs for example *isoniazid*, reach about the same concentration in monocytes as in the extracellular fluid. With others, such as streptomycin, the intracellular concentration is only a small fraction of the extracellular concentration. Finally the biochemical environment of the established focus of infection is a complex one and may result in significant interference with drug action.

The distribution of drugs varies greatly in different tissues. Some antibiotics delivered by the blood stream reach certain internal organs and others do not. In the absence of large accumulations of pus, parenterally administered antibiotics are effective against acute infections involving soft tissues, lung parenchyma, biliary tree, peritoneal cavity and urinary tract. Diffusion of the drugs from blood serum into these tissues appears

to be facilitated by the reaction of inflammation. Diffusion from blood into acute inflammations in bone the pleural cavity the tracheobronchial tree, the brain and the meninges results in local tissue and exudate levels which are lower than those obtained in blood serum. This handicap is overcome by increasing the total daily dose of antibiotic and by supplemental topical application where feasible.

The tetracyclines and chloramphenicol reach the brain in therapeutic concentrations via the blood stream, but penicillin, streptomycin and erythromycin do not. In the meninges, sulfadiazine and chloramphenicol diffuse readily from the blood, whereas diffusion of penicillin and the tetracyclines is slight unless acute inflammation is present. Parenterally administered sulfadiazine, penicillin and erythromycin can be detected in pancreatic secretions but not the tetracyclines, chloramphenicol, bacitracin or polymyxin. The tetracyclines and erythromycin can be assayed in therapeutic concentrations in the prostate and prostatic secretions whereas only a trace of penicillin, streptomycin and chloramphenicol may be found when optimal blood serum levels are present. The internal compartments of the eye are barred from blood borne antibiotics with the exception of chloramphenicol.

Topical Therapy. Topical therapy in addition to or in place of parenteral therapy may be of value for the localized lesion. The principal indications for topical therapy are (1) accessible *Pseudomonas* infections, as in external otitis (polymyxin) (2) proved monilial infections (nystatin) (3) superficial infections caused by microorganisms sensitive *in vitro* to the antibiotic employed, e.g. infected granulating surfaces and (4) infections in regions of the body difficult to reach by blood borne drugs. Good nursing care of the lesion is an important concomitant of topical therapy.

The requisites for successful topical therapy are absence of necrotic tissue, susceptibility of the organisms to the antibacterial agent, use of an agent that is neither allergenic nor toxic to the tissue cells, constant and adequate contact between the site of the infection and the drug and prevention of recontamination of the wound with resistant organisms by a rigid aseptic technique in wound management. Bacitracin, neomycin, polymyxin, chloramphenicol and the tetracycline antibiotics are preferred for local therapy because they are usually well tolerated when so administered.

HOST PARASITE RELATIONSHIPS

Antimicrobial drugs may alter these relationships in the tissue response in the immunologic response and in the bacterial flora indigenous to the body.

Alterations of Tissue Response. Whereas chemotherapy is aimed at sterilization of the infective process—a goal rarely achieved—recovery can only be obtained by means of the defense mechanisms of the body. Antibiotic action may injure the infecting agent sufficiently to alter its invasiveness or to reduce its ability to produce harmful substances. When the number of organisms is kept to a minimum by an antimicrobial agent, a lower level of resistance of the host may be effective in checking spread of infection and promoting recovery. There is no convincing evidence, however, that chemotherapy inactivates the toxic metabolites of bacteria after they have formed and entered the general circulation. Consequently in order to minimize the effects of the infection, large accumulations of pus must still be drained, antitoxin must be administered for clinical tetanus, hopelessly damaged muscle must be removed promptly in gas gangrene and circulation must be supported in vascular collapse due to gram negative bacillary endotoxemia. When indicated, general measures must also be instituted to bolster the body defenses.

The tissue response may also be modified under circumstances in which the drug suppresses multiplication of microorganisms but does not eradicate them. With bacteriostatic drug therapy only temporary response may occur in diseases such as acute osteomyelitis, pyelonephritis, staphylococcal empyema and subacute bacterial endocarditis. In this way an acute infection may be converted to a chronic one.

Alterations of Immunologic Response. As the intensity and duration of therapy are modified by antibiotic therapy the immunologic response of the host may also be modified. This alteration is of no serious consequence in many acute bacterial inflammations, but in others it may be. For example in typhoid fever chloramphenicol may greatly accelerate recovery but a relapse may occur in some patients as soon as the drug is withdrawn. At times an alteration of the immunologic response may be desirable. For example, infection with beta hemolytic streptococci is often followed by the development of antistreptolysin antibodies and, occasionally by the development of rheumatic fever. If the infective process can be

interrupted early and completely by a bactericidal drug such as penicillin the development of antibodies and of rheumatic fever can be prevented, presumably by interference with host reactions.

Alteration of Microbial Flora. Blood borne antibiotics have an effect not only on microorganisms of the infectious process, but also on members of the bacterial flora of the body wherever they reside. Drug-susceptible members of the normal flora are suppressed and an imbalance is created which may in itself lead to disease—superinfection. Patients are especially vulnerable to superinfections who have lowered resistance associated with anatomic defects, cachexia from any cause, blood dyscrasias and hormonal or metabolic disturbances.

In debilitated patients the bacterial flora of the nasopharynx is suppressed, allowing resistant staphylococci, coliform bacilli and yeasts to multiply freely; these resistant forms occasionally cause thrush, pneumonia or septicemia. In women taking large oral doses of a broad-spectrum antibiotic, the normal vaginal flora may be suppressed permitting marked overgrowth of yeasts; this leads to moniliasis. In postoperative patients in whom alimentation by mouth is interrupted for a few days suppression of fecal flora may lead to overgrowth of staphylococci and to staphylococcal enteritis.

Disturbances in normal microbial flora can give rise to secondary disorders other than superinfection. Nutritional deficiencies may occur when the normal intestinal flora is suppressed for a long time. Bleeding may develop because of a deficiency of vitamin K that is normally synthesized in part by the intestinal flora.

HOST DRUG RELATIONSHIPS

The condition of a patient governs the route of administration and choice of antibiotic. Coma, paralytic ileus and vomiting are examples of conditions which may preclude the oral route of administration except by gavage. The inability of certain antibiotics, such as bacitracin, polymyxin, kanamycin, neomycin and streptomycin to be absorbed into the general circulation after oral administration precludes oral use of these drugs except for intra-enteric action. Peripheral circulatory collapse or other cause of interference with absorption of a drug from the upper gastrointestinal tract is also a contraindication for oral antibiotic therapy. Under such circumstances intramuscular or intravenous therapy is necessary to

insure blood borne delivery of therapeutic concentrations of the drug.

In some patients the administration of antibiotics may be attended by harmful effects. They may be classified into complications caused by sensitization and direct toxic effects. These effects may be manifested with the first dose of the drug and are known as idiosyncrasies. However toxic symptoms usually do not appear until the patient has been receiving drug therapy for several days. Direct drug toxicities are associated with the use of large doses of a particular drug prolonged courses of therapy frequent repetition of short courses or topical applications.

The toxic manifestations of the various antibiotics are listed in Table 1

ANTIBIOTICS AND THEIR IMPORTANCE IN SURGICAL INFECTIONS

Practically all of the antibiotics are available in three dosage forms tablets or capsules for oral administration, vials of the sterile drug to be used in solutions for intramuscular intravenous and local injections, and topical preparations in the form of dry powders, ointments, lotions and solutions. Confusion often occurs because of the various proprietary names that different manufacturers have given to the same antibiotic. Therefore, the physician should be familiar with the generic name of every preparation.

Penicillin. One of the first of the antibiotics, penicillin, has been and still is potentially the most useful of all of the

antimicrobial agents. Group A hemolytic streptococcal infections peritonitis osteomyelitis pneumococcal pneumonia, syphilis gonorrhea, anthrax, gas gangrene and Wolff Israel actinomycosis are indications for penicillin therapy. This antibiotic is highly effective against staphylococcal infections when the organisms are penicillin sensitive. Penicillin is the drug of choice for prophylaxis against impending hemolytic streptococcal and toxigenic clostridial infections.

The dosage form and amount of the drug to be given depend entirely on the sensitivity of the organism and the nature and location of the lesion. Penicillin G 250 mg. given orally every four hours alone or with probenecid (Benemid) administered concurrently to blockade renal secretion of the drug, is adequate for streptococcal infections in circumstances where intramuscular penicillin therapy is undesirable. For intramuscular therapy there is a choice between solutions of aqueous penicillin G to produce especially high, but short lived, blood levels, and repository or depot forms such as procaine penicillin G in aqueous solution to produce lower therapeutic blood serum levels for a period in excess of twelve hours. Intravenous drip of an aqueous penicillin G solution produces a high and more or less constant blood level.

Unfortunately the general application of penicillin for susceptible infections has been curtailed because a significant number of patients have become sensitized to the drug. It was mentioned previously that penicillin

Table 1 Toxic Reactions of Antimicrobial Agents

PENICILLIN G	Allergic Reactions rashes eruptions serum sickness-like reactions exfoliative dermatitis anaphylactic shock contact dermatitis.
STREPTOMYCINS	Rashes contact dermatitis vertigo (streptomycin) deafness (dihydrostreptomycin) drug fever eosinophilia, transient cylindruria.
CHLORAMPHENICOL	Skin eruptions—rare aplastic anemia, others as under tetracyclines.
TETRACYCLINES	Skin eruptions—rare vomiting diarrhea, stomatitis glossitis proctitis, enterocolitis superinfection from overgrowth of resistant organisms (Staphylococci, Monilia, Pseudomonas, Proteus) thrombophlebitis after intravenous administration.
ERYTHROMYCIN AND OLEANDOMYCINS	Nausea, vomiting, diarrhea.
NOVOBIOCIN	Skin eruptions, eosinophilia granulopenia, yellow plasma.
RISTOCETIN	Skin eruptions diarrhea drug fever neutropenia.
VANCOMYCIN	Skin eruptions phlebitis, intravenous route fever
NEOMYCIN AND KANAMYCIN	Topical—harmless, except in peritoneal cavity apnea, respiratory arrest from over dosage in anesthetized patients (neomycin). Oral—loose stools. Injection site—pain. Parenteral—deafness nephrotoxicity
POLYMYXIN	Topical—harmless rashes rare. Injection site, pain, headache hyposthenus fever transient albuminuria, cylindruria.
BACITRACIN	Topical—harmless rashes rare. Injection site, pain, transient nephrotoxicity

also has a diminishing sphere of influence in staphylococcal infections because of the growing prevalence of resistant strains.

A search for drugs effective against staphylococci resistant to penicillin and other antibiotics has led to the discovery of bacitracin, erythromycin, oleandomycin, ristocetin, novoblocin, vancomycin and kanamycin. Other antibiotics will undoubtedly be discovered and added to this list. These antimicrococcal drugs usually show no cross-resistance when used with the major antibiotics. Their action may be bacteriostatic (erythromycin, oleandomycin, novoblocin) or bactericidal (bacitracin, vancomycin, kanamycin). It is preferable to give erythromycin and novoblocin orally, whereas kanamycin, vancomycin and bacitracin must be administered by injection, except when used for intra-enteric infections, such as staphylococcal diarrhea, because of failure of absorption from the bowel. Bacitracin is especially useful for topical therapy of pyogenic infections because of its low allergenic potential.

Streptomycin This drug is bactericidal for gram-negative and gram-positive bacteria, including tubercle bacilli. Both gram-positive and gram-negative organisms commonly have mutants which are resistant to high concentrations of streptomycin. These may emerge after three to five days of streptomycin therapy and supplant previous susceptible flora unless all infecting bacteria have been destroyed during that interval. For this reason, streptomycin is usually employed in combination with another drug to which the organisms are also susceptible.

At present there are four sets of circumstances indicating systemic streptomycin therapy: (1) in tuberculosis therapy being supplemented by another antitubercular drug such as isoniazid or para-aminosalicylic acid, (2) in subacute bacterial endocarditis, adding penicillin, (3) in secondary bacterial peritonitis also using penicillin and (4) in miscellaneous gram-negative bacillary infections. Among the more important of the latter are bacteremia, urinary tract infections and meningitis. Streptomycin is given intramuscularly in doses of 2 to 3 gm. a day for acute nontuberculous infections and it is administered in divided doses at six-hour intervals. The dosage in tuberculosis is 1 gm. given three times a week. The problem posed by the development of streptomycin-resistant strains in the treatment of tuberculosis has led to a continuing search for antitubercular agents.

Neomycin. The antimicrobial spectrum is essentially the same as of streptomycin but with only selective cross-resistance. However, nephrotoxicity and ototoxicity have confined its uses primarily to oral and topical administration. It is sometimes used intramuscularly in divided doses of 0.75 gm a day in life-endangering gram-negative sepsis which is completely resistant to other agents, particularly that due to *Proteus* and *Aerobacter*. Principal indications by mouth are for preoperative suppression of the intestinal flora for treatment of staphylococcal enteritis for treatment of impending hepatic coma in which intestinal ammonia-producing organisms contribute to toxemia, for aerosol therapy of chronic bronchial suppuration, and for local therapy of infected granulating wounds.

Kanamycin has essentially the same spectrum of activity and clinical applications as neomycin.

The Tetracyclines. Tetracycline and its two analogues chlortetracycline (Aureomycin) and oxytetracycline (Terramycin) are almost identical (1) in their wide range of antimicrobial activity extending to the rickettsial species and including gram-positive and gram-negative bacteria, (2) in the fact that the three agents give almost complete cross-resistance (if a microbial strain is resistant to one of the three drugs, it is usually resistant to all) and (3) in the pattern of side effects incident to therapeutic use. In hospitals, staphylococci and species of *Proteus* and *Pseudomonas* are frequently resistant and resistant coliform bacteria are encountered with increasing frequency from widespread use of these antibiotics. The principal untoward effects of tetracycline therapy are gastrointestinal upsets and replacement of indigenous bacterial flora with drug-resistant species with impending superinfection. Therefore, caution is necessary when a tetracycline drug is prescribed prophylactically and for minor pyogenic infections or chronic suppurations of the respiratory tract. The principal indications for tetracycline in surgical practice are (1) infections of the liver and biliary system, (2) peritonitis, diverticulitis and amebic colitis, (3) pyogenic infections in patients who are allergic to penicillin, (4) gram-negative bacillary infections of the urinary tract, and (5) other infections in which the causative organisms are sensitive to tetracycline but are not to other antibiotics. The usual adult dosage of tetracycline administered orally or intravenously is 1.0 to 1.5 gm. a day in two

or three equal, divided doses. The addition of certain excipients (citric acid, glucosamine) to capsules containing tetracycline enhances absorption of drug from the gastrointestinal tract into the blood stream. Following absorption, high concentrations of the drug can be recovered in bile, stools, urine and peritoneal fluid. Wherever superinfection with monilia is a serious threat tetracycline may be given concurrently with the antimonilial drug, Mycostatin. This antifungal agent is also used alone in doses of 500,000 units four times a day for monilial infections of mouth, bowel or vagina.

Chloramphenicol. This antibiotic, produced by chemical synthesis, has about the same spectrum of effectiveness as the tetracyclines, but it produces only a slight cross resistance with them. Thus tetracycline resistant organisms often remain chloramphenicol sensitive. The toxicity of this drug for the hematopoietic system has been greatly exaggerated. In the rare instances when toxicity occurs, it follows protracted usage and heavy doses, often with repeated courses for such conditions as chronic bronchiectasis or chronic urinary tract infection. In children, however it may follow treatment for five or more days of such acute infections as nonspecific upper respiratory tract infections, whooping cough and gastroenteritis. The principal indications for chloramphenicol therapy are (1) staphylococcal, gram negative bacillary or mixed infections resistant to other antibiotics but sensitive to chloramphenicol, (2) typhoid fever, (3) pyogenic infections of the central nervous system, and (4) pyogenic infections of the eye. The usual dosage regimen for adults is an initial "loading" dose of 1 gm. given orally and followed by 0.5 gm. at four to six or eight hour intervals, depending on the severity of the infection.

Polymyxin B. Polymyxin B, a basic polypeptide, is rapidly bactericidal for many species of gram negative bacteria and it is the most uniformly effective of all the known antibiotics in treating infections due to *Pseudomonas*. It exerts no action on *Proteus* or the gram positive bacteria. Polymyxin produces unpleasant, but usually harmless, neurotoxic effects in some patients (paresthesias, dizziness) and is somewhat nephrotoxic, particularly in individuals having impaired renal function. Therefore, the daily dose must not exceed 2.0 mg. per kg. per day in divided doses, injected intramuscularly for not more than ten days. Pain and swell-

ing at the site of injection are minimized by dissolving the drug in a solution of a local anesthetic agent. The principal indications for systemic polymyxin therapy are gram negative bacteremia and severe urinary tract infections, especially those due to *Pseudomonas* and for mixed infections, when used with tetracycline or chloramphenicol. The large molecular size of the drug prevents good diffusion and penetration in cerebrospinal and pleural fluids and in relatively avascular tissues. *Pseudomonas meningitis* has been treated by direct intrathecal injections of small doses of polymyxin with no ill effects. Topical application of polymyxin alone, or in combination with neomycin or bacitracin is useful for surface infection because of low allergenic potential.

PREVENTION AND CONTROL OF NEW INFECTIONS ACQUIRED IN HOSPITALS

Control of infection in the hospital population is a continuing problem. To keep the incidence of acquired infections to an absolute minimum is of paramount concern to all. Staphylococci and the various gram negative bacilli are most commonly the causal organisms and they are frequently multi-antibiotic resistant. These bacteria are especially important risks for those with reduced general or local tissue resistance. The primary reservoirs of these bacteria are acute and chronic suppurations and asymptomatic carriers. Secondary reservoirs include all hospital equipment as well as clothing. A number of surveys have revealed that infection rates in hospitals using antibiotics routinely as the first line of defense against infection are higher than in those not doing so. It has been shown also that rigid application by all concerned of clean and aseptic techniques in operating rooms and wards and in the treatment of established infections is effective in reducing the incidence of acquired infections among patients, staff members and personnel. The fundamental approaches, therefore, are two: (1) reduction of the incidence of resistant bacteria and (2) reduction and prevention of cross-infections by these bacteria.

Reduction in the Prevalence of Drug Resistant Bacteria. As stated previously a fundamental principle of antibiotic therapy is restricting their use to conditions in which they have been proved effective. Administration of antibiotics for prophylaxis now outnumbers prescriptions for therapy of in-

fection. The accepted indications for prophylactic antibiotic therapy are much more limited than generally practiced.

The limiting factors in antibiotic prophylaxis are these (1) the drug must be chosen in the absence of bacteriologic data (2) no antibiotic is universally effective against all potential invaders (3) the wound environment—tissue necrosis, hematomas foreign bodies—may inhibit the effectiveness and (4) the nature of the lesion may prevent contact between blood borne drug and bacteria.

The principal indications for prophylactic antibiotic therapy are prevention of hemolytic streptococcal infection and control of bacterial invasion in contaminated mechanical injuries and in operations on structures of the body which naturally harbor a large population of bacteria.

Antibiotic prophylaxis against pyogenic infection is probably of value in elective surgery in (1) preventing further attacks in patients with rheumatic fever (2) preventing subacute bacterial endocarditis in patients with valvular heart disease or congenital heart disease undergoing surgery in a contaminated field (3) infants with congenital anomalies, (4) presence of chronic obstructive emphysema and (5) patients receiving prolonged high dosage steroid therapy.

Antibiotic prophylaxis is also probably warranted in delayed or nonoperative treatment of acute pancreatitis, bleeding esophageal varices, acute diverticulitis and perforated peptic ulcer or gastrointestinal carcinoma.

Traumatic Wounds. Wounds of soft tissues do not become infected, or infection is trivial and wound healing uncomplicated, when such injuries are subjected to early precise definitive surgery. It follows that antibiotic therapy is not justifiable for every bruised or lacerated wound. It is justifiable when operative management is inadequate or delayed beyond two to four hours, when the danger of invasive infection already exists and when casualties have sustained penetrating wounds of the joints or the major body cavities which extend into structures harboring resident bacterial flora.

Penicillin is probably the safest and most tested antibiotic today, although cases of hypersensitivity may preclude its use and call for the substitution of another antibiotic. Therapy is instituted at the time of definitive surgery and it is continued postoperatively for three to five days. Immunization against tetanus is also effected.

Thermal Injuries. The burn wound requires special consideration. The magnitude of the injury often involves extensive areas of the body surface. Surgical excision and closure by skin graft are sometimes difficult, if not impossible, because of frequent inability to diagnose accurately the depth of the burn on first inspection and because secondary infection threatens until complete healing is achieved. The rationale for antimicrobial therapy in the burned patient is demonstrated by the rare instances of death from hemolytic streptococcal septicemia since its institution.

As in other traumatic wounds, chemoprophylaxis is but one factor in the total problem of prevention of wound suppuration in the burn eschar.

Antimicrobial therapy therefore, seems justified only when (1) the total area of partial and full thickness burn involves 15 to 20 per cent or more of the body surface (2) an associated complex injury increases the hazard of infection, (3) the patient has concurrent respiratory tract, genitourinary or other pyogenic infection remote from the burn, (4) the patient is exposed to radiation or to steroid therapy and (5) extensive débridement and skin grafting are carried out.

Surgical Procedures in Contaminated Fields. In elective surgery the healing process is not disturbed to as great a degree as in the traumatic or thermal wound. The danger of bacterial dissemination is also lessened by careful preoperative management. In contaminated fields the principles of prophylactic antibiotic therapy are emphasis on preoperative reduction of the regional bacterial flora by mechanical cleansing and adjunctive antibiotic therapy the choice of antibiotic tailored to the bacterial flora of the region, and prevention of seeding contiguous tissues by proper quarantine. Postoperatively antibiotic therapy is seldom necessary unless spillage has occurred. The following are examples of contaminated cases in which antibiotic prophylaxis may be warranted.

Head and neck. Plastic operations involving the oral cavity radical surgery for fungating carcinoma of the oral cavity and neck, esophageal diverticulum.

Thoracic. Presence of infection in the lesion cannot be ruled out preoperatively—surgery extends across potentially infected lung, mediastinal structures or esophagus bronchopleural or tracheoesophageal fistula, cardiac or peripheral vascular surgery.

Abdominal. Transduodenal choledocholithotomy plastic repair of bile ducts porta caval shunt for impending hepatic coma, pancreatic or enteric fistula regional enteritis with fistula, colon resection for inflammatory disease, strangulation, obstruction, perforation, carcinoma and after spillage during anastomosis closure of colostomy anoplasty with primary closure of wound evisceration of laparotomy wound

Obstetric and gynecologic. Premature rupture of membranes prolonged labor rectovaginal fistula transplantation of ureters to bowel pelvic organ exenteration

Genitourinary. Presence of severe urinary infection valvular heart disease, extreme debilitation contamination of dead space, postoperative extravasation of urine ureteroplasty transplantation of ureters.

Surgical Procedures in "Clean" Operative Fields. A number of recent studies have shown that in patients given antibiotics there was an increased rather than decreased rate of "clean wound sepsis." Following abolishment of routine prophylaxis and institution of a rigid program to prevent cross contamination, the infection rate returned to the low level of the preantibiotic era. Obviously chemoprophylaxis should be abandoned if there is no evidence that it is effective and if there are potentialities for harm.

Postoperatively antibiotic therapy is not warranted for unexplained fever heart failure, cerebrovascular accident, atelectasis thrombophlebitis or tracheostomy.

Reduction of Spread and Persistence of Drug Resistant Bacteria. The ultimate success in preventing wound infection will depend upon concerted efforts to decrease exposure within the hospital environment. Infection arises within the operating room or on the wards.

Even under the most favorable conditions all wounds are contaminated at operation and whether or not infection supervenes depends as much on precise deft surgical technique as on any other factor. Infection will seldom arise in wounds which are handled gently and closed without foreign bodies or excess necrotic tissue. Measures, other than surgical techniques which require constant checking, are those which apply to inoculations of the wound by bacteria from the air and by contact. The checks to reduce these hazards include (1) periodic check on sterilizing equipment to insure sterile instruments, gloves, solutions and textiles, (2) careful preparation of the hands and forearms of the operating team as well as of

operative fields (3) prompt change of a punctured glove or wet gown (4) adequate masking with a change of mask after each case (5) avoidance of unnecessary conversation during surgery (6) removal from duty of all personnel who harbor any open infection (7) reduction of traffic within the operating room to a minimum and (8) prohibition of street clothing within the operating pavilion, with similar regulations against the wearing of operating room attire for ward rounds.

Since hospital personnel are carriers of staphylococci, they should protect wounds from contact infection by the wearing of masks and the employment of an all instrument or sterile glove technique or both, for changes of dressings in septic cases, with washing of hands before and after each dressing. In addition, for the patient with a suppurating lesion, segregation of the wound and not necessarily the patient should be the prevailing practice. One of the main causes of wound infection is carry-over of infected discharges from one patient to another by members of the medical or nursing staff.

Physicians and attendants must be trained to realize that the bare hands must be considered dirty at all times. Once dirty bandages and dressings have been touched, the hands must be washed before the wound is treated. To avoid contamination the hands of the dresser should never touch the wound or surrounding skin area which has also been covered by dressings. The inner dressings are removed with sterile hemostats. These and other necessary sterile articles are provided for the dresser on a sterile towel. The latter then serves as the work area. The dresser never touches the reapplied dressings with his hands. Between dressings the hands are again scrubbed or washed with hexachlorophene.

In order to prevent dissemination of infected discharges, the used dressings are discarded in several thicknesses of old news paper or in waxed paper bags and promptly incinerated. Contaminated instruments are then dropped by the dresser into a vessel containing germicidal solution. Dressings are changed only as necessary as frequent changes enhance the opportunity for contamination. It is well to remember the World War II adage that inspection means infection.

Dressings should be bulky and occlusive enough to avoid soaking through, when this happens it should be considered an emer-

gencies cause for redressing. Patients who can do so should be encouraged to take total baths with a germicidal soap such as hexa chlorophene as soon as wound healing is not jeopardized. When a wound is drained surgically it should be covered with an occlusive dressing to prevent secondary contamination through soaked or soiled dressings. Since bacteria from the external environment readily penetrate moist gauze especially if soaked with serum, open wounds receiving wet therapy should have water proof (external) dressings.

For those who cannot be prevented from contaminating their surroundings heavily (extensively burned infected patients those with generalized skin infections tracheostomies) a reasonable regimen of isolation should be instituted. The room or cubicle contents should be treated as heavily contaminated. Needless to say quarantine is effective and can succeed only when it is complete.

Once infection in a wound is established there is a tendency to rely on antibiotic therapy—which is usually ineffective—rather than prompt surgical intervention. There is a reluctance also among surgeons to make incisions of adequate extent to ensure proper drainage. This tendency to use small incisions which afford inadequate drainage allows puddling of pus and bacteria and causes undermining and imprisonment of the necrotic tissue upon which bacteria thrive. Invasion progresses in the depths of the tissues, masked by overlying intact skin that in itself affords no tensile strength to the wound. If the infection is allowed to progress unrecognized and undrained until bacterial invasion destroys fascia, disruption and/or evisceration are imminent. To depend on antibiotics to abort an established invasive wound infection and to treat it by conservative drainage through a small incision are conducive to dehiscence rather than insurance against it. Advent of the antibiotics has not obviated the necessity for applying the principle of surgical drainage once an infection has become established. Such conservative treatment frequently leads to prolonged hospitalization, many painful dressings, protein depletion and the subsequent development of hernias. The treatment for the established wound sepsis is early adequate, dependent drainage before invasion of tissue and fascia has occurred.

When drainage is secured, the wound also must be rid of necrotic tissue. The protective action of appropriate blood borne

antibiotics allows the surgical débridement of all involved tissue without fear of aggravating the process or causing extension of the invasion. Usually twenty four to forty eight hours of systemic antibiotic therapy will control the invasive component (cellulitis) and allow adequate débridement at the time of drainage. The surgeon is now confronted with an open clean wound, the treatment of which is surgical closure, either by suturing or skin grafting. Such wounds will heal by second intention if kept clean, but the process is prolonged and results in excessive scar formation.

READING REFERENCES

- Amwar A. A., and Turner T. B., Antibiotics in Experimental Tetanus. In Vitro and In Vivo Studies. Bull. Johns Hopkins Hosp. 99:65-101, 1956.
- Dubos, R. J., Biochemical Determinants of Microbial Diseases. Cambridge, Mass., Harvard University Press, 1954.
- Editorial: Antibiotics, Staphylococci and Enterococci. New England J. Med. 253:201-203, 1955.
- Editorial: Cortisone ACTH and Infection. New England J. Med. 254:41-42, 1956.
- Finland, M., Changing Patterns of Resistance of Certain Common Pathogenic Bacteria to Antimicrobial Agents. New England J. Med. 252:570-580, 1955.
- Garland, J., ed., Current Concepts in Therapy. New England J. Med. publ. 1955.
- Hoffman, E., and Rebeck, J. F. Subcutaneous Space Contamination. Ann. Surgeon, 24:364-366, 1958.
- Howe, C. W.: The Problem of Postoperative Wound Infections Caused by Staphylococcus Aureus. Ann. Surg. 146:384-398, 1957.
- Jawetz, E., Metnick, J. L., and Adelberg, E. A., Review of Medical Microbiology. Los Altos, Calif., Lange Medical Publications, 1958.
- Levenson, S. M., Pulaski, E. J., and Upjohn, H. L., Metabolic Changes Associated with Injury. In Zimmerman, L. M., and Levine R., eds. Physiologic Principles of Surgery. Philadelphia, W. B. Saunders Co., 1957. Chap. 1 pp 1-35.
- Martin, G. J., ed., Proteolytic Enzymes and Their Clinical Application. Ann. New York Acad. Sc. 68. Art. 1 pp. 1-244, 1957.
- McDermott, W.: Microbial Persistence. Yale J. Biol. & Med. 50:257-291, 1958.
- Meleney F. L., Clinical Aspects and Treatment of Surgical Infections. Philadelphia, W. B. Saunders Co., 1949.
- Nungester W. J. Mechanisms of Man's Resistance to Infectious Diseases. Bact. Rev. 14:105-129, 1951.
- Oakley C. L., Gas Gangrene. Brit. M. Bull. 10:52-58, 1954.
- Peterson, C. G., and Krippachne, W. W. Shock in Sepsis. Am. J. Surg. 96:158-171, 1958.
- Pittenger C. B., and Long, J. P., Neurovascular Blocking Action of Neomycin Sulfate. Antibiotics and Chemotherapy 8:196-203, 1958.
- Pulaski, E. J.: Surgical Infections, Prophylaxis, Treatment, Antibiotic Therapy. Springfield, Ill., Charles C. Thomas, 1954.
- Pulaski, E. J. Discriminate Antibiotic Prophylaxis in Elective Surgery. Surg. Gynec. & Obst. 109:385-388, 1959.

- Pulaski, E. J., and Bowers, W. F.: Antibiotic Prophylaxis. Possibilities Limitations and Hazards. *S Clin. North America*, pp. 1459-1471 1957
- Reedy, R. J., Oswald, E. J., and Wright, W. W.: The Effect of Gamma Globulin and Specific Antiserum Combined with Antibiotics in Experimental Infections in Mice. In *Antibiotics Annual, 1957-1958*. New York, Medical Encyclopedia, Inc., 1958 pp. 581-584
- Rogers, D. E., ed. *Staphylococcal Infections*. Ann. New York Acad. Sc. 65 Art 3, pp. 55-246 1956
- Sandusky, W. R., Pulaski, E. J., Johnson, B. A., Meloney, F. L.: The Anaerobic Nonhemolytic Streptococci in Surgical Infections on a General Surgical Service. *Surg. Gynec. & Obst.* 75 145-156, 1942.
- Schweinsburg, F. G., Shapiro, P. B., Frank, E. D., and Fine, J.: Host Resistance in Hemorrhagic Shock IX. Demonstration of Circulating Lethal Toxin in Hemorrhagic Shock. *Proc. Soc. Exper. Biol. & Med.* 85-646-650 1957
- Sherlock, S.: Pathogenesis and Management of Hepatic Coma. *Am. J. Med.* 24 805-813, 1958
- Symposium on Tetanus. *Proc. Staff Meet. Mayo Clin.* 32:141-167 1957
- Weinstein, L., Goldfield, M., and Chung, T.: Infections Occurring During Chemotherapy: A Study of Their Frequency Type and Predisposing Factors. *New England J. Med.* 251 247-255, 1954.
- Welch, H., Lewis, C. N., Weinstein, H. L., and Boeckman, B. B.: Severe Reactions to Antibiotics, a Nationwide Survey. In *Antibiotics Annual, 1957-1958*. New York, Medical Encyclopedia, Inc., 1958, pp. 296-309
- Williams, R. E. O.: The Progress of Ideas on Hospital Infection. *Bull. Hygiene* 31-965-979 1956.
- Wilson, G. S., and Miles, A. A.: *Topley and Wilson's Principles of Bacteriology and Immunity* Ed. 4. Baltimore: Williams and Wilkins Co., 1933 2 vols.
- Wood, W. B. Jr.: Studies on Cellular Immunology of Acute Bacterial Infections. *Harvey Lect.* (1951-1952) 47 72-98 1953.

BASIC PRINCIPLES OF TECHNIQUE IN SURGICAL CARE

By FRANKLIN LOUNSBURY M.D.

FRANKLIN LOUNSBURY is the son of a surgeon and received his education at the University of Wisconsin and Northwestern University. Dr. Lounsbury is an Assistant Professor of Surgery at Northwestern University Medical School. He is particularly suited to describe procedures about which residents and interns are often in doubt. One may be well versed in the principles of the care of catastrophic surgical situations and yet be quite ignorant of the meticulous methods which, when applied promptly and judiciously, prevent the crippling complications which too often follow an apparently simple surgical lesion.

EMERGENCY ROOM PROCEDURES

Every member of the house staff in the average general hospital is certain at some time during his career to face a desperate situation in the emergency room. The life of a critically ill or injured patient may depend upon his accurate and quick appraisal of the patient's problems and rapid institution of therapy.

Examination of the Patient. An emergency examination of necessity must be rapid and not so detailed as the more leisurely routine through which the doctor takes an office patient or a hospitalized but not acutely ill patient. However it must be searching in nature and should disclose within a matter of moments the essential pathology. The presence of a bystander who witnessed the patient's accident, or of a relative who is familiar with the patient's medical history should help the examiner in his

search for pertinent historical data, but this information must not be obtained at the expense of the examination of the patient.

When the examiner is feeling the pulse he can observe the character of respiration. If the patient is in coma, and particularly if he is having labored respiration, the examiner must satisfy himself that there is no obstruction of the airway. The tongue should be pulled forward, and if this does not relieve respiration it may be that the patient has already aspirated mucus. The chest should be auscultated for breath sounds to see whether the air is actually getting through into the lungs. If there is any doubt, the trachea should be aspirated with a small-caliber catheter attached to a suction apparatus. In the conscious patient, continuing difficulty in respiration with cyanosis should suggest the possibility of tension pneumothorax, cardiac failure, a suck-

ing wound of the chest or some other acute process changing the intrathoracic physiology. If a determination of the pulse has established that the patient is not in cardiac arrest or in severe cardiovascular collapse, the remainder of the examination can be carried out and the information about the cardiac status mentally fitted into the rest of the picture assembled by the continuing examination.

After the pulse and respiration have been checked the head is examined. It is possible to palpate the cranium quickly and thoroughly in a matter of moments in a search for lacerations, contusions and swelling. One should look particularly for fluid issuing from the nose or ears. The size and regularity of the pupils should be noted, as well as any facial asymmetry appearing on simple examination. The cervical spinous processes posteriorly and the trachea and thyroid cartilages anteriorly can be gently but easily palpated for evidence of asymmetry. In almost the same gesture the neck can be palpated for evidence of crepitation.

The chest should then be examined carefully for evidence of wounds and a series of respiratory excursions should be watched. Breath sounds should be listened for on each side of the chest in representative areas and the heart tones should be briefly noted in the significant areas at apex and base. The examining finger can be run lightly but quickly over the rib cage on each side for evidence of rib fracture or subcutaneous emphysema. As the examination is carried posteriorly the scapulae should be felt on each side and then, anteriorly the clavicles. As the examiner's hands are passed laterally along the clavicle a gross survey of the structures around the shoulder joints can be easily made. At this point both upper extremities should be quickly and deftly palpated for gross evidence of a fracture by moving the humerus about its articulation with the clavicle and scapula, and subsequently moving the forearm on the arm at the elbow in extension and flexion as well as in supination and pronation.

The patient's hands should be moved in all possible directions at the wrist. Most fractures involving the bones of the hand are apparent in the slight deformity or asymmetry produced in the area of the fracture. Thus a fracture of the fifth metacarpal, unless it is impacted in good position will produce a shortening of the fifth finger as the fingers are bent into the palm and will obscure the normal protrusion at the end of

the fifth metacarpal. If the patient is conscious he should be asked to make some of the movements of the hand which would indicate involvement of the median, ulnar or radial nerves. These movements are opposition of the thumb or tip of the thumb to tip of the little finger as a check for median nerve function; extension of the hand at the wrist, or the thumb at the metacarpophalangeal joint, as a test of radial nerve function, and the ability to make a cone with the fingers and thumb as indication of ulnar nerve function.

If the patient is unconscious weakness or paralysis of the upper extremity can be determined by raising the patient's arm passively above his face and allowing it to fall. If normal muscle tone is present the extremity will not strike the examiner's hand which protects the patient's face, but will fall to the side of it.

Examination of the abdomen is important in the conscious as well as in the unconscious patient. First, simple visual examination should be employed to determine whether the abdomen is scaphoid or distended and whether its movements are normal for a patient who is breathing normally with an uninvolved diaphragm. Next, the abdomen should be palpated for muscle guarding, tenderness and masses. The conscious patient can give considerable help by indicating whether or not there is tenderness in the abdomen and if so exactly where, as well as whether the tenderness is rebound in character. The palpation of the abdomen can be combined with gentle compression of the thoracic cage from side to side as a gross method of determining the possibility of rib fracture. If the abdomen is distended and the patient is unconscious, the abdominal examination should be extended to include careful auscultation of bowel sounds. If the patient is conscious and does not complain in particular of the abdomen, this phase of the examination may be postponed pending completion of the cursory examination of the lower extremities.

The pubic symphysis should be gently palpated and pressed against, just as the iliac crests should be subjected to gentle compression, in an effort to determine grossly whether or not there is a pelvic fracture. Both inguinal areas should be inspected and palpated for the possibility of a strangulated inguinal hernia. It is important to examine the perineum and buttocks in both male and female patients for any evidence of contusions and particularly for any evidence of

wounds which might involve the rectum at a higher level. The scrotum should be quickly palpated and the external urethral meatus of the male examined for gross evidence of bleeding. In female patients the labia should be gently separated and the urethral orifice and introitus briefly inspected.

At this point, attention can be directed to the lower extremities as the final part of the emergency examination. The contour and symmetry of these extremities should be examined and then the tissue should be palpated gently but definitely in search for evidence of fracture of any of the long bones. In the conscious patient, voluntary effort should be requested to see that there is no limitation of motion of any of the major muscle groups or of any joints. In the unconscious patient, the thigh should be flexed passively on the abdomen with the heel still on the examining table and then released to determine whether the extremity will slide down in an externally rotated position indicative of paralysis.

Care of Simple Wounds. In the average hospital, simple wounds such as lacerations, contusions and even minor avulsions are treated in the emergency room. A simple rule of thumb for determining whether an injury is serious enough to require formal treatment in the operating room is that the latter is reserved for injuries in which deeper structures such as tendons, nerves, bones and blood vessels are involved. Obviously head wounds of any severity, chest wounds, abdominal wounds, compound fractures and even simple lacerations of any great length or depth will require formal débridement and repair in a better-equipped operating room as opposed to the more simply outfitted emergency room.

All wounds should be gently cleansed with soap and water. This is best done by covering the actual wound with a simple, sterile gauze dressing while the skin about it is initially and briefly cleansed with soap and water using frequent irrigations of soapy water or normal saline solutions from a separate container. When the skin adjacent to the wound has thus been cleansed, the wound itself should be similarly washed with soap and water and irrigated with saline solution. Irrigation is particularly important in wounds which have some depth or in which there has been considerable contusion of the soft tissue with indriven dirt and similar debris. It is usually accepted that a ten-minute cleansing with soap and

water coupled with saline irrigation provides the optimum preparation of the wound. At this point the devitalized tissue should be excised and the various layers involved should be gently débrided, trimming away only the jagged and irregular edges and those pieces of skin, fat or muscle which are either obviously dead or so badly contused that their recovery is questionable.

Insofar as possible all foreign material should be removed. This is particularly true of clothing, hair and street dirt. However, considerable judgment must be exercised because sometimes a search for an innocuous foreign body, such as a small piece of metal which was hot at the time of its entry, may be more devastating to the tissue than simply leaving the foreign body in place. In some abrading injuries there is a great deal of tattooing of the skin in its more superficial layers. Endless time can be spent in attempting to pick out these small pieces of gravel or dirt, whereas most of them will slough if they are simply left alone and allowed to work out as the cells in which they are imbedded are pushed out toward the epidermis.

In wounds which have been adequately cleansed and débrided and which are seen within six to eight hours of their infliction, it is perfectly safe to close the skin edges primarily. In wounds which are seen after this "golden period" has passed, it is probably safest to leave the wound edges open. If upon inspection a week to ten days later the wound is clinically clean, it may be possible to close it secondarily as was done with many thousands of wounds in World War II and the Korean conflict. The closure need not involve actual suturing because sometimes sterile tape bridges or collodion-impregnated fine mesh gauze bridges can be used to approximate these skin edges almost as effectively as actual suture material.

Care of Animal Bites. A wound produced by animal bite presents particular problems not usually present in wounds inflicted by inanimate objects. Dog bites are by far the commonest of animal bites and they always carry with them the risk of rabies. All such animal bites must be reported to the proper authorities, usually the local police station, but in some communities it is required that the bite be reported to the Public Health Department directly.

The wound is treated like any other wound and when adequately débrided and cleansed it may be closed primarily if it is seen sufficiently early. Special problems con-

cerning dog bites revolve about the question of whether or not the dog is rabid. If the dog can be positively identified and kept under observation, the patient need not be given any prophylaxis against rabies. If the dog was in a pre-rabid phase of the disease at the time the wound was inflicted, the animal can be expected to develop clinical signs of rabies within ten to fifteen days. At this time positive immunization of the patient with rabies vaccine can still be successfully undertaken. If the dog does not develop rabies during the period of observation, then the matter can be dropped.

Technically human bites are in many ways much more serious than the average animal bite because the symbiotic action of Vincent's organisms found in the mouth of the human being produces an extremely virulent infection. For this reason, human bites must be given especially meticulous care and in most instances should not be closed primarily even if seen early.

Bites by other animals are quite infrequent but in many instances carry the same danger of rabies as do dog bites. Squirrels, skunks, cats and other small animals have been known to transmit rabies to human beings. In the instance of a bite from such a small animal, if the animal can be kept under observation the same procedure is followed as is done with the dog. In most instances, however it is impossible to make a positive identification of the animal, much less keep it under observation. Therefore to be on the safe side individuals bitten by such animals should be given the Pasteur antirabies treatment.

Protection against Tetanus. Many wounds carry with them the potential danger of tetanus. This is particularly true of wounds inflicted by rusty implements or nails and of wounds contaminated with street dirt or farm dirt. The spores of tetanus are known to be widely distributed in nature and to occur as contaminants of various animal furs and hairs. Since tetanus organisms are anaerobic, they will multiply best and elaborate their toxin in the depths of a closed wound. It is possible that they thrive even better in an infected wound where other pathogens are present to use the oxygen, thus producing a more truly anaerobic environment. Thus, one form of prophylaxis against tetanus consists of removing as far as possible contamination in the depths of the wound and in every way possible preventing any wound infection however this alone is not sufficient because tetanus car-

ries such a high mortality when it does occur. Therefore two means of giving the wounded individual immunity are in use. The first is to confer passive immunity by means of tetanus antitoxin. The second is that of developing the patient's own active immunity to tetanus by means of tetanus toxoid.

An effective passive immunization which will last for at least one week can be conferred on an individual by giving *tetanus antitoxin* which is derived from the serum of horses. The standard dose has been 1500 units but many physicians now are giving a minimum of 3000 units and some give 5000 units. This confers immunization immediately but immunization is not effective longer than one week. If the wound for which this was given continues to be infected the dosage of tetanus antitoxin must be repeated. Because there is an appreciable incidence of sensitivity to horse serum, tetanus antitoxin should not be given to a patient without first testing the individual for sensitivity. This may be done by means of intradermal tests or as some authorities feel, more effectively by means of conjunctival tests in which a drop or so of very weak solution of antitoxin is used. If there is a sensitivity and the situation requires definitely that antitoxin be given, it can be given in very small doses with some intravenous antihistamines and/or 5 mm of 1:1000 Adrenalin chloride. In some instances it may be necessary to use a rapid desensitization procedure.

Tetanus toxoid is an alum precipitated toxin which has thus been rendered non-toxic but which retains its antigenic properties. It is given in a series of three doses, usually at biweekly intervals to develop the individual's own immunity to tetanus. This procedure has many advantages over immunization with tetanus antitoxin, not the least of which is the fact that there is no real sensitivity to the tetanus toxoid. Occasionally a patient may develop a local reaction at the site of injection but this is transient and is never accompanied by systemic signs. Another advantage of tetanus toxoid is that the titer of immunity so produced can be raised to an effective level with a so-called booster dose of 0.5 cc. of toxoid at long intervals after the original immunization series has been given. Authorities now feel that there is still a responsive level almost ten years after the original series of immunizations. It might be pointed out that tetanus toxoid cannot be invoked

as a means of protection against a particular wound. If the patient has not been previously immunized with tetanus toxoid the level of antibody rises so slowly that it will not be effective in the particular instance where the possibility of tetanus is feared. Immunization with tetanus toxoid is definitely an interval procedure which is done in anticipation of the patient's receiving wounds in the future which might carry the risk of tetanus. Practically all youngsters today who are under the care of a family pediatrician are routinely immunized against tetanus. All persons entering the Armed Forces of the United States similarly receive immunization with tetanus toxoid so it is a relatively easy matter to determine whether or not an individual has had a previous immunization. If he has then a booster dose of tetanus toxoid is all that is needed.

Care of Severe Injuries. When the house officer is confronted with a severe injury which requires care in the operating room, he must have a definite logical approach to the problem. This should have been thought out and planned in advance to the extent that it is almost second nature with him. The basic tenets of this approach are: keep the patient alive, find out what is the matter with him, get whatever help is needed, restore normal physiology as soon as possible. The tenets are listed in the order of their priority although they are obviously not mutually exclusive.

Although usually it is fairly obvious what must be done to keep the patient alive, occasionally as in the instance of severe spontaneous pneumothorax or a recently perforating ulcer the young surgeon may have to do a little searching to find exactly what is wrong with the patient before he can attempt to save his life. It may seem to some that the statement of four such tenets is unnecessarily fundamental. However there are few surgeons who have not had the experience of seeing a befuddled intern, stunned by newspaper reporters and photographers, trying to placate the weeping relatives while attempting in his own mind to decide whether to summon an attending man, call the operating room, take the patient to the x-ray room or try to obtain a history. Such an intern, remembering these tenets should immediately clear the emergency room of all relatives, bystanders, photographers and police while he makes a rapid assessment of the patient's vital signs and determines the degree of jeopardy. He should then institute symptomatic therapy

for what seems to him most threatening—oxygen for obvious respiratory distress in travenous fluids for signs of shock—while having a sample of blood taken for immediate typing and crossmatching. He may then summon an appropriate attending man and send a nurse or emergency room aide out to ask the relatives or bystanders a few simple questions to fill in the clinical picture already developing from his findings gleaned while he conducted the remainder of his emergency examination. Fulfillment of the final tenet usually involves definite treatment, surgically or medically as the case may be—slow decompression of the affected lung in the spontaneous pneumothorax, closure of the hole in the duodenum in the instance of a perforating ulcer, alignment and immobilization of the fragments of a fractured bone or ligation of a severed artery in the instance of severe hemorrhage from a laceration. The intern or resident, however inexperienced he may be in handling emergencies, who knows what needs to be done and has a systematic way of achieving his objectives may be able to do as much for the severely injured or acutely ill patient as the most skilled and experienced physician. This matter of having a system for handling emergencies, a pattern for activity in the face of life-threatening wounds and illnesses becomes even more important when the hospital is suddenly confronted with a mass influx of patients from some local catastrophe such as a fire, explosion, hurricane, flood or similar misfortune.

VENIPUNCTURE

Perhaps the most important feature of venipuncture is selecting an appropriate vein. Since the antecubital veins are usually quite prominent and fairly easily entered, they are often used for withdrawing blood samples. However the amount of motion possible at the elbow makes them poorly suited for continuous intravenous infusion. Also in a patient who is to require intravenous fluids over a period of many days it is wiser to start as far peripherally as possible so that as thrombosis occurs there will still be veins available proximalward from the site of the first venoclysis. In general, the superficial veins on the flexor and extensor surfaces of the forearm are best for the administration of intravenous fluids. However the veins of the foot are often easily used and the constancy and size of the greater saphenous vein at the medial malleolus make it a frequent site of election for venoc-

cerning dog bites revolve about the question of whether or not the dog is rabid. If the dog can be positively identified and kept under observation, the patient need not be given any prophylaxis against rabies. If the dog was in a pre-rabid phase of the disease at the time the wound was inflicted the animal can be expected to develop clinical signs of rabies within ten to fifteen days. At this time positive immunization of the patient with rabies vaccine can still be successfully undertaken. If the dog does not develop rabies during the period of observation then the matter can be dropped.

Technically human bites are in many ways much more serious than the average animal bite because the symbiotic action of Vincent's organisms found in the mouth of the human being produces an extremely virulent infection. For this reason, human bites must be given especially meticulous care and in most instances should not be closed primarily even if seen early.

Bites by other animals are quite infrequent but in many instances carry the same danger of rabies as do dog bites. Squirrels, skunks, cats, and other small animals have been known to transmit rabies to human beings. In the instance of a bite from such a small animal if the animal can be kept under observation the same procedure is followed as is done with the dog. In most instances, however it is impossible to make a positive identification of the animal, much less keep it under observation. Therefore, to be on the safe side, individuals bitten by such animals should be given the Pasteur antirabies treatment.

Protection against Tetanus. Many wounds carry with them the potential danger of tetanus. This is particularly true of wounds inflicted by rusty implements or nails and of wounds contaminated with street dirt or farm dirt. The spores of tetanus are known to be widely distributed in nature and to occur as contaminants of various animal furs and hairs. Since tetanus organisms are anaerobic, they will multiply best and elaborate their toxin in the depths of a closed wound. It is possible that they thrive even better in an infected wound where other pathogens are present to use the oxygen, thus producing a more truly anaerobic environment. Thus, one form of prophylaxis against tetanus consists of removing so far as possible contamination in the depths of the wound and in every way possible preventing any wound infection however this alone is not sufficient because tetanus car-

ries such a high mortality when it does occur. Therefore, two means of giving the wounded individual immunity are in use. The first is to confer passive immunity by means of tetanus antitoxin. The second is that of developing the patient's own active immunity to tetanus by means of tetanus toxoid.

An effective passive immunization which will last for at least one week can be conferred on an individual by giving tetanus antitoxin which is derived from the serum of horses. The standard dose has been 1500 units, but many physicians now are giving a minimum of 3000 units and some give 5000 units. This confers immunization immediately but immunization is not effective longer than one week. If the wound for which this was given continues to be infected the dosage of tetanus antitoxin must be repeated. Because there is an appreciable incidence of sensitivity to horse serum, tetanus antitoxin should not be given to a patient without first testing the individual for sensitivity. This may be done by means of intradermal tests or as some authorities feel, more effectively by means of conjunctival tests in which a drop or so of very weak solution of antitoxin is used. If there is a sensitivity and the situation requires definitely that antitoxin be given, it can be given in very small doses with some intravenous antihistamines and/or 5 mm of 1:1000 Adrenalin chloride. In some instances it may be necessary to use a rapid desensitization procedure.

Tetanus toxoid is an alum precipitated toxin which has thus been rendered non-toxic but which retains its antigenic properties. It is given in a series of three doses, usually at biweekly intervals to develop the individual's own immunity to tetanus. This procedure has many advantages over immunization with tetanus antitoxin, not the least of which is the fact that there is no real sensitivity to the tetanus toxoid. Occasionally a patient may develop a local reaction at the site of injection but this is transient and is never accompanied by systemic signs. Another advantage of tetanus toxoid is that the titer of immunity so produced can be raised to an effective level with a so-called booster dose of 0.5 cc. of toxoid at long intervals after the original immunization series has been given. Authorities now feel that there is still a responsive level almost ten years after the original series of immunizations. It might be pointed out that tetanus toxoid cannot be invoked

ber beneath the intravenous fluid bottle will tell whether the flow of blood is being compromised by any of the fixation procedures.

As a final step it may be necessary or desirable to place the extremity used for the intravenous administration on an arm board or other splint. This is mandatory of course, when the vein being used is one on the back of the hand or on the foot. When the vein is on a relatively flat surface on the extensor part of the forearm or on the flexor surface, the forearm itself may provide sufficient immobilization of the vein.

"Cut-down" and Cannulating of a Vein
Occasionally it is desirable to have a more dependable route for administration of intravenous fluids or blood than is afforded by the ordinary venoclysis procedure. In such instances a vein usually the greater saphenous at the medial malleolus, is exposed by cutting down on it after the surrounding area has been locally anesthetized. The distal end of the segment exposed is then tied and the ligature held with a hemostat to provide traction on the proximal portion of the vein. Another ligature is passed under the proximal end of the exposed segment and one tie taken loosely. The middle of the exposed segment is then partly cut through with a small scissors of the manicure type. The V-shaped flap of vein wall thus produced is gently lifted up by a small tissue forceps while a cannula is slipped into the lumen immediately beneath it far enough for the knob on the cannula to lie proximal to the loose tie (Fig. 2). The tie is then secured by squaring the knot and taking an additional loop in that knot. This ligature is cut so as to leave the ends $\frac{1}{4}$ to $\frac{1}{2}$ inch long.

This facilitates the subsequent cutting of the knot and removal of the cannula when the intravenous therapy is to be discontinued. The distal knot is cut flush as it will be left in. One or two skin sutures are placed in the skin and taken down loosely so that the cannula may be subsequently removed and the ends of the ligatures then drawn down snugly and tied to close the skin wound. It is important to note the location of the greater saphenous vein before making the cut-down so that the incision can be made in the proper place. The cut-down should be made parallel to the direction of the vein instead of transversely so as to facilitate the exposure of a fairly long segment of vein through a relatively small skin incision paralleling it. A transverse wound must be unduly long to permit the exposure of an adequate segment of vein. After the cannula has been well placed, fluid should be started immediately to keep the blood from clotting in the cannula.

An alternative to the use of the cannula is the placing of an indwelling polyethylene tube. This method of giving intravenous fluids is particularly popular when the fluids must be given for many days, or even weeks. The cut-down is accomplished in the same way as it would be for cannulation. Usually it is not necessary to place a tie distal to the opening in the vein because the pressure of the tubing below the cut-down against the distal segment of the vein occludes it. If the size of the tubing is such that it fits snugly in the vein and if it is fed proximal ward for at least 3 inches, there should be no retrograde leaking of blood or fluid through the wound. A snug pressure dressing over the cut-down wound helps to prevent such leakage.

CARE OF MECHANICAL EQUIPMENT

Surgical techniques have now advanced to the point where one is often dependent to great degrees upon mechanical aids, such as suction and tidal drainage systems, sump arrangements and underwater drainage bottles like those used in draining the pleural space after thoracotomy. These mechanical aids are accepted and employed so routinely that often it is not realized how significantly they may affect the success or failure of a given operative procedure. A simple account of selected incidents cannot begin to give a conception of the misery for the patient and the heartache for the surgeon who are both victims of mechanical failure but it may serve to alert the members of the surgical

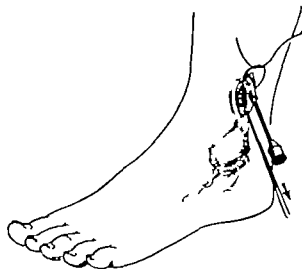


Figure - Method of cut-down and cannulating of vein for introduction of fluids.

lysis, particularly when this is accomplished by cut-down and the placing of a cannula.

Obviously the vein must be full to be located and particularly to be entered by the needle. Superficial veins are best filled by occluding their centripetal flow by means of a blood pressure cuff placed above the vein and inflated to a pressure of 40 mm. of mercury. Usually it is not necessary to invoke such an elaborate procedure and an ordinary tourniquet can be gently tightened around the arm or leg above the place where the venipuncture is to be done. Active or passive contraction of a muscle distal to this tourniquet will free more blood to fill the chosen vein.

Different individuals have different ways of making the actual introduction of the needle into the lumen of the vein. The beginner will do well to bear several fundamental facts in mind. First the segment of vein which is to be entered must be relatively fixed so as to offer some resistance to the point of the needle. This fixation is best accomplished by drawing back on the skin over the vein at a distance of 1 to 2 inches below the projected site of puncture: this leaves a segment still filled with blood relatively fixed so that it does not roll over the point of the needle (Fig 1). Second, when it is felt that the point of the needle lies against the wall of the vein a short, swift jab is required to put the point of the needle which is impinged against the wall, actually through the wall. A little experience will teach the beginner much about the amount of force that is required for this maneuver. However the stroke should be short so that the needle does not go completely through both walls of the vein and it should be directed at an acute angle to the vein wall, preferably with the open end of the needle facing out away from the vein wall instead of down and parallel to it. Once the tip of the needle is in the vein a meticulous effort should be made to feed the needle slowly into the lumen of the vein so that at least an inch of it is inside the lumen. With the needle held at its point of entrance through the vein wall, this gives a long arm and consequently poor mechanical advantage to any movements which might force the tip of the needle through the vein at still another point: consequently it makes it much less likely that the needle will come out of the vein or that there will be any extravasation.

The moment of entry of the needle through the vein wall is usually signalled by the appearance of a small spurt of blood just

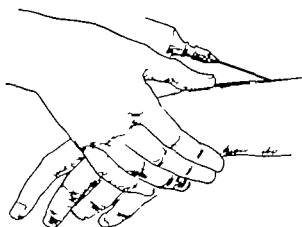


Figure 1 Fixation and puncture of superficial vein for introduction of intravenous fluids.

proximal to the hub of the needle within the syringe or the intravenous tubing. If one feels that the needle is in the vein and yet the spurt of blood fails to appear gentle traction may be made on the column of blood by pulling back on the plunger of the syringe or by milking back on the tubing to invoke such an outpouring. If blood still fails to appear and one is still certain that the needle is within the lumen of the vein the injection should be given slowly or the clamp on the intravenous fluids released to allow flow of a small amount of fluid. If the needle is out of the vein, there will be immediately a swelling at the site where the fluid is being deposited outside the vein wall. In this event, the venipuncture must be attempted again at a point proximal to the point of failure. The latter point, the site of the original entry should be covered with a small dressing and pressure maintained to prevent undue extravasation of blood through the point of rupture of the vein wall.

Once the needle is well established in the lumen of the vein, it must be fixed to the skin to prevent its being dislodged subsequently. This is best done by placing a small gauze pad under the hub of the needle and then placing adhesive tape over the hub and gauze to hold the needle down against the skin. Additional pieces of tape should be used to hold the intravenous tubing to the skin at a point 3 to 4 inches beyond the site where the needle entered the vein. In all this taping and positioning of the needle, one must be sure that the tip of the needle is not angulated against the wall of the vein or that the pressure is not so severe as to cause the needle to push the vein wall down into the tissues beneath it and thus occlude the flow of blood past the needle. Observation of the dripping of the fluid within the glass cham-

ods of performing any given procedure to accomplish the same result

Surgical Dressings. The application of surgical dressings is an old and time-honored procedure which is done differently by various surgeons. The basic principle in applying a surgical dressing is that it should be done with the same precision and careful attention to aseptic technique as is practiced in an operating room. Such an exacting end can be achieved by the simplest management of details by the attending surgical staff with the help of a cooperative and intelligent house staff.

In most modern hospitals satisfactory provisions have been made for surgical dressings by means of dressing trays or carts. In some institutions, individual packages are wrapped with materials for individual dressings. Regardless of the technique of carrying the dressing equipment and materials to the patient's bedside, the basic principle should be the same.

The equipment on the standard surgical dressing cart should include sterile canisters which contain gauze dressings of various size, depending on the particular type of work being done on the service, standard sizes being 3-inch and 4-inch square gauzes. There should also be canisters containing sterile towels. Other canisters will be filled with smaller gauze squares, sterile test tubes and applicators for the ready culturing of drainage from wounds. A variety of hemostats, forceps and scissors should be assembled in a flat, sterile, covered basin and there should be bottles for the various types of solutions which are used in changing dressings. Also included should be various types of thicker pads containing either cotton or cellulose material, sterile and wrapped in toweling. These dressings will vary in size depending upon the nature of the surgical service. Most surgical dressing carts should contain pads, 8 by 12 inches in size, which are commonly labeled "ABD pads," signifying abdominal dressings but which, of course, may be used for other purposes as well. At some institutions larger pads of the same type are labeled "arm pads," since they will enclose an entire extremity. When a great many burned patients are being taken care of, even larger dressings of this same general type should be available. Various sizes of gauze, roller bandage, elastic bandage, muslin bandage and adhesive tape should also be supplied on the dressing cart.

Provision should be made for the collection of soiled dressings which should not be

allowed to touch the bedclothes or the sterile canisters on the cart. In some institutions, provision is made for these dressings by means of a garbage-can type of container on the floor in the hospital or attached to the dressing cart so it may easily be removed. This should be covered so as to prevent insects from spreading the organisms about the hospital. Provision is also made for the collection of used or soiled instruments on the cart which may be returned to the central supply depot for re-sterilization and re-use.

The solutions on a surgical dressing cart vary somewhat with the desires of the particular surgeon involved. On most dressing carts such solutions as benzine, acetone and alcohol which are necessary in removing adhesive tape from the skin are commonly present. Most modern dressing carts do not have the stronger antiseptic agents on them since few surgeons employ these agents which were so commonly used in the past, but they do contain a bottle of liquid soap, or one of the emulsifying agents, for use in cleansing skin. Also available should be flasks of normal saline solution and of sterile water. There should be a lifting forceps immersed in alcohol in a jar of some type, either metal or glass. This sterile forceps is used for lifting materials from the sterile canisters for each dressing so that the canisters will not be contaminated by repeated use of the same instruments which are employed in doing the dressings. Surgical masks should be required supplies, sterile gloves and gowns may be provided as desired.

Regardless of the type of special dressing which may be needed in a particular case, the technique of handling all dressings should be essentially the same. All in attendance, including the patient, should have on a surgical mask to prevent the contamination of the wound by organisms from the mouth and nose. At the patient's bedside, a sterile towel is removed from the canister by means of the lifting forceps and spread out on a convenient bedside or overbed table. It has been found useful to place a piece of folded newspaper beneath this towel to protect the surface of the table from any of the solutions which may reach it. Toward the edge of this towel are placed one or two sterile hemostats and forceps from the tray containing the instruments and scissors if these are needed. These instruments are placed with the handles close to the edge so that they may be picked up without contaminating the center of the sterile towel.

house staff to the importance of simple attention to all mechanical aids.

A patient who had had a subtotal gastrectomy was returned to his room in good condition with the Gomco suction working properly. During the course of the remainder of the morning and the first two hours of the afternoon the suction did not function well. Unfortunately no member of the house staff was available to examine it or to make sure that it was properly irrigated and that its function was re-established. When the patient was finally seen after 3 P.M. a few blood clots were aspirated through the small caliber Levin tube but the patient's general condition made it obvious that he was bleeding into the stomach and that large clots had formed, making it necessary for him to be returned to the operating room. There the gastrectomy was taken down and the stomach emptied of approximately 1200 cc. of blood most of which was clotted. No discrete bleeding point was found in the site of the anastomosis and the general conclusion was that had the suction been working well the stomach would not have become sufficiently distended to allow continued oozing from the stoma.

A patient on whom a mitral valvulotomy had been done withstood the procedure quite well. Her chest was closed with two drainage tubes leading out from the pleural cavity to 1000-cc. flasks filled with 700 cc. of sterile water. The drainage tube was connected to a piece of glass tubing which emptied well beneath the surface of the water in the bottle. In one of the bottles the glass tube had been cracked at a level above that of the water. When the patient was being put into bed and the jars adjusted on the floor the broken piece of glass fell off its insecure connection to the tubing which returned through the cork of the bottle. This permitted immediate influx of air into the patient's pleural cavity and a pneumothorax of several thousand cubic centimeters developed almost at once in this patient whose condition was serious at best. The source of the difficulty was immediately detected and the drainage tube was clamped until oxygen under pressure could be administered through an endotracheal tube and the lung blown out at once as the air which had been inadvertently allowed to enter was exhausted through the other underwater seal.

An elderly woman who had been subjected to choledochotomy for numerous common duct stones, which had produced jaundice, was returned to her room in fairly

good condition but had a period during which she was somewhat irrational. At this time she managed, in thrashing around, to pull out the T tube which had been inadequately secured to the skin of the abdomen. Another operative procedure was required to replace the T-tube and, of course, this second operation was an unnecessary demand on her feeble reserves. Fortunately she survived, but on the seventh postoperative day developed a biliary fistula about the T tube which required sump drainage with a Chaffin tube and Stedman pump. Because of an unnoticed kink in the tube connecting the Chaffin sump tube to the pump no effective pressure was put on the sump arrangement for twenty four hours. When it was noticed that there had been no drainage accumulating, more careful attention was paid to all details of the apparatus and the obstruction was immediately detected. When this was relieved there was a gratifying return through this sump.

Every surgeon could elaborate at some length on this brief list of misadventures resulting from failure of mechanical equipment—failure contributed to by the negligence of nurses or doctors, failure which should have been noticed in time to prevent the difficulties enumerated. For example, most surgeons have had T tubes and catheters whose lumina were plugged with debris the presence of which they have detected by testing the equipment before using it.

Nothing can be taken for granted in the utilization of mechanical aids in the care of surgical patients. Every piece of equipment must be checked before it is used and its continuing function must be checked at frequent intervals as long as the patient is dependent upon that particular equipment.

SURGICAL TECHNIQUES

The senior surgical clerk often approaches his clerkship in a hospital with little or no knowledge of many of the technical procedures which he is asked to perform. First year surgical interns who have not had a broad general surgical clerkship may also have had no contact with many of the procedures which are commonly performed in a hospital by a junior surgical intern. The techniques of these various procedures are obviously best taught person to person by one of the surgical residents, or attending staff of the hospital, directly to the man involved. Of course, there are several meth-

a better inflow of arterial blood with more effective rapid healing. Compression dressings are often used in the treatment of burns on the head, the chest and the extremities. Such dressings are difficult or impossible to apply on the abdomen and seldom find a useful place there except possibly in the instance of burns. The general principle in applying a compression dressing is that the compression must be uniform and not tight in any particular locality so as not to obliterate venous return or arterial inflow into a part. The gauze dressings are applied in a manner basically similar to that used for general dressings. The gauze protective dressing is then padded with a mass of either fluffed up dressings derived from "flats" of gauze or as in some institutions, sterile mechanics waste. Another material often used for compression is sponge—either synthetic rubber or sea sponges. These materials are then bound down by an elastic bandage which may be reinforced or tightened by means of safety pins or adhesive tape. The materials for an elastic bandage vary somewhat with different institutions.

On the chest the most commonly used materials for compression dressings, particularly after radical mastectomies and procedures of that type, are stockinet of sufficient width, usually 6 inches or elastic bandages which may be the two-way stretch type or rubberized elastic bandages. The elastic type bandage used on the extremities may be of either of these types as well. An important fact to remember is that such a dressing applied to an extremity must start at the distal end of the extremity and extend proximally as far as desired and not in the reverse direction. Leaving an exposed area of extremity below such an elastic compression dressing usually results in edema and discomfort and may actually do harm to the patient. Elastic type compression bandages have a wide usefulness but are particularly helpful in the handling of burns and open wounds. They also have considerable utility in the dressing of any wound about an extremity, but one must avoid extreme pressure over the bony prominences, which must be well padded. The dressing must be applied smoothly and without wrinkles, which are uncomfortable to the patient and may produce undue pressure in a particular spot. It is usually applied without reversing the bandage as is done in placing a roller bandage.

For large wounds of the scalp and for craniotomy incisions, the most satisfactory dressing is one designed by Cushing which

employs a 5-yard roll of sterilized gauze 5 inches in width. After applying the original dressing, two turns of the 5-yard roll fix the dressing about the forehead and occiput. The roll is then taken down under the chin and while an assistant holds this gauze under the chin at a convenient distance so as to prevent making it too tight, two or three turns are made over the vertex so as to cover the remainder of the head completely going under the chin each time. It is then turned back to complete the circle around the forehead and occiput to fix this chin strap. The turns of this dressing are then fastened in place by means of safety pins which may be twisted so as to tighten the whole into a snug, close-fitting dressing. Straps of 1 inch adhesive or ties of 1 inch roller bandage are then applied about the chin strap, one on each side in front of the ear and one at the bottom of the loop under the chin so as to make a single, neat, compact chin strap. Several pins are then applied between the circular layers about the forehead and occiput and the deeper layers over the head so as to fix the latter firmly in place. This dressing is so secure, if properly applied, that even a disturbed patient who is not responsive for his actions may have difficulty in getting it off (Fig. 3).

For small lacerations or incisions in the scalp sterile fine-mesh gauze made adherent to the skin by means of flexible collodion makes an excellent dressing. The gauze is trimmed to appropriate size from a sterilized roll of gauze roller bandage; usually four to six layers of this material are incorporated in the dressing. After being trimmed with the sterile scissors to the size desired, a layer at a time is applied to the scalp over the wound and the flexible collodion is painted

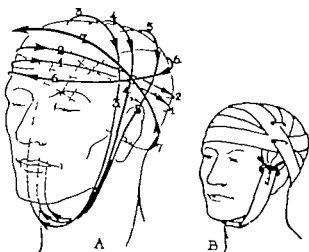


Figure 3 Diagram of Cushing type head dressing.

Various quantities of dressings of appropriate sizes are placed on the towel. Then every thing is in readiness for the changing of the dressing.

It should be stressed that unless it is absolutely necessary for adhesive tape to be removed from the patient's skin each time a dressing is changed, it is better to clip the tape with a pair of bandage scissors at the edge of the dressing so as to avoid the irritation caused by the repeated application and removal. The outer part of the old dressing is discarded onto a newspaper which can be folded up and placed in the receptacle provided for dirty dressings. The deeper dressings next to the wound are removed by means of the sterile instruments that were placed on the sterile towel. These dressings are likewise dropped on a paper for discard. Frequently the first dressing may be somewhat stiff and adherent to parts of the wound because of the collection of serum or even blood which has dried on the dressing. As a consequence, the dressing should be removed cautiously to prevent pain to the patient as well as injury to a healing wound. Experience in doing this thoughtfully and carefully with concern for the patient's comfort increases the dexterity and skill of the operator.

Once the dressing has been removed the wound may be inspected but should not be touched with the fingers since it is regarded as relatively sterile. If sutures are to be cut or removed, this is done carefully using sterile instruments from the sterile towel and avoiding contamination of the working portions of these instruments. If the sutures are removed it is often found helpful to apply support to the skin which, though healed, may be easily separated by ordinary pull which may produce an unnecessarily wide scar. Materials most commonly used for support to the skin are adhesive bridges made by folding under the portion of the adhesive tape which will come in direct contact with the wound. These bridges are made relatively clean by cutting them with clean scissors and then painting them with an antiseptic solution or flaming them. They are applied in such a manner as to take the tension from the healing wound. They are particularly useful in a long wound and it is better that they be applied as the sutures are removed, rather than afterward, since the sudden movement of the patient may separate the wound. Another excellent material for relieving tension from the healing wound is fine-mesh gauze, usually obtained

from roller bandage which has been sterilized and is cut into short strips and made adherent to the skin by means of flexible collodion applied with sterile applicators to the whole of each strip of gauze.

Sufficient gauze dressings are reapplied to the wound by means of sterile hemostats or thumb forceps to cover the wound adequately. This means an overlap at both ends of the wound of perhaps 1 or 1½ inches and 2 to 3 inches on each side. This dressing is usually covered by an abdominal pad or some thicker layer of material which prevents further contamination.

The smallest amount of fixation possible in abdominal wounds is advisable. The amount of irritation to the skin from adhesive tape, which is most commonly used to fix these dressings in place, is reduced by using a minimum of tape. However tape strips should be long enough to fix the dressings well so that they do not slip off. The use of any sort of liquid on a healing wound that is dry and clean seems superfluous. A wound that is open or draining, of course, should be cleansed and the most satisfactory materials for this are soap and water which should be sterile, applied with sterile gauze sponges held in the hemostats and thumb forceps supplied on the dressing carts.

Unused gauze which has not been soiled, but has been brought out of the sterile containers is never returned to the containers but is placed in a receptacle provided for that purpose, to be returned to the central supply room for re-sterilization.

Although the technique for surgical dressings described above may seem time consuming to those who have been somewhat less formal about dressings, it is a safe and a sure one for preventing contamination, regardless of the type of wound involved and is a sound one for young surgeons to develop.

Within the last few years, hospitals in this country and abroad have been plagued by severe and often fatal surgical infections in which the offending organism was a coagulase-positive staphylococcus. In the face of this extremely grave problem, the importance of following a good aseptic surgical dressing technique cannot be overemphasized.

Special types of dressings. Most surgeons have found that mild elastic compression upon wounds is helpful in the healing process. This is true because support of the venous system, particularly in the extremities, improves drainage from the area so that less edema occurs and consequently there is

the wound thoroughly with a safe margin on each side. Such dressings are usually fixed by means of adhesive tape the narrowest type practicable for the size of dressing being used. If some support for the abdominal wall is desired with the dressing, it is common practice to use 2 inch tape. In those cases in which this is not necessary, 1 inch adhesive tape straps supply enough protection with half as much irritation to the skin. For repeated dressings upon abdominal wounds of smaller size, even $\frac{1}{2}$ inch adhesive straps may be used. It should be emphasized that although these straps must go far enough past the edge of the gauze to become firmly adherent to the skin they need not cover large expanses of skin which would lead to unnecessary irritation. If on changing dressings such adhesive straps are cut at the margin between the adherence to the skin and the adherence to the gauze, rather than being removed each time much less irritation of the skin occurs. The new straps can be applied over the old and, in the average elective case, by the time it is unnecessary to have further dressings the elastic adhesive will be so free as to be readily removed with out much pain to the patient. Another means of preventing irritation to the skin from adhesive tape is to treat the skin with tincture of benzoin before the application of the tape.

Spinal Puncture. Lumbar puncture is a procedure which is often done on surgical and medical patients. Every house officer should try to develop facility and dexterity in this useful procedure. Depending upon the circumstances for which the puncture is done, the patient may be put in a sitting posture or may be laid on his side. In either position it is important to have flexion of the lumbar spine so that the spinous processes adjacent to the fourth interspace, the usual site of puncture be opened as much as possible. When the puncture is done with the patient lying on his side, this can be effected by having him bring his knees up on his chest and bend his head down upon his chest. With the patient in this position it is important that he be brought well to the edge of the bed where the added support of the bed frame will maintain the relatively straight alignment of the vertebral bodies, as opposed to the scoliosis which may occur on a soft bed when a patient is allowed to lie on the center of it.

The house officer should never attempt a lumbar puncture without previous instructions from his attending surgeon and should

always be supervised in his initial attempt at this procedure. Sterile technique should be observed in that those present should wear caps and masks and those actually doing the procedure should wear sterile gloves. The skin should be scrubbed over a wide area from the sacrum up to the lower thoracic spinous processes and from one flank to the other using soap and water. It should be draped with sterile towels. The point directly between the tips of the third and fourth lumbar spinous processes in the midline should be infected with 1 per cent procaine until a 1-cm. wheal has been raised. Then, with a longer needle inclined slightly cephalad but maintained directly in the midline, the injection should be continued as the needle is pushed forward to infiltrate the interspinous ligament. Usually infiltration need not be carried deeper than 2 inches. The spinal puncture needle is introduced into the skin with a short thrust and then slowly pushed forward. If it is arrested by striking bone the needle should be pulled out and introduced at a less acute or more acute angle as the case may require. Although its inclination somewhat caudad of perpendicular may be altered to some degree, the needle must be maintained exactly in the sagittal plane at all times.

When the needle has finally penetrated the spinal canal, this event will be signalled by its suddenly "falling into" this space. It is imperative that the needle be guided by both hands and that it be always held in such a manner that it cannot penetrate too deeply in a sudden thrust when the resistance of ligaments ahead of it gives way. This, of course, is the principle in all procedures involving the introduction of a needle into a hollow cavity. When the needle is in the spinal canal the stylet is gently withdrawn to see whether spinal fluid will drip from it. If the needle is correctly placed, there will be a slow dripping of fluid from the hub of the needle. If there is no such dripping, the needle may be presumed not to lie in the spinal canal and it should be withdrawn and reinserted gently and slowly. If the needle is advanced too far it will strike nerve roots and the patient will complain of sharp pain, usually down the back of the thigh. If on withdrawing the stylet the operator obtains a flow of blood, the needle should be withdrawn and the puncture attempted at a different level. Once an adequate flow of spinal fluid is obtained, the stylet should be inserted in the needle while preparations are made to attach a three-way stopcock for de-

on with an applicator around the periphery of the gauze, layers being added one after another with collodion being applied to the edges of each layer to make it adherent. This produces a neat dressing which does not become involved with hair and will remain in place for several days. These dressings are useful for wounds on the face or an exposed extremity where not too much is needed to cover the wound. An occasional patient is sensitive to collodion and may develop skin irritation from its use.

Compression dressings on the chest are usually applied following radical surgery upon the breast. These are used primarily because the large skin flaps necessary in this operation have to be kept firmly in place against the deeper structures in the chest wall if they are to adhere without the accumulation of fluid beneath them. A similar type of dressing is sometimes used by thoracic surgeons after extensive procedures on the chest. However the need for skin flaps in this situation is unusual and therefore compression is not so essential as it is following radical mastectomy or skin grafting to the chest wall. After the usual occlusive dressing is applied over the wound, a large mass of resilient material such as fluffed up gauze or mechanic's waste is mounted up in bulky and generous fashion over the area for which compression is wished. It is important to make certain that any bony prominences which may be encountered in applying the dressing are covered well with padding. The dressing is usually started with a 6-inch stockinet roller at the waistline and is fixed there by two turns of stockinet around the waist pinned in place with safety pins. This is continued as a simple roller bandage overlapping some 50 per cent on each turn until the entire chest is covered running from the back up over the shoulder on the involved side, catching the arm in a figure-of-eight spica, and coming back after encircling the arm to go once more around the chest, thus completing the spica bandage. This type of compression can then be tightened by means of safety pins, as in head dressings so that firm compression can be maintained with comfort to the patient and without embarrassment of the respiratory function. Such a dressing is often left in place for three, four or five days, depending upon the conditions for which it was applied. A similar type of dressing can, of course, be applied with elastic bandage materials but firmer pressure and more effective dressings result with the use of the stockinet.

Recently some surgeons have dispensed with the use of heavy compression dressings. To prevent the accumulation of serum and blood beneath widely undermined skin flaps, they have resorted to a type of sump drainage. This involves the placing of catheters or polythene tubes beneath the skin flaps where fluids might be expected to accumulate. These tubes have several holes of generous size cut into the part which is to be buried. The outer end of the tube is connected to a Gomco suction apparatus. If this technique is properly employed, it prevents the accumulation of fluids beneath the skin flaps and eliminates the need of bulky dressings.

Often it is better not to use dressings about the face if the patient is to be kept in a sheltered location such as a hospital room, since the application of any dressings to small wounds may result in collection of serum or a few drops of blood along the suture line. The blood or serum may harden and interfere with the healing process, particularly if the dressings are removed without the greatest of care. This is especially true of the face because dressings often are taken off quite early to permit removal of the suture material in the skin in order to prevent scarring due to the sutures themselves. Wounds of the face treated without dressings are simply wiped off intermittently every few minutes for the first hour or two by means of a sterile applicator gently rolled over the wound so as to pick up any small amount of serum or blood which may have accumulated on the wound. After one or two hours these wounds, if they have been properly closed, are quite dry and sealed so that further contamination by bacteria is unlikely especially if the hands and foreign materials are kept away from them. For nicely closed fresh wounds, this is a satisfactory procedure. For patients who are to leave the hospital or doctor's office some sort of a dressing should always be applied. The most satisfactory dressing on the face for small wounds is a fine-mesh gauze collodion dressing. For those wounds about the face which need compression dressings, one must be improvised from some of the materials which have been mentioned depending upon the location of the injury or wound.

Dressings on the abdomen need not be too large, or too thick, since in the average clean elective case they are not expected to absorb purulent materials. They are simply used to prevent contamination of the wound by bacteria from the air, bedclothes and fingers. However abdominal dressings should cover

sion, but often for diagnostic aspiration of minor effusions which are thought to have resulted from neoplasms of the lung. Unless circumstances dictate otherwise, puncture is usually made in the posterior axillary line at the lowest level in which it is expected that fluid can be encountered. This level is determined by percussion and x ray findings. The puncture of the pleural cavity is usually made with the patient in the sitting position although circumstances may require that the patient be left on his side. Many surgeons feel that the patient should be given $\frac{1}{4}$ to 1 grain of atropine at least one-half hour before the procedure to eliminate any possibility of a noxious reflex mediated by the vagus nerves. The procedure should be done under conditions of sterile technique with the house officer capped, masked and wearing sterile gloves. The skin may be scrubbed widely with soap and water or iodine and alcohol, or another acceptable antiseptic used.

The field is draped with sterile towels. A 1 per cent solution of procaine is injected into the skin to produce a wheal through which the deeper tissues are infiltrated with another 5 to 10 cc. of the same anesthetic. The thoracentesis needle is slowly introduced through this infiltrated area just beneath the edge of the rib until the feeling of resistance suddenly disappears and the needle sinks in rather quickly. The house officer should be prepared for such an event and should be holding the needle so that it cannot suddenly fall into the pleural space to any depth. The pleural fluid is usually withdrawn by means of a three-way stopcock which prevents the influx of air into the pleural cavity. When the thoracentesis has been performed for relief of pneumothorax, it is particularly important that this precaution be observed. It is also important that a large pneumothorax not be aspirated too quickly as a sudden shift in the mediastinal contents could produce cardiac distress.

When the aspiration has been completed, the needle is quickly withdrawn through sterile gauze held close about its point of entrance through the skin. The area is then sealed with a small piece of cotton impregnated with collodion.

OBSERVING THE AMENITIES

Any member of the house staff who enters a patient's room, or comes to the bedside of a patient in a ward, should introduce himself and explain his relationship to the attending physician, as for example "I am Dr. Smith,

Dr. Jones' associate" or "I am Dr. O'Brien, a resident on Dr. Jones' service." There can never be any excuse for omission of this simple act of courtesy when an individual member of the house staff comes into contact with a patient. The attending surgeon will usually make such introductions on the first visit he makes to the patient in company with his intern and/or resident, but he knows that these men usually have seen the patient in the hospital before he has, so that introductions are not necessary.

Before the intern or resident undertakes any procedure with the patient, even so simple a thing as taking his history, he should tell the patient simply but in a clear manner just exactly what he plans to do. This may seem very fundamental, but far too many patients are examined, questioned, subjected to rectal examination, venipuncture and sometimes even spinal puncture without any preliminary explanation of what is about to take place. This is discourteous and leads to misunderstanding, fear and resentment. It is particularly important in dealing with children that simple, yet adequate, explanations be given in advance of any surgical procedures, even though it be so minor as the drawing of blood or the changing of a dressing. Children tolerate pain and unpleasantness surprisingly well if they have been warned about it in advance and if they do not feel they have been deceived by those who are caring for them.

Of course, the young surgeon's concern for propriety in relationships with the patient is not limited to the patient's room. When the patient is being taken to the operating room, it is imperative that the trip be made as smooth as possible. The cart should be handled gently and not bumped in the course of moving the patient from his room to another floor for surgical procedures. In the course of such a trip conversation between the house officer and the patient, if there is to be any, should relate to innocuous subjects. If there is any discussion of the forthcoming procedure, it should be brief and reassuring. The attending surgeon will, of course, have prepared the patient in advance for what is to occur in the operating room and, therefore, the house officer should do nothing to disturb the patient's serenity presumably already achieved by preoperative medication.

In the operating room and in the corridors of the operating suite, it is essential that an air of quiet prevail. Laughing, loud talking and even persistent whispering are incommen-

termination of manometric pressure and collection of fluid samples. If the puncture has been made while the patient is in the sitting position it is extremely important that a minimum amount of fluid be allowed to escape, since a sudden lowering of pressure could cause a herniation of the cerebellar tonsils through the foramen magnum. When increased intracranial pressure is suspected, the puncture should not be done with the patient sitting.

After a three-way stopcock has been attached, manometric pressures can be determined and spinal fluid samples collected. The stylet is then replaced in the needle and a small sponge is placed on the skin around the needle while the needle is quickly withdrawn. The puncture site is covered with a small piece of sterile cotton impregnated with collodion, this makes an adequate seal for the wound.

At the conclusion of the procedure, the patient should be instructed not to raise his head or to move about vigorously in bed as such activity may induce the so-called post spinal puncture headaches. Twenty four hours after the lumbar puncture, it is safe for the patient to do whatever was permitted before the procedure.

Passing the Stomach Tube. Occasionally in the emergency room, it will be necessary to empty a patient's stomach. In such instances a large Ewald tube is usually employed and it is passed through the mouth. It may be necessary to pass it on an unconscious patient, e.g. one who has taken an overdose of sleeping pills in a suicide attempt. The tube should be cold and lubricated with ice water or mineral oil, it should be arched posteriorly as much as possible so as to avoid an endotracheal passage. Once the tube is in the esophagus, it should be passed quickly until it is in the stomach. At this point a large aspirating bulb is applied to the end of the tube and the contents of the stomach quickly aspirated. Should the tube be inadvertently passed into the trachea, usually a tremendous gag response will be evoked. Should there be any question at all whether or not the tube is in the trachea, the issue can easily be settled by feeling the outer end of the tube for evidence of expiratory movement of air out of the tube.

In most instances the stomach tube used is a much smaller one, usually a Levin tube, which is passed through the nose and then through the pharynx and down the esophagus into the stomach. In many instances it is done as a preliminary step to some type

of surgery on the gastrointestinal tract in an effort to eliminate postoperative distention and to keep the gastrointestinal tract at rest. Such a tube can be passed in the patient's room. It should be cooled in ice and passed with no lubrication other than the ice water which adheres to its surface. The patient should be asked whether there is any impediment in one nostril or the other so that the tube can be put through the more patent of the two nostrils. When the tube reaches the patient's pharynx it may produce some gagging. At this point the patient should be instructed to swallow hard and if necessary even be given some water to drink as this will help carry the tube down the esophagus. Patients differ in their reactions to this procedure and some gag strenuously when the tube is in the pharynx. Again, if there is any suspicion that the tube has gone into the trachea, the outer end of the tube should be held next to the cheek or back of the hand to see whether expired air is issuing from it. If such is not the case and the gagging seems disproportionate to what might be expected the pharynx should be examined by means of a flashlight to see whether or not the tube is curling up in it. If it has curled up in the pharynx, the tube should be withdrawn through the nose until the tip can just be seen above the middle of the soft palate and then the swallowing should be begun again as the tube is slowly passed. Having the patient pant or breathe rapidly through the mouth may facilitate the process. The position of the tube within the stomach can be confirmed by aspirating gastric contents. Very rarely will a tube properly placed within the stomach, fail to yield some gastric contents. However if it seems that the tube is in the stomach and yet nothing can be aspirated, the tube should be gently irrigated with 50 cc. of water most of which should return on aspiration.

When a tube of the Miller Abbott type is being employed in an effort to apply suction at some point beyond the pylorus, it is important that the tube have appropriate markings on it so that the amount which has been passed into the stomach can be easily determined by glancing at the part which remains outside. When extreme difficulty is experienced in passing a tube, it may be necessary to spray the pharynx with a 2 per cent solution of cocaine, as is done before bronchoscopy.

Thoracentesis. This procedure is frequently employed. It is usually performed for the removal of fluid from a pleural effu-

SOFT TISSUE INJURIES FROM MECHANICAL FORCES

By JOHN L. BELL, M.D.

JOHN LOUIS BELL received his education at Duke and Northwestern Universities. He has devoted his special talents to the surgery of the hand and the treatment of burns and soft tissue injuries. He is an Assistant Professor of Surgery at Northwestern University. He represents the third generation of the school of surgeons originated by Kanavel, interested in the hand.

The meticulous and thorough efforts characteristic of the surgical teachings of this group to create a closed, clean wound out of an open dirty destructive injury have been multiply repaid by shortening the convalescent period, restoring function more completely and decreasing economic loss for the patient.

The body is threatened constantly by accidental trauma from extrinsic forces: burns, mechanical injuries, chemical injuries, cold injuries, blast injuries and radiation injuries. In recent times there have been as many as 93,000 accidental deaths and 9,500,000 injuries annually in this country. Many of the victims are killed outright but others die during their course of hospitalization. Traffic accidents account for 38,000 deaths annually or over one-third of the total. It should be obvious that any member of the medical profession may be called upon to render initial care to the injured.

Wounding or injury from mechanical forces is the most frequent form of trauma requiring emergency treatment. There may be no limit to the anatomic distribution of injuries, particularly in those individuals in-

jured in automobile accidents, tornadoes or other civilian disasters. These injuries often simulate the trauma of warfare. Both military and civilian authorities are aware of the potentials for mass casualties in this thermonuclear age.

All accidental injuries can be divided into closed or open wounds and the difference is determined by the integrity of the body surface. Wounds may be superficial or deep, obvious or concealed, and the tissue damage ranges from slight to the most severe.

A rational approach to the care of the injured is necessary because the deleterious effects from wounds are frequently widespread as well as local. Hemorrhage, shock due to trauma, and asphyxia produce life-threatening situations, which demand immediate treatment and take precedence over

patible with the competence and business-like performance which the patient expects in a room where his life may well be in jeopardy.

Even in conditions of extreme quiet, however a prolonged wait may be very disturbing to the patient's peace of mind, so he should not be brought to the operating room any earlier than is necessary for expeditious handling. When he has reached the operating floor he should not be left unattended in the corridor for any period but should be transferred immediately to the room where his operation is to be performed.

In the operating room the attending surgeon is responsible for whatever goes on. This responsibility is a moral and ethical one as well as a medicolegal one. In the last respect, it is only partially shared by the anesthesiologist and by the hospital through its salaried employees such as nurses, orderlies and other attendants. The surgeon must take full charge of all that goes on in the operating room and is completely responsible for whatever happens to the patient. Therefore, members of the house staff should do whatever tasks have been assigned to them and wait quietly for the operation to begin. It is not required of any house officer to ask the anesthetist about the patient's condition or to inquire of the scrub nurse whether certain instruments have been included. His greatest service to the surgeon lies in his readiness to do what he is told and in his ability to assist as directed in the actual operative procedure.

At the conclusion of the operation, some member of the house staff should assist the other attendants and the anesthetist in transferring the patient from the operating table to the cart and accompany the patient to his room to assist in the transfer from the cart to the bed. In the patient's room he should make certain tactfully that all members of the family are out of the room while the patient is being returned to bed and intravenous fluids and other paraphernalia are being adjusted and while he is checking the patient's vital signs. He should also check the functioning of mechanical apparatus at this time. He can render the nursing service a great help by asking that excessive quantities of flowers, magazines, cigarettes and other trivia be removed from the bedside table and other important working surfaces until the patient's convalescence has reached a point where these items do not interfere with efficient nursing care.

READING REFERENCES

- Davis, L. *The Principles of Neurological Surgery*. Ed. 4. Philadelphia, Lea & Febiger, 1953.
- Dunphy J. E., and Botsford, T. W.: *Physical Examination of the Surgical Patient*. Ed. 2. Philadelphia, W. B. Saunders Company, 1958.
- Ilgenfritz, H. C. *Preoperative and Postoperative Care of Surgical Patients*. St. Louis, C. V. Mosby Company, 1948.
- Patey D. H., ed. *An Introduction to Surgery*. Chicago, Year Book Publishers, 1958.
- Tuzewski, M. S. Simplified "Cut Down" (Venotomy) Technique. *J.A.M.A.* 157:572-575, 1958.

epithelium has regenerated. A clean abrasion heals by spontaneous epithelialization from the undamaged epithelial elements and skin appendages. Infection impairs healing and at times a partial thickness injury to the skin is converted into a whole-thickness loss.

A *laceration* is a linear wound created by either sharp instruments or blunt objects. Lacerations vary from neatly incised wounds to those with irregular torn and contused surfaces. Damage to the skin and underlying tissues is localized to the path of the wounding agent. Contused lacerations result in some tissue loss and beveling injuries often impair the blood supply to the thinner skin flap.

A *penetrating wound* is created by a missile or object. The energy which is imparted to the tissues is dependent upon the mass and velocity of the wounding agent. Tissue damage from low velocity missiles or instruments is confined closely to the tract of the wounding agent. The wound of entrance is usually small but the tract may be of any depth. Pieces of skin or clothing are frequently imbedded in the wound along with the retained missile. A perforating wound occurs if the missile or instrument passes completely through the tissues and emerges at a different location. Wounds of exit are usually larger than wounds of entrance in perforating injuries from missiles. High velocity missiles used in warfare produce penetrating wounds with extensive tissue damage beneath the surface. The external appearance of these wounds is misleading. An instantaneous but temporary cavity forms around the missile and its tract. The volume of this cavity depends more upon the velocity of the missile than its size. The energy imparted to the tissues in the creation of the temporary cavity can severely damage blood vessels, nerves and other structures at varying distances from the course of the missile. Bone fragments or shell fragments frequently act as secondary missiles and cause further and widespread tissue damage.

Avulsions are wounds characterized by the tearing of tissues from their attachments. Skin and subcutaneous tissues are either partially or completely avulsed from the underlying tissues and any degree or extent of damage to deeper structures can occur. Many flaps are so severely mangled that there is little doubt concerning their salvation. Frequently it is difficult to determine the viability of a partially avulsed flap, since gross damage to the skin, fat and blood

vessels may not be evident upon inspection. The survival of a flap is dependent largely upon the adequacy of the circulation to the part. The efficacy of venous return from the flap is a factor which determines ultimate survival of the tissue, even though the arterial supply appears to be adequate. If an obviously devitalized flap is replaced it soon becomes necrotic and infection frequently occurs.

Every combination of the various types of accidental wounds may be present in *open crushing injuries*. Damage to both the covering tissues and deeper tissues is extensive. These wounds are similar to some combat injuries. Two factors determine the severity of the compression damage—first, the area of the body involved and, second, the nature of the force and the duration of the application of pressure to the part.

First Aid. First aid is the immediate care given to the injured person at the scene of the accident. The manner in which it is performed may at times mean the difference between life and death. Frequently the services of a trained layman much less a physician, are not immediately available. The seriously injured person should not be moved hastily or roughly. Obvious fractures have to be splinted and an attempt should be made to control external bleeding. Obstruction of the airway is an immediate threat to survival, but often the relief is as simple as turning the head to one side and pulling the tongue forward to clear the oropharynx. Sucking wounds of the chest should be securely closed by an airtight dressing.

Open wounds are contaminated at the time of injury but further contamination can be avoided by applying a sterile or if such is not available, a clean dressing. Antiseptics, ointments or powders should never be applied to wound surfaces because of further contamination and possible chemical injury to the tissues. Usually external bleeding from extremity wounds can be controlled by applying compression to a dressing overlying the site of injury. Elevation is helpful if it can be done without too much manipulation. Routine use of tourniquets at the accident scene is to be condemned, for the harm from improper application outweighs any possible advantage in almost every case. If venous return is impeded but arterial inflow is not stopped, bleeding from an open wound of an extremity is accentuated until the constrictor is removed. When a tourniquet is applied too tightly the blood supply to a limb may be jeopardized, or direct and

all other measures of care. Further trauma to the patient and to the wound itself is prevented by careful handling. An accurate history and systematic physical examination are essential to assess the patient's condition and the total reaction to trauma, as well as to evaluate the wound. For patients with multiple injuries, priority of care is established for those wounds which threaten life. The wound is treated only after a working diagnosis has been established and other decisions involved in management are made, such as the necessity and urgency for surgical intervention and the choice of anesthesia.

There is no practical all inclusive classification for wounds or injuries. Terms differ and frequently many combinations of types are present in the same wound. However all accidental wounds are either closed or open. It must be emphasized that the external appearance of an open wound does not indicate the extent or degree of damage to deeper tissues. Many serious closed injuries occur without early or obvious external signs or manifestations.

Closed Wounds. Closed wounds are also called nonpenetrating injuries. Any tissue may be involved even though the skin remains intact. These injuries are characterized by the crushing, tearing or rupturing of tissues and by internal hemorrhage. Contusions, ruptures and sprains are specific examples.

A *contusion* is produced by a blow to the body by a blunt force or object and there is no obvious break in the continuity of the skin. Usually the damage is confined to the subcutaneous tissues, but deeper structures may be involved, depending upon the force of the impact. The tearing of small blood vessels causes bleeding and blood infiltrates the tissues. The damage to tissues results in exudation of serum. Discoloration beneath the skin and swelling are frequent external signs in superficial contusions. If a larger blood vessel is torn, hemorrhage may be profuse and extravasated blood may form a hematoma between the layers of the tissues. The blood supply to the surrounding tissues may be jeopardized by an expanding hematoma. If the overlying skin is deprived of its blood supply it may undergo varying degrees of destruction and lose its capacity to act as a protective barrier against infection. If swelling is excessive and confined to a rigid tissue compartment, the tissues may be injured by the effects from direct pressure. Severe contusions may lacerate underlying viscera or solid organs, particularly

in the head, neck and thoracoabdominal regions. Injuries to structures far removed from the site of the external impact are not uncommon because of two factors: inertia and the variable tissue resistance.

Ruptures of tendons, muscles and ligaments are caused by either direct or indirect violence. Muscle rupture may occur when two antagonistic groups of muscles contract violently at the same time. Ligamentous tearing is called a *sprain*. Ruptures and sprains vary from incomplete to complete disruption of the continuity of the tissues. The resulting deformity, swelling and loss of function are dependent upon the severity of the injury. Violent nonpenetrating blows may rupture hollow viscera or lacerate solid organs, particularly in the abdominal region. Serious closed injuries may occur in any region of the body.

Open Wounds. Open injuries denote a break into or an actual loss of the protective skin barrier. The underlying tissues are damaged to varying degrees and extent. Open wounds are contaminated by bacteria at the time of the trauma either by the wounding agent or foreign material introduced into the wound. Further contamination after injury is possible until the wound is closed.

There are many types of open wounds: *abrasions, lacerations, penetrating injuries, avulsions and open crushing injuries*. Military surgeons differentiate between high velocity and low velocity missile wounds. Frequently the degree of damage to deeper tissues is suspected by the nature of the trauma. In wounds involving the extremities, injuries to the nerves, muscles, tendons and blood vessels can be diagnosed by testing the function of parts distal to the site of injury. In penetrating injuries of the neck, abdomen and perineum, the ultimate extent of the injury may have to be proved by surgical exploration. No wound in any location should be probed in an attempt to establish the depth or extent of the injury. This maneuver cannot be expected to give reliable information but can cause further harm by accentuating hemorrhage and introducing further contamination.

The most superficial type of open wound is an *abrasion* which involves destruction of varying depths of the skin. Bleeding from the injured skin surface is slight and is accompanied by serum exudation. A dried crust of blood and serum usually forms with in a few days and unless infection occurs, the crust should not be disturbed until the

another form of trauma and tissues must be protected from excessive operative injury. Aseptic technique, gentleness in handling tissues and meticulous hemostasis are essential to any surgical intervention.

The optimal time after injury for the initial care is within the first eight hours which is usually considered to be the time prior to actual bacterial invasion of tissues. This interval is often called the "golden period" however there are many other factors which have to be considered other than an arbitrary limit of time. For example because of an excellent blood supply most wounds of the face can be cleansed and repaired safely as long as twenty four hours after injury. The history of the contamination, the type and location of the wound and the extent of tissue damage will influence the extent and character of the initial treatment.

The adverse effects upon wounds from the presence of devitalized tissue are impaired wound healing and local or invasive infection. Devitalized tissues soon become necrotic and provide an ideal culture medium for bacterial growth. Only living and healthy tissues can effectively combat certain degrees of bacterial contamination. It should be recognized, however that if the number of virulent microorganisms present in any wound is excessive, infection may not be avoidable in spite of the inherent ability of living tissue to combat bacteria.

Wound Cleansing Wound cleansing, using aseptic precautions, is an integral part of the initial treatment. Every open wound is contaminated and frequently foreign material is imbedded in the wound. For extensive wounds preparation of the proposed operative field is performed after the patient has been anesthetized. The surrounding skin is shaved and carefully cleansed with soap or a bland detergent and sterile water while the wound is protected by a sterile dressing. Following thorough cleansing of the skin, the wound is carefully irrigated with sterile saline solution and any loose foreign material is removed. The details of technique may vary with different surgeons, but the procedure is essentially a mechanical process with care being taken to avoid irritation of the skin or trauma to the tissues. For the least serious type of wound at least ten minutes is required. In extensive injuries, more than one change of sterile gloves and materials is required and additional time should be taken to insure the cleanest field possible.

It is necessary to prepare an area large enough to carry out the operative procedure without fear of contamination from unprepared skin surfaces for the original wound may have to be enlarged. For wounds of the extremities the entire circumference is prepared, and in hand injuries the entire wrist and lower forearm are cleansed.

Wound Excision Wound excision, often called débridement, is the foundation of initial operative care of open wounds and it is most effective if performed as soon as possible after the injury. Its objective is to convert the accidental wound into a surgically clean wound which contains only viable tissues with an adequate blood supply. This goal is accomplished by the removal of devitalized tissues, foreign substances and tissues which are hopelessly damaged. The extent of the procedure varies with the type of wound. For example, an incised laceration may differ little in appearance from an operative incision, but blood clots have to be removed and bleeding vessels ligated in order to prevent impaired wound healing. In such a wound little or no removal of tissue would be necessary. Avulsions, crushing injuries and penetrating wounds, particularly high velocity missile injuries are associated with extensive tissue destruction and require meticulous and thorough wound excision. If the nature of the injury type and location of the wound and a working diagnosis have been established beforehand, the depth and extent of tissue damage and degree of contamination can be anticipated.

Actually débridement or excision is an exploratory procedure which usually begins on the surface and progresses into the depths and recesses of the wound. Each type of tissue encountered is carefully assessed as to its viability or extent of damage. Blood clots and foreign bodies are removed and careful hemostasis is achieved. Knowledge of the regional anatomy is necessary to protect important structures, nerves and blood vessels from any possible operative injury. Injured structures may be distorted or blood stained and should be identified in normal tissue and traced to the site of the injury. When there is deep damage in the presence of a small external wound, additional operative exposure is attained by enlarging the original wound along physiologic lines. A decision is made to remove any tissue only after careful evaluation of its viability for there must be no needless sacrifice of tissue. If tissue is devitalized, however it must be

at times irreparable damage to the underlying neurovascular structures may occur. Handling the seriously injured person requires skillful and competent care. Speed and rough handling during transportation may accentuate bleeding or shock and aggravate the injury.

Definitive Care. When the injured person arrives at a location where medical services are available, a rapid survey of the situation must be made by the first physician in attendance. Any delay in the prompt recognition and immediate treatment of shock, hemorrhage or asphyxia may result in a fatality. A history of the injury should be obtained and the patient should be thoroughly examined to determine the extent of the trauma. Further injury to the patient should be prevented by careful handling during all phases of care. It is essential to establish a working diagnosis and assess the general condition of the patient before proceeding with definite treatment of local wounds.

Failure to obtain a history and perform a comprehensive physical examination leads to many errors in total management of the injured patient and his wounds. The past medical history may influence treatment: does the patient have heart disease or diabetes, is there a history of recent or prolonged steroid therapy, are there allergies or known sensitivities to drugs and serum? A history of the nature of the injury is an invaluable aid to the examiner in determining the probable locations and extent of the injuries. The history of the circumstances of the injury as to the exact time, place of the accident and type of first aid gives information which can be used to anticipate the degree of bacterial contamination. Careful appraisal of the information obtained from the history and examination serves as the basis for total evaluation of the patient. Only then can further care be outlined and attention focused upon local wounds.

In many cases operative intervention may be an integral part of resuscitation. The patient who has an obstructed airway may not be relieved by the simpler maneuvers and may require tracheostomy. Operative exploration to arrest continuing internal hemorrhage and closure or stabilization of chest defects to correct severe cardiorespiratory alterations are other examples. If there are multiple injuries, the wounds which are the greatest threat to life should receive priority of treatment. Some examples are those injuries due to local tissue defects which result

in asphyxia, respiratory obstruction or other serious disturbances in the cardiorespiratory function. Others include cases of massive hemorrhage, perforation of intra-abdominal viscera or organs and serious open or closed cerebral injuries.

Treatment of Closed Injuries. Operative intervention may be necessary at varying intervals after occurrence of closed injuries. Most superficial closed injuries, namely contusions are treated by nonoperative surgical measures. For sprains and incomplete muscle tears of the extremities an efficient compression dressing, rest and elevation may be all that are necessary; however, complete ruptures of musculotendinous and ligamentous structures frequently require operative correction as soon as possible. In regions other than extremities, serious damage to underlying organs and vital structures can be obscured initially by the absence of external marks of violence and a paucity or slowness in the development of abnormal physical findings. Repeated examinations at frequent intervals are essential to determine any changes in physical findings which may indicate the necessity for emergency operation. An exploratory laparotomy is a life-saving measure to control internal hemorrhage or repair damaged intra-abdominal viscera. In any location an expanding hematoma has to be controlled to alleviate the harmful effects of pressure against tissues or organs. Established hematomas are frequently aspirated or evacuated surgically for the same reasons. Whenever the blood supply to the skin is jeopardized by uncontrolled swelling and hemorrhage in the underlying tissues, the original closed injury may be converted into an open wound by breakdown of this protective barrier.

Treatment of Open Wounds. The first objective of care in all open injuries is to convert the open contaminated wound into a surgically clean wound, which should be closed as early as circumstances permit. Operative care should not be undertaken prior to the establishment of an accurate working diagnosis and a thorough evaluation of the patient. The selection of anesthesia will depend upon the wound and the condition of the patient. When a wound is known to be superficial and local anesthesia can be employed, the initial wound treatment may be performed in a well equipped dispensary or emergency room using the same aseptic precautions that are carried out in the operating suite. One must remember that any operative procedure is

another form of trauma and tissues must be protected from excessive operative injury. Aseptic technique, gentleness in handling tissues and meticulous hemostasis are essential to any surgical intervention.

The optimal time after injury for the initial care is within the first eight hours which is usually considered to be the time prior to actual bacterial invasion of tissues. This interval is often called the "golden period" however there are many other factors which have to be considered other than an arbitrary limit of time. For example because of an excellent blood supply most wounds of the face can be cleansed and repaired safely as long as twenty four hours after injury. The history of the contamination, the type and location of the wound and the extent of tissue damage will influence the extent and character of the initial treatment.

The adverse effects upon wounds from the presence of devitalized tissue are impaired wound healing and local or invasive infection. Devitalized tissues soon become necrotic and provide an ideal culture medium for bacterial growth. Only living and healthy tissues can effectively combat certain degrees of bacterial contamination. It should be recognized, however that if the number of virulent microorganisms present in any wound is excessive infection may not be avoidable in spite of the inherent ability of living tissue to combat bacteria.

Wound Cleansing. Wound cleansing, using aseptic precautions, is an integral part of the initial treatment. Every open wound is contaminated and frequently foreign material is imbedded in the wound. For extensive wounds preparation of the proposed operative field is performed after the patient has been anesthetized. The surrounding skin is shaved and carefully cleansed with soap or a bland detergent and sterile water while the wound is protected by a sterile dressing. Following thorough cleansing of the skin, the wound is carefully irrigated with sterile saline solution and any loose foreign material is removed. The details of technique may vary with different surgeons, but the procedure is essentially a mechanical process with care being taken to avoid irritation of the skin or trauma to the tissues. For the least serious type of wound at least ten minutes is required. In extensive injuries, more than one change of sterile gloves and materials is required and additional time should be taken to insure the cleanest field possible.

It is necessary to prepare an area large enough to carry out the operative procedure without fear of contamination from unprepared skin surfaces for the original wound may have to be enlarged. For wounds of the extremities the entire circumference is prepared, and in hand injuries the entire wrist and lower forearm are cleansed.

Wound Excision. Wound excision, often called débridement, is the foundation of initial operative care of open wounds and it is most effective if performed as soon as possible after the injury. Its objective is to convert the accidental wound into a surgically clean wound which contains only viable tissues with an adequate blood supply. This goal is accomplished by the removal of devitalized tissues, foreign substances and tissues which are hopelessly damaged. The extent of the procedure varies with the type of wound. For example, an incised laceration may differ little in appearance from an operative incision, but blood clots have to be removed and bleeding vessels ligated in order to prevent impaired wound healing. In such a wound little or no removal of tissue would be necessary. Avulsions, crushing injuries and penetrating wounds, particularly high-velocity missile injuries are associated with extensive tissue destruction and require meticulous and thorough wound excision. If the nature of the injury, type and location of the wound and a working diagnosis have been established beforehand, the depth and extent of tissue damage and degree of contamination can be anticipated.

Actually débridement or excision is an exploratory procedure which usually begins on the surface and progresses into the depths and recesses of the wound. Each type of tissue encountered is carefully assessed as to its viability, or extent of damage. Blood clots and foreign bodies are removed and careful hemostasis is achieved. Knowledge of the regional anatomy is necessary to protect important structures, nerves and blood vessels from any possible operative injury. Injured structures may be distorted or blood stained and should be identified in normal tissue and traced to the site of the injury. When there is deep damage in the presence of a small external wound, additional operative exposure is attained by enlarging the original wound along physiologic lines. A decision is made to remove any tissue only after careful evaluation of its viability, for there must be no needless sacrifice of tissue. If tissue is devitalized, however, it must be

removed, regardless of the resulting defect. The greatest problem may be to appraise correctly tissues which have been injured but remain attached and are not obviously devitalized.

An assessment of the viability of skin may be difficult at times. In most sharp lacerations there is little destruction of skin, but in avulsing and crushing injuries the damage is often extensive. Viable skin bleeds actively from its cut surface and when gentle pressure is applied it blanches temporarily. The rapidity of the return to normal color should be observed. One can compare and test the skin in question with that in locations distant from the site of injury. The flap that does not have a correct color is usually devitalized or destined to become necrotic. One must never save skin flaps that are severely crushed, whose vessels are thrombosed, or which are deprived of an adequate blood supply. Although the color may appear normal at the time of the operation, if venous return is impeded the flap may die. Skin on the scalp and face has an excellent blood supply and rarely requires radical excision. Every effort should be made to save all viable skin on the palmar surface of the hand and the plantar aspects of the feet.

Muscle viability is determined by inspection and testing its properties. Viable muscle is red, its surface bleeds briskly and it contracts when touched or stimulated. Dark, soft muscle suffused with blood is usually devitalized. Massive damage to major muscle groups produces harmful by products which are absorbed into the blood stream and may lead to deep shock. Nonviable muscle is an excellent culture medium for bacterial growth, particularly of anaerobic organisms.

Fascia withstands trauma well and only jagged edges need be excised. Fasciotomy however may be necessary to relieve pressure on underlying tissues within tight fascial compartments.

Tendons should not be excised unless there is extensive fraying or soiling from foreign material.

Nerves have an excellent blood supply and excision should always be minimal, for even a severely contused nerve may retain some function or be repairable.

Major blood vessels should be carefully evaluated before ligation is considered. Repair or anastomosis may be possible. Torn or divided smaller blood vessels in distal

portions of the extremities can usually be ligated without jeopardy to the blood supply of the part.

If excision has been properly performed, further steps toward repair and closure are possible in the majority of wounds sustained in civil life. In situations involving mass casualties or in wartime combat injuries, the initial operative procedure should terminate after excision and correction of life-threatening defects in the wound.

Wound Closure. The goal of initial surgery is to obtain a clean, closed wound which will heal by primary intention but unless a wound has been properly cleansed and excised, primary wound closure will result in impaired healing or infection. With proper excision most civilian injuries can be closed primarily if the time interval or degree of contamination has not been excessive. Healing of the depths can be no better than healing of the surfaces. An example is an avulsing injury of the hand with a skin flap overlying tendon repair. If the flap fails to survive, the tendons soon become exposed and necrosis and infection ensue.

There are many ways to close accidental wounds. Many wounds may be closed by suture alone provided that there has been no great loss of skin. No wound should be closed with undue tension on the tissues. Tension causes strangulation of tissues and necrosis of wound edges eventually occurs. Healing is impaired and a pathway for bacteria is opened to the underlying structures. If the skin does not approximate without tension, undermining the subcutaneous tissues may allow sufficient relaxation to permit proper closure. This is applicable to face and neck wounds and other areas where there is laxity of the tissues. Another method for skin closure is the use of a relaxing incision with undermining to permit local shifting of skin and subcutaneous tissue. More extensive local shifts of adjacent tissues are called rotation flaps.

If the skin defect is too large to be closed by suture alone or local shifts of tissue, a skin graft may be applied. Skin grafts are of two types: the split or partial thickness and the whole-thickness graft. These are called free skin grafts because either type of graft is obtained by detaching it completely from the body surface and transplanting it to another location. The donor area of split grafts contains sufficient epithelial elements to allow spontaneous epithelialization of this newly created wound which is similar to a

superficial abrasion. A whole thickness graft donor site leaves a wound completely devoid of skin and this wound has to be closed. It is possible to close small areas by undermining and suture but a donor defect which is too large may require application of a split thickness graft. The split thickness skin graft is used frequently in primary procedures to cover large defects. Full thickness skin grafts are more difficult to apply and are not as useful in the initial care.

Another method to provide covering tissue is the use of a pedicle flap, which consists of skin and its attached subcutaneous tissue having an adequate circulation. Pedicle flaps are used to cover exposed tendons, nerves, bones and joints when there is no areolar tissue or skin locally which can be shifted to cover these structures. Pedicles are transferred from one part of the body to another with an intact blood supply. The proximal portion of the flap has to remain attached at its site of origin for several weeks until a new blood supply from the recipient area can grow into the transferred tissue. When adequate circulation has developed in the flap from the recipient area it is then detached from its donor site at a secondary operation.

Although every open wound if not grossly infected, should be cleansed and excised, there are many factors which determine the necessity or advisability for deep repair and surface closure. The location and extent of the injury and the condition of the patient as well as the degrees of contamination are basic considerations. The extent to which deep repair and closure can or should be carried out is a matter of both knowledge and judgment. In wounds of the abdomen repair of injured viscera is essential for survival. Open joint capsules are repaired to prevent infection in joints. Sucking wounds of the chest must be closed with muscle flaps even though the skin may be left open for later closure. In brain injuries primary closure of the dura mater and scalp is necessary to avoid infection, herniation and fistula formation. Exposed tendons, nerves and bone have to be covered with muscle or areolar tissue to prevent maceration and eventual necrosis.

In some situations the skin is closed primarily and repair of deeper structures is either minimal or is postponed completely until the wound is well healed. Although this may necessitate a secondary operation at a later date, the chances for optimal recov-

ery of function may depend upon this decision.

The management of wounds sustained in warfare and some civil disasters requires staging of the operative procedures. These wounds are cleansed and excised initially but closure is delayed until it is certain that the danger of wound infection has passed. Exceptions to this policy are wounds of the face and scalp which are closed at the time of the initial procedure. Some hand injuries are partially or completely closed to protect the underlying structures. After débridement the wound is covered by a compression dressing and the injured part is immobilized. Within three to seven days, if the wound reveals no evidence of gross infection and the tissues appear healthy the wound may be closed. This second procedure is called *delayed primary closure* by most military surgeons but is often referred to as *secondary* or *delayed suture*. Reconstruction or reparative surgery is undertaken at a later date when the tissues are healed soundly and the patient's condition is well stabilized.

Healing of all wounds is assisted by rest. In extremity and facial wounds resilient compression dressings help to minimize edema, obliterate dead spaces and provide rest. Injured extremities should be immobilized by splints, well padded plaster molds or bivalved casts. Some degree of swelling is a natural response to injury but if it can be minimized the tissues will heal more kindly. Elevation promotes more adequate lymphatic and venous return from extremities. Swelling may however be accentuated by constrictive dressings to such a degree that the blood supply to a part is compromised. Local pressure necrosis can be avoided by adequate padding over bony prominences.

Prophylaxis against tetanus is an important consideration in every person who has sustained an open injury. A tetanus toxoid booster is given when there is a definite history of previous active immunization. Tetanus antitoxin provides temporary passive immunization but should be given with caution by proper testing for sensitivity. Even then, serious delayed reactions to the serum may occur.

The value of antibiotics is undoubted, but they are not always needed. Their use should be restricted to cases in which specific indications exist, such as gross contamination, extensive tissue destruction or inoculation

of the wound with highly virulent bacteria. Adequate, early wound surgery is the best protection against infection.

READING REFERENCES

- Allen, H. S.: Management of Large Contaminated Wounds. *Arch. Surg.* 65:151, 1952.
- Brown, J. B., and McDowell, F.: *Skin Grafting*. Ed. 8 Philadelphia, J. B. Lippincott Company, 1958.
- Churchill, E. D.: American Surgeon, A.U.S. Surg. Gynec. & Obst. 84:529, 1947.
- Committee on Trauma, American College of Surgeons: Early Care of Acute Soft Tissue Injuries. Chicago, American College of Surgeons, 1954.
- Hampton, O. P., Jr.: Wounds of the Extremities in Military Surgery. St. Louis, C. V. Mosby Company, 1951.
- Koch, S. L.: Injuries of the Parietes and Extremities. *Surg. Gynec. & Obst.* 76:1, 189, 1943.
- Mason, M. L.: Wound Healing. *Internat. Abstr. Surg.* 69:303, 1939.
- Reid, M. R.: Some Considerations of the Problems of Wound Healing. *New England J. Med.* 215:753, 1936.
- United States Department of Defense: Emergency War Surgery. U. S. Armed Forces Issue of NATO Handbook. Washington, Government Printing Office, 1958.
- Welch, C. S., and Powers, S. R., Jr.: *The Essence of Surgery*. Philadelphia, W. B. Saunders Company, 1958.

SHOCK

By ISIDOR S. RAVDIN, M.D.
and JAMES E. ECKENHOFF, M.D.

ISIDOR SCHWARTZ RAVDIN, John Rhea Barton Professor of Surgery at the University of Pennsylvania, is a Hooper by birth. He was educated at Indiana University and the University of Pennsylvania. He has directed the Harrison Laboratory of Surgical Research from which have come many fundamental contributions to surgery. Consultant in Surgery in the Far East during World War II. Dr. Ravidin has played an active role as a member of the Armed Forces Medical Policy Council of the Department of the Defense and has an intimate knowledge of the most recent developments in the field of surgery with which this chapter deals.

JAMES EDWARD ECKENHOFF, of Maryland, was educated at the University of Kentucky and received his medical training at the University of Pennsylvania. Actively interested in problems of research relating to the field of anesthesiology, he serves as Professor of Anesthesiology at the University of Pennsylvania.

Shock is a common clinical entity extensively studied for over a half a century yet the basic mechanisms are still incompletely understood. Shock is usually envisioned as accompanying or following trauma; indeed, a great impetus for its study has been the relatively concentrated mass wounding accompanying war. However, the syndrome is not confined to trauma or to surgery but is frequently seen in conjunction with medical, neurologic and even psychiatric disorders. This implies extensive ramifications of the etiologic processes of shock and casts doubt upon a single exciting cause.

Shock has been variously qualified, e.g., surgical shock, wound shock, hemorrhagic shock, neurogenic shock. Such terms are likely to refer to probable initiating causes. However, the clinical picture is essentially

the same under all of these conditions. Therefore, the only qualifications of shock we shall use will be *impending shock* and *irreversible shock*. The first qualification implies that the patient does not appear to be in shock but does demonstrate signs and symptoms suggesting that shock will occur within a short time unless preventive steps are taken. When the symptoms of shock have persisted sufficiently long to preclude recovery in spite of therapy, irreversible shock has occurred. The period required to produce irreversibility is variable; shock in the patient in poor physical condition may become irreversible within minutes. In a usually healthy but recently injured young adult, irreversible shock may not appear for hours.

The clinical picture of shock is so well

known and widely taught that even the twelve-year-old Boy Scout can describe it reasonably accurately. The patient's skin is pale, cold and moist. Respirations are shallow, rapid and often grunting in character. However, in profound shock, respirations may be only two to three per minute. Occasionally the lips and fingernails are slightly cyanotic. The pulse is weak and rapid. The blood pressure is low. Urine formation is scanty or absent. Thirst is a common feature. Complaints of pain may be absent except when the patient is moved. Apprehension and restlessness are usually displayed. The sensorium may be clouded.

ETIOLOGY

Some exciting factors of shock are fairly clear. The most obvious and most common is acute hemorrhage or protracted fluid loss. The early mechanisms of shock following trauma without blood loss, neurogenic shock coming on after cranial injury, cardiogenic shock preceded by myocardial infarction or shock occurring from toxicity or poisoning are not clearly understood. Least well explained is why shock develops into irreversibility.

Originally attempts were made to implicate a single factor in the development of irreversible shock. Early theories suggested that the major defect was inadequate cardiac output, prolonged maintenance of hypotension because of regional vasodilatation, a reduced blood volume due to leakage of fluid from the blood vessels, or vascular depression produced by toxic material released from wounds. The consensus today appears to be that no one factor predominates but that many things contribute to produce circulatory insufficiency following which irreversibility occurs.

Freeman has presented this chain of

events diagrammatically as shown in Figure 1.

The circulatory response to stress should be understood for orientation in a discussion of the etiology of shock. Table 1 is a modified version of Bazett's calculation of the distribution of the circulation in man and the uptake of oxygen by organ systems. It is apparent that, under normal conditions, the major demand for blood flow and oxygen uptake is by vital organs (brain, heart and hepatic portal system) yet these represent only 7 per cent of the total body weight. On the other hand the principal mass of the body (skeletal muscle, connective tissue, bone and skin) representing 93 per cent of the total weight, receives only 30 per cent of the cardiac output and 40 per cent of the oxygen intake (Table 2).

Under conditions of stress, widespread changes occur in blood volume distribution. With muscular exercise, blood flow to muscle increases as does oxygen consumption. Such changes are met by diminution in flow to other tissues, notably the kidney. These are normal reflex mechanisms.

Following hemorrhage, blood flow to vital organs is maintained at the expense of the principal mass of body tissue. Initially blood pressure is supported by generalized peripheral vasoconstriction which simultaneously diverts blood from the peripheral circulatory beds (e.g., muscle, skin) to vital organs. Kidney blood flow diminishes most sharply and with the slightest stress—even with emotion. As blood volume is reduced by hemorrhage, blood flow through nonvital tissue is gradually reduced by increasing vasoconstriction until finally a peak of compensation may be reached when the functional circulation consists solely of blood flow to and from the brain, heart and liver. Evidence suggests that finally even the liver may be shunted

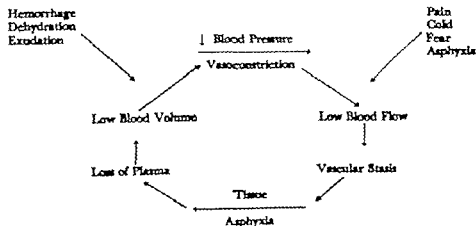


Figure 1 Diagrammatic representation of chain of events contributing to the production of inadequate circulation following which irreversible shock can occur

Table 1 Regional Blood Flow and Oxygen Consumption

REGION	WEIGHT (KG)	PERCENTAGE OF TOTAL WT	ORGAN BLOOD FLOW (CC./MIN.)	CARDIAC OUTPUT (%)	ORGAN OXYGEN UPTAKE (CC./MIN.)	TOTAL OXYGEN UPTAKE (%)
Brain	1.4	2.2	750	13.9	46.2	22.9
Heart	0.3	0.5	255	4.7	29.1	11.6
Hepatic-portal	2.6	4.1	1500	27.8	51.0	20.4
Kidney	0.3	0.5	1260	23.3	17.6	5.0
Skeletal muscle	31.0	47.2	840	15.6	49.6	20.0
Skin	3.6	5.7	460	8.4	10.9	4.8
Residual tissue	23.8	37.8	380	6.4	50.0	15.3

out of the circulation and all blood flow from the inferior vena cava ceases yet with flow in the superior vena cava persisting. This implies that under these conditions the brain continues to be supplied with blood.

The common denominator of irreversible shock is a reduced effective blood volume. In hemorrhage the factor responsible for the reduction is clear but when major hemorrhage is absent the cause of hypovolemia is obscure. While the total amount of blood within the body may be unchanged, the volume circulating can be reduced by leakage or sequestration—thus the effective blood volume becomes inadequate.

Formerly it was believed that, in the absence of significant hemorrhage blood volume was reduced by "leakage" of fluid from blood vessels into tissue. Blalock and his associates were the first to prove that leakage need not occur for hypovolemia to appear in shock. More recent evidence indicates that blood may become trapped within peripheral blood vessels or liver and thus become "lost" to the circulation so far as the prime purpose of circulating blood is concerned—transportation of oxygen foodstuffs and metabolic products. The first sign of slowing blood flow and impending shock at operation may be increasing venous oxygen desaturation. This may occur despite a satisfactorily maintained blood pressure or pulse and without evidence of reduced arterial oxygen concentration. It signifies that blood is passing through

capillaries so slowly that a greater than normal amount of oxygen is being withdrawn from it. It also suggests marked arteriolar compensation in order to maintain blood pressure.

Zweifach and his associates have demonstrated the activity of precapillary arterioles, capillaries and venules in the rat mesentery during normal conditions and in hemorrhagic shock. Normally all capillaries do not function simultaneously but with stress every channel can open, tremendously increasing the capacity for blood. As blood flow slows these vessels become engorged with blood, flow finally ceases and in some areas the capillary bed is bypassed trapping or "sequestering" significant quantities of blood. This removes blood contained within the circulation from the "effective" blood volume. It must be remembered however that the applicability of these observations to all capillary beds has not been proved.

There are no studies to indicate even the approximate amount of blood that can be contained within vascular beds whose tone and reactivity have been abolished. Plethysmographic studies have indicated that simple warming of the legs can increase the volume of blood within these parts by 500 cc. or more. Investigations upon wounded soldiers in shock have demonstrated the need for infusion of vast quantities of blood in order to maintain blood pressure. In Korea, it was not uncommon to administer

Table 2 Comparison of Regional Blood Flow and Oxygen Consumption to Vital Organs and Other Tissue

REGION	WEIGHT (KG)	PERCENTAGE OF TOTAL WT	ORGAN BLOOD FLOW (CC./MIN.)	CARDIAC OUTPUT (%)	ORGAN OXYGEN UPTAKE (CC./MIN.)	TOTAL OXYGEN UPTAKE (%)
Vital organs (excluding kidney)	4.3	6.8	2506	46.4	126.3	54.9
Other tissue	58.4	92.7	1680	30.4	110.5	40.1

twice the patients normal blood volume even though there was no longer evidence of active bleeding. Attempts to locate large volumes of sequestered blood by means of chromium-tagged red cells or by means of the Evans blue dye technique have failed, but the evidence is far from conclusive. Chromium tagged red cells may be rapidly destroyed. Also the tagged cells or dye may be unable to reach the sequestered blood, thus preventing complete dilution. Evidence in favor of sequestering was obtained from postmortem studies of men who died in shock in spite of massive transfusion. These studies revealed skeletal muscle, liver and lungs choked with blood.

Obviously the factors mentioned all work in the same direction, namely production of an inadequate circulation. The lowered blood volume leads to decreased cardiac output. Sequestered blood and diminished capillary blood flow lower venous pressure and diminish venous return which likewise contributes to a reduced cardiac output. With prolonged hypotension, Wiggers found that the myocardium itself may be damaged and, even though the blood volume be restored, the myocardium is unable to recover. Myocardial failure then relentlessly progresses to death.

There are other factors which play a part in the progressive decline of the circulation, but their exact role has not been defined. Several lines of investigation have been followed, some of which are challenging. These include

The balance between VEM (vasoexcitor material) and VDM (vasodepressor material) Shorr and his coworkers have demonstrated the existence of a vasoexcitor principle elaborated by the kidney and appearing in the blood stream following renal hypoxia. VEM seems to be integrated with adrenal cortical and sympathetic nervous system activity. It appears following hemorrhage or in impending shock. During this phase, VDM identified as ferritin and formed in the liver also appears, but under aerobic conditions the liver transforms it into an inactive form. With deterioration of the hepatic blood supply oxygen lack occurs, the transformation of VDM fails and it appears in the systemic circulation. Experimentally in irreversible shock, VDM has been detected in large quantities. However blood samples withdrawn from Korean casualties in shock have failed consistently to yield ferritin in significant quantities.

Bacterial factor Fine and his associates

have observed that in dogs treated with antibiotics prior to hemorrhagic shock, the survival rate was higher than in untreated shocked dogs. They have postulated that, during shock, bacteria flourish and by either direct action or by toxic metabolites produce circulatory changes leading to irreversible shock. Antibiotics minimize and prevent this action. The antibiotics are more effective orally than parenterally presumably because of the influence upon the intestinal bacterial flora. Likewise, some antibiotics are more effective upon organisms of the intestinal flora than are others.

There is evidence that the ability of antibiotics to prevent irreversible shock may be principally a species effect. The tissue of many animals (rat, cat, dog) are not sterile, whereas those of man, except for the gastrointestinal tract, are. Factors preventing the growth of bacteria already located in tissue may not be significant when applied to species whose tissues are not contaminated. Nevertheless the bacterial factor may be important in certain types of shock in man, such as that accompanying peritonitis, burns and massive tissue destruction. It seems more difficult to implicate the factor in cardiocirculatory or neurogenic shock.

Autonomic blocking agents Evidence has accumulated to show that certain of the autonomic blocking agents are of value in preventing or retarding the onset of irreversible shock in animals. Among the foremost of these agents are chlorpromazine, Dibenzylamine and hexamethonium. Atropine has generally been ineffective. All of these drugs are protective if given prior to exposure to traumatic (drum) or hemorrhagic shock but are ineffective if administered after trauma has been inflicted or shock has developed. The theory of the use of autonomic blocking agents is based on the fact that during shock vasoconstriction occurs. Because of this, capillary circulation is reduced, tissue perfusion becomes inadequate, anoxic damage occurs, vessel reactivity is lost and, finally blood is sequestered within capillaries. If vasoconstriction could be minimized or prevented by the use of autonomic blocking agents, tissue perfusion could be maintained even though arterial pressure is lowered.

So long as the blood volume is maintained within reasonable limits, the theory is feasible. It is common to see patients with lowered blood pressure without vasoconstriction who experience little difficulty. The recent use of hypotensive anesthetic tech-

niques has even popularized this entity. However, if vasodilatation occurs in the patient who has lost considerable blood without replacement, the circulatory beds in skin and muscle are kept patent at the expense of the circulation in the vital organs. As a consequence, cardiac, cerebral, hepatic and renal complications may develop earlier and with more disastrous results.

The influence of metabolites. In the early phases of shock, metabolites do not appear to play a part in the development of irreversibility. The metabolic derangements are principally those which follow progressive tissue anoxia. As irreversibility develops however the accumulation of metabolites may assume a more important role. An interesting field of investigation in this regard concerns the importance of rising blood ammonia levels. Experimentally it has been demonstrated there is an increased production of ammonia in the kidneys and intestines in shock. The liver loses its ability to utilize the ammonia to synthesize urea, presumably because of hypoxic parenchymal damage. As a result, blood ammonia rises significantly. Efforts are being made at present to evaluate the significance of this observation.

Role of the adrenal cortex. The liberation of adrenal cortical steroids is one of the mechanisms by which homeostasis is maintained. In the absence of the adrenal cortex or when adrenal cortical reserves are limited the response of the vascular system to stress such as shock may be minimal. Under such conditions the administration of adrenal cortical steroids may replenish the depleted stores and restore the reactivity of blood vessels to norepinephrine and possibly epinephrine. The importance of this factor has not been completely evaluated. In the usually healthy individual who is suddenly wounded and develops shock, the adrenal cortical response has usually been found to be normal. On the other hand, adequate data have been obtained from the chronically ill individual to indicate a diminished adrenal corticosteroid level. In these people the circulatory response to shock is often poor but may be improved by the intravenous injection of corticosteroids.

It is apparent that considerable recent interest has been directed toward the liver and its relationship to irreversible shock. While the liver may not be of much importance in the early phases of shock, as hypotension, hypovolemia and hypoxia become manifest significant metabolic alterations

occur as a result of these influences upon the liver. These metabolic changes may be the principal factor determining irreversibility. There is experimental evidence that death from shock can be prevented by circulation of oxygenated blood through the liver.

One might summarize the factors underlying shock by saying that circulatory homeostasis is dependent upon many and varied factors: (1) the pump (heart) must be adequate; (2) the blood vessels must be patent and reactive; (3) the lungs must be functional and oxygen supply sufficient; (4) there must be sufficient blood to circulate; (5) the center controlling nervous impulses (brain) must be performing satisfactorily; (6) the addition of vital substances to the blood stream must be optimal, and the mechanisms to alter metabolic products and to eliminate waste substances must be functioning satisfactorily. Interference with the aforementioned system at any point reduces the ability of the body to withstand shock and hastens the onset of irreversibility. Some factors are more important than others. For instance, the patient with a sudden myocardial infarction suffers acute injury to the pump and as a consequence may go into shock rapidly. Other factors may be of less immediate importance but may ultimately lead to shock.

THE TREATMENT OF SHOCK

Prevention. Obviously the most important factor in the treatment of shock is its prevention. This is not always possible in shock that follows sudden myocardial infarction or that appearing after cranial injury. However much can be done to prevent shock accompanying trauma, operations, infections and burns.

The idea of anticipating the possibilities of wound shock in soldiers by the routine preventive use of chlorpromazine is intriguing. If chlorpromazine had no undesirable side effects this use might be feasible. However the drug is a depressant in its own right and a decrease in mental acuity in all soldiers might counterbalance the possibilities of protection from shock in a few. Nevertheless in civilian surgical practice, the use of chlorpromazine preoperatively in patients in whom significant blood loss is expected may be worth while. The value of such protection awaits further study.

The movement of wounded, acutely ill, unconscious or anesthetized patients must be accomplished with great care. If shock is

apparent, it is better to postpone movement than to jeopardize the patient's life. Unnecessary or rough movement may produce sudden, severe hypotension or even cardiac arrest under these circumstances. The same caution should apply to movement of any unconscious or anesthetized patient. Hypotension is often seen following transferral of these patients from operating table to litter or bed. When movement is accomplished, it should be after broken bones have been splinted or properly supported. Pain, however, is not the sole cause of hypotension, as is evident from the fact that the blood pressure of anesthetized patients falls with movement. The answer appears to lie in loss of vasomotor control.

Patients with trauma may not require opiates for comfort. A relatively small proportion of men severely wounded in combat complain of pain, and pain should be the indication for the use of narcotics. These agents depress the reflex control of the circulation. In shock, impending shock or situations in which surgical shock may occur, circulatory reflexes should be functioning maximally. When opiates must be used for pain in shock, they should be injected intravenously in small doses. Drugs injected subcutaneously or intramuscularly may not be absorbed by the blood stream because of the inadequate circulation.

The principal means of preventing shock is to keep the blood volume within normal limits. All patients who have been injured and in whom shock may occur, those undergoing major surgery and those first seen in shock, should have intravenous infusions. Great care and foresight must be used in the placement of intravenous needles or cannulae. These must be of a size adequate to allow infusion of large quantities of blood within short periods. Effort must not be wasted in persistent attempts at venipuncture in patients in impending shock. The vein should be exposed surgically and cannulated under direct vision. Both surgeons and anesthesiologists should be facile in performing these operations. The largest stores of blood or plasma expanders are of little consequence unless the means for getting the material into the patient are available. In Korea, it was necessary to infuse as much as 4500 cc. of blood within a thirty-five-minute period in order to keep alive a patient in shock. In severe shock or when major hemorrhage is expected, more than one intravenous route should be provided so that

the required amount of blood can be administered.

Surgeons and anesthesiologists should become more skilled in estimating blood loss. In major surgical procedures associated with considerable blood loss, the "dry sponge" technique should be used. In this method, the dry weight of the sponge is subtracted from the blood-soaked weight, the difference in grams approximating the amount of blood in cubic centimeters contained in the sponge. While not completely accurate, it can be used as a suitable index for blood replacement.

Careful preparation of patients for operation will minimize the occurrence of shock in the operating room. Blood volume studies are useful, particularly in the elderly sedentary or chronically ill patient who is to have a major operative procedure. Often these groups will be found to have significant hypovolemia. Transfusion prior to operation under these circumstances may prevent or minimize the onset of shock.

One cannot be dogmatic about the amount of blood loss tolerated without need for replacement. The healthy adult can lose 500 cc. to 800 cc. of blood without need for replacement. The elderly or acutely ill patient may not tolerate such loss. The need for blood replacement is usually indicated by the appearance of hypotension with tachycardia. However, neither of these symptoms need appear. In general, if the loss exceeds 500 cc., replacement is indicated either with a plasma expander or with whole blood. If the loss exceeds 1000 cc., whole blood replacement of at least 500 cc. is probably indicated. It is not the province of this discussion to consider the indications for plasma expanders versus blood. The patient in shock needs blood and there may be little usefulness for plasma expanders. In the maintenance of blood volume by replacement of minor blood loss, however, they have value. Constant watchfulness by the surgeon and anesthesiologist with particular attention to fluid replacement will do much to prevent surgical or hemorrhagic shock.

Shock may be averted following sudden hemorrhage by elevating the patient's legs. The amount of blood estimated to be returned to the systemic circulation by this maneuver may equal or exceed 500 cc. However, if vasoconstriction has previously existed to a significant degree, little blood may be contained in the extremities and elevation of the legs, therefore, may be of little

value. It has generally been assumed that the optimal position for the patient with hypotension or impending shock is a slightly head-down position. This facilitates venous return and may improve circulation to the brain. Data to support this belief are lacking.

Conversely, caution should be exercised when after a patient has been operated upon in the lithotomy position, the legs are put down. Often this is accompanied by a precipitous fall in blood pressure due to filling of peripheral vascular channels. This can be minimized by wrapping the legs with elastic bandages prior to lowering the legs.

Hypoxic damage may be averted or minimized in impending shock if the oxygen supplies to the body are increased. This can be accomplished by increasing the oxygen concentration of the inspired gas either by means of a nasal catheter or by a mask. The former method, with the catheter at the tip of the epiglottis and with an oxygen flow of 6 liters per minute, will insure an oxygen concentration in the inspired air of about 40 to 45 per cent. The latter method, if properly applied, will supply 90 to 100 per cent oxygen. The circulation time becomes slower as the blood volume is reduced. The closer a patient approaches a shock state, the more completely his blood is desaturated of oxygen following its passage through the capillaries. Therefore, the greater the oxygen concentration in the arterial blood the more is available to tissues. All hypotensive patients, whether in shock or not, should be given oxygen for this reason.

In patients who have had chronic illnesses or those who have shown signs of adrenal insufficiency and in whom major surgery is contemplated, adrenal cortical extracts may be of value for preoperative preparation. This may improve the response of the circulation to endogenous *l*-norepinephrine or epinephrine and to stress in general. It may also prevent the appearance of shock.

Careful anesthetic management will minimize the occurrence of shock. Prolonged deep planes of anesthesia predispose to shock, particularly if anesthesia is accompanied by rough surgical manipulation and fluid loss. General anesthetics are myocardial depressants; the degree of depression being directly proportional to the concentration of anesthetic. Diethyl ether and cyclopropane cause a liberation of norepinephrine in the body thus preventing the appearance of the depressant effect. If catechol amine stores are exhausted, myocardial depression may be more apparent, depending upon the

depth of anesthesia. The depth of anesthesia should never exceed that needed to produce satisfactory operating conditions.

Treatment of Shock. The principles of the immediate treatment of existing shock center around a few essentials. These are:

Intravenous administration of blood. This should be given rapidly and continuously until the blood pressure returns within normal limits and the symptoms of shock abate. There is considerable disagreement concerning the value of intra-arterial versus intravenous transfusions of blood. The proponents of intra-arterial transfusion argue that blood is placed in the best position to enhance coronary and cerebral blood flow and in general to raise arterial blood pressure. In practice, however, one should realize that not more than 100 cc. of blood per minute can be pumped through an intra-arterial cannula whereas the heart ejects up to 5000 cc. per minute. In shock, cardiac output and venous return are low. Therefore there may be greater advantages in infusing large volumes of blood into the veins.

In usually healthy adults in shock there is little danger of overtransfusion and production of pulmonary edema. However in the elderly or chronically ill patient, the possibility is greater. In general, if the blood pressure remains low it is safe to infuse blood at a rapid rate until the blood pressure has returned to physiologic levels. The infusion should then be continued at a slower rate.

Plasma expanders should be used in the absence of blood or while blood is being crossmatched. However it is unlikely that shock can be satisfactorily treated without blood.

Oxygen. A high concentration of oxygen should be administered. As mentioned previously a nasal catheter with a flow of 6 liters of oxygen per minute will produce a 40 to 45 per cent concentration in the inspired air. A bag and mask, if properly arranged with a high flow rate will insure nearly 100 per cent. The latter concentration is desirable in treating shock, although the mask is more uncomfortable to the patient.

Position. A slightly head-down position is probably most beneficial to the patient. If the tilt is too great, the patient's respiration may be compromised by the abdominal organs pushing the diaphragm upward, reducing tidal exchange.

It is not necessary nor is it wise to apply heat to the patient's body during treatment. This dilates cutaneous blood vessels and may

interfere with maintenance of essential circulation. As blood volume is replaced surface warming can be accomplished gradually. Hypothermia has been advocated for the treatment of patients in shock. At present, there are few data to support this contention. Published work suggests that hypothermia alone is of no greater benefit than is chlorpromazine alone.

Vasopressor drugs Previously it was believed that vasopressor drugs had no place in the therapy of shock. There is evidence now however that pressor drugs may be of value as a temporary expedient combined with the simultaneous administration of blood. The theory of the use of vasopressors is to produce vascular constriction to accommodate the reduced blood volume. As the blood volume is increased, the need for pressor drug should be reduced.

The ideal drugs for this use would be those with maximal peripheral constrictor effect and minimal cardiac or cerebral stimulant action. The most potent agent meeting these requirements is *l*-norepinephrine. This drug is a powerful peripheral vasoconstrictor which reduces the size of the peripheral circulatory bed, raises the blood pressure and facilitates venous return. It also has a myocardial stimulant effect. *l*-Norepinephrine may produce an effect when all other drugs have failed. Vasopressor therapy is not a substitute for blood replacement in most patients in shock, but it should be considered a stopgap measure to be used along with blood. Attempts should be made to withdraw support by pressor drugs as soon as a reasonable blood pressure can be maintained. Patients in shock of myocardial origin may require vasopressor therapy without transfusion.

The usual method for administering *l*-nor epinephrine is to dilute 4 mg. of the drug in 1 liter of saline or glucose. This concentration can be increased at will. Its effect is manifest within seconds after beginning the intravenous infusion and disappears within minutes of termination. The rate of flow must be carefully regulated to keep the blood pressure at the desired level. One must be certain that the injection is intravenous and not paravenous, since the latter may result in tissue slough. This appears most commonly after injection into the veins of the lower limbs, but it has been observed in the arms. It can be prevented by the addition of phenitolamide to the infusion. One should never inject *l*-norepinephrine intra arterially.

When is the patient who is in shock or who has been in shock ready for operation? There are no definitive tests to suggest the patient's ability to withstand operation after he has been in shock or while he is in shock. A recently advocated test is one wherein the patient thought to be ready for operation is tilted in the head up position for five minutes. If the blood pressure remains stable, the patient is more likely to withstand operation well. However if the patient becomes hypotensive with the tilt, further preparation is necessary.

Another suggested test is to inject intravenously a small amount of epinephrine or *l*-norepinephrine and observe the response of the patient's blood pressure. If there is no elevation in blood pressure, the patient is probably not ready for operation. If his blood pressure rises satisfactorily he should withstand operation.

What are the problems of management of these patients during operation? A severely wounded patient or a patient in shock or impending shock, anesthetized by an inadequately trained technician and operated upon by a surgeon of limited experience has a greater chance of dying during operation than would the same individual in the hands of an experienced anesthesiologist and surgeon. Too often the problems of anesthesia and surgery for patients in shock are learned by sad experience rather than from firsthand instruction from physicians who have already learned the fundamentals of treating patients in shock. This has been particularly true in times of war.

The problems of management can be divided into those pertaining to anesthesia and those relating to surgery. For the former the following facts are of especial significance. Narcotics should be omitted preanesthetically since they may predispose to further hypotension. There are abundant data showing that narcotics cause respiratory and circulatory depression. Under conditions of stress it would be desirable to avoid such depression and to have the circulatory system at peak reactivity. There is little need for narcotics and the hazards following their use are great.

An anesthetist must be aware that minimal amounts of anesthetic agents are required for patients in shock. In this condition, the circulation to the major portion of the body is absent or minimal. Therefore the brain and other vital organs receive most of the administered anesthetic agents. It is apparent why an overdose of anesthetic

agent readily occurs. Patients who have been in shock, or are in shock, may not tolerate any anesthetic. Occasionally even the mild est and most expertly administered anesthetic will convert shock to irreversible shock or death. In our opinion cyclopropane is probably the anesthetic agent of choice for patients in shock. Recent work suggests that ether is to be avoided. Thiopental has likewise been incriminated for its depressant qualities in the severely ill or wounded. Muscle relaxants are rarely needed in the shocked patient. Whatever agent or method used, ventilation should be adequate, oxygen concentration kept high, and hypercapnia prevented.

Frequently the first sign of worsening of a shocked patient's condition during anesthesia and surgery is the observation that decreasing quantities of anesthetic agents are required to keep the patient anesthetized. Failure of the anesthesiologist to realize this fact or to apprehend the sign will lead to a delay in the institution of treatment and the demise of the patient.

From the surgical viewpoint, it must be pointed out that speed is essential in performing operations upon patients in shock. Definitive surgery must be deferred until the patient's condition improves. The mortality rate associated with prolonged surgery in these situations will always remain high. The surgeon must be gentle with his manipulations. Roughness combined with the mandatory light plane of anesthesia may give rise to disastrous traction reflexes disastrous in the sense that further hypotension or sudden cardiac arrest may occur or in that it may become necessary to maintain a deeper plane of anesthesia to avoid reflex activity. For abdominal operations, minimal retraction should be employed, since that too may impede venous return to the heart.

Careful observation of patients in shock must be continued into the immediate postoperative period. As mentioned previously, movement of patients in shock or under anesthesia must be accomplished with caution and gentleness. Sudden turning or shifting of position can result in further hypo-

tension deepening of shock and death. Oxygen should be administered constantly. Norepinephrine may have to be administered continuously for hours or even days. Electrolyte studies must be made in order to detect and treat deficiency or excess of any particular ion. Narcotics and sedatives must be used sparingly. Usually these patients are more restless from hypotension and relative hypoxia than from pain. Consequently small doses of barbiturates may be more efficacious if sedation must be resorted to.

All patients who have received multiple transfusions must be watched carefully for continued bleeding at the end of the operative period or in the postoperative period. The cause for such oozing is not completely understood. This is discussed in detail elsewhere in this text. At present, the treatment normally employed for such excessive oozing is to administer freshly drawn blood or fresh frozen plasma.

Much investigative work concerning shock is in progress. The current thinking on the subject may well be outdated in a matter of months. Excellent symposia upon the subject have appeared and will continue to appear.

READING REFERENCES

- Bard, P., ed.: *Medical Physiology*. St. Louis, C. V. Mosby Company 1958.
- Dripps, R. D., Eckenhoff, J. E., and Vandam, L. D.: *Introduction to Anesthesia*, Philadelphia, W. B. Saunders Company 1957.
- Enklne, J. M.: *The Relation of the Liver to Shock*. *Internat. Abstr. Surg.* 106:207 1958.
- Price, H. L., Linde, H. W., Jones, R. E., and Black, G. W.: *Sympatho-adrenal Responses to General Anesthesia in Man*. *Anesthesiology*. (In press.)
- State, D., and Lichtenstein, I.: *A Study of the Genesis of Shock Associated with Experimentally Induced Hepatic Necrosis in Dogs*. *Surgery* 39:12, 1956.
- Symposium on Shock. *Army Medical Service Graduate School*, 1951.
- Symposium on the Shock Syndrome. *Ann. New York Acad. Sc.* 53:345-542, 1952.
- Wiggers, C. J.: *Myocardial Depression in Shock*. *Am. Heart J.*, 33:633 1947.
- Zucker, G.: *Use of Phentolamine to Prevent Necrosis due to Levartetanol*. *J.A.M.A.* 163:1477 1957.

PRINCIPLES OF PREOPERATIVE AND POSTOPERATIVE CARE

Preoperative, Operative and Postoperative Care

By RICHARD L. VARCO M.D.

RICHARD LYNN VARCO, born in Montana, was educated at the University of Minnesota, and has had teaching and research experience in the Departments of Medicine, Physiology and Surgery in his training for surgery. He is now Professor of Surgery. In their investigative work, he and his co-laborators exemplify the rewards to the patient which can result from practical application of the principles of the basic sciences to the art of the practice of surgery.

INTRODUCTION

Medical students often freely concede that the numerous, at times intricate and perhaps tedious problems of preoperative and postoperative care fail to capture their interest quite as readily as do the more glamorous operative aspects of surgical practice. With experience, however, they are more willing to acknowledge that efforts devoted to these less dramatic activities are necessary to ensure a smooth or even successful convalescence. In fact, during recent years, the steady decline in the mortality and morbidity rates

for surgical procedures has depended in a fair measure on this wider recognition and practice of the principles of preoperative and postoperative care. Besides making any procedure safer this knowledge has eased the restrictions against operating on both elderly persons and the very young. Now these patients need rarely be refused the most formidable type of operation solely because of age. Finally through careful attention to these details, it has become possible to broaden, generously without incurring an unreasonable hazard, the magnitude of sur-

gery undertaken to meet the most complex therapeutic problems

PREOPERATIVE CARE

An orderly plan for the questioning and examining of each surgical patient, even in an emergency case, is of fundamental importance. In an emergency situation two major considerations deserve primary attention. First the patient's ventilation should be adequate. The airway should be patent and the chest wall and lung function acceptable for the maintenance of respiration. Ideally problems related to this region are solved with the cooperation of an anesthesiologist equipped to insert an endotracheal tube, suction the air passages of any blood mucus, gastrointestinal contents or foreign material and provide to the lungs a free flow of an oxygen-enriched mixture. As well he can temporarily stabilize a flail chest wall and thereby overcome any respiratory paradox which had made voluntary respiration inefficient.

The second major consideration is the need for controlling persistent external bleeding. Internal hemorrhage of traumatic origin usually requires an operating room and the paraphernalia available there to insure its successful management. For visible blood loss, firm compression will usually staunch the flow even when this is partly due to brisk arterial bleeding.

When adequate ventilation is established and means to maintain it, as well as control of external hemorrhage, blood should be secured for crossmatching and urine for analysis, the patient being catheterized if necessary.

Preoperative Routine. The student should learn to listen well in order to secure a maximum of information from the patient. To avoid the omission of pertinent details, many institutions provide outline guides for the history taking and the physical examination. The student's methodical use of these forms is recommended until repetition and experience have stamped their essentials into his memory. Such discipline will contribute to the avoidance of grave and occasionally embarrassing oversights. A lengthy transcription of the history and a detailed description of the physical findings may try the student's patience until he realizes how that background becomes an important preamble to the intelligent care of each individual. After the patient's historical and physical data have been recorded, the following laboratory tests and roentgen-ray studies should regularly be obtained:

Analysis of the urine specific gravity
albumin sugar acetone or diacetic acid and
microscopic sediment

Blood study hemoglobin estimation and/or red blood cell count, total white blood cell and differential counts measurements of the bleeding* and clotting times, typing of the patient's blood and prothrombin time, particularly when the history suggests the possibility of liver damage

X-rays Chest roentgenogram on 14-inch by 17 inch film. The surgeon should review all x-ray studies which he has ordered. Through such day-to-day comparison of the patient's clinical status with the salient features of the roentgenograms, he develops a keen ability to analyze even the more complicated problems.

A careful appraisal and correlation of the history physical findings and laboratory data will serve as a guide to initiating care for the usual patient. In many surgical patients, it will represent an adequate preoperative work-up. Yet, the pathologic-physiologic alterations of starvation, dehydration, anemia, diabetes and cardiovascular renal or pulmonary disease may be recognized in others. In some, the situation may prove to be so intricate that a variety of complex laboratory roentgenographic and diagnostic procedures are required. Each patient's needs will have to be decided upon and these will sometimes require consultation to determine the best use of the more complicated and costly diagnostic tools. It is advantageous whenever an excretory cholecystogram or urogram is likely to be included in the diagnostic work-up along with roentgen studies using barium as the contrast agent to schedule the former first so as to avoid difficulty in interpretation by the residual blobs of opaque. For all laboratory tests forethought should be given as to their relevance, cost and urgency. Abuse of an emergency service by requests which are not genuine but rather are a substitute for reasonable anticipation will dull even the most cooperative technologist's ardor for service.

Nutrition Oral preparation The person who has been eating well and has lost little or no weight will require no special preoperative dietary management. On the con-

During the menstrual phase in some women and frequently in patients with cyanotic congenital heart disease markedly abnormal bleeding times are encountered. This need not preclude an emergency operation or a carefully evaluated elective one since with special attention to hemostasis the risk remains reasonable.

trary the patient who has sustained a recent serious weight loss is malnourished i.e., represents a substandard operative risk. Major operations performed on a group of these individuals will carry increased mortality and morbidity rates. This has been repeatedly demonstrated by many surgeons through sad personal experience. Therefore, surgical benefits are not infrequently denied the malnourished patient. If he is accepted without special nutritional rehabilitation after appraisal and acknowledgment of the increased hazards, the operative manipulations are often poorly tolerated. The patient may react unfavorably from the outset, with an instability of blood pressure out of proportion to the actual blood loss. Numerous transfusions at the time of operation seem to provide only transient support to the blood pressure. Convalescence is slower than normal and is disturbed by a greater incidence of complications such as ileus, impaired wound healing and disruptions, or stomal obstruction. This patient already enervated by starvation, lacks the vitality to overcome such hazards and the surgical risks are greater than for a properly nourished subject.

Those bodily derangements produced by severe starvation are hepatic dysfunction, impaired wound healing, hypoproteinemia and a contracted blood volume. These pathologic changes are reversible when appropriate dietary management is possible. The return to normal is slow and it requires many days or weeks of therapy to achieve recognizable nutritional restitution. Sudden and dramatic benefits are rarely obtained. The clinical problem is also complicated by a dearth of simple, accurate methods for precisely measuring the effects of starvation. Likewise, there is no test to indicate the quantity of nourishment required for the re-establishment of sufficient health to allow major surgery without excessive risk. Fortunately the restoration of the bodily economy to less than ideal performance suffices. After an adequate but partial nutritional restoration of the patient the surgeon can operate and anticipate a convalescence as free from complications as in a standard risk individual.

During periods of relative starvation, the patient becomes autocannibalistic. His energy requirements are derived principally from body protein or fat and he subsists on a low calorie, low protein, high fat diet. Logic suggests and experience confirms, the characteristics of a diet most likely to cor-

rect the consequences of such starvation. In the laboratory and from clinical studies, it has been found that this diet should be rich in protein and carbohydrate, high in calories and low in fat content. Of all these constituents, protein plays a leading role. The principal sources of protein in natural foods are meat, fish, eggs, certain dairy products and cereals. Yet in each of these, the protein contributes but a small fraction of the total bulk. It becomes necessary, therefore, to give an enormous quantity of food when a large protein intake is desired. To achieve a protein intake of 300 gm. when beef is the source, about 1400 gm. of lean meat would have to be eaten. This gargantuan feat is usually beyond the capacity of a patient who is weakened and anorectic from prolonged starvation. However reasonable quantities of tastily prepared and attractively served foods will foster the acceptance of a relatively high protein diet. Too the stimulus of a freshly devised daily menu and the cooperative appeal from bedside consultations among the surgeon, dietitian and nursing staff will add vitality to this nutritional program. In analyzing the effectiveness of this regimen, reasonably accurate calculations of the caloric intake can only be made from reappraising those quantities which the patient does not eat. An erroneous judgment is almost certain if reliance is placed on equating a prescription for a high protein and caloric diet with its consumption. Certainly every patient who willingly obtains the necessary quota from the available hospital fare should be encouraged to continue. Physical factors will, however tend to limit the accomplishments possible by this means. In such cases liquid feedings will provide a useful means of supplementing the protein intake. Milk is an excellent vehicle for such mixtures and will hold substantial quantities of protein concentrate and carbohydrate. A wide variety of protein concentrates or digests are available. Skimmed milk powder about 38 per cent protein is among these. It is inexpensive, is widely available, stores well in bulk form and can be disguised to enhance its palatability. Its proteins are complete and possess a better than average capacity to induce plasma protein regeneration.

The use of a liquid diet to supplement the hospital fare, or as the sole source of calories, is valuable and finds frequent application in patients with lesions of the esophagus, stomach and duodenum. When mild to severe degrees of obstruction complicate the pre-

operative status, a liquid diet is particularly beneficial. Not uncommonly incomplete mastication of solid food results in even greater degrees of obstruction. When this occurs, one should lavage the chunks of swallowed food by means of a large-bore gastric tube (30 French) and thereafter a satisfactory amount of a liquid diet will pass the stenotic area each twenty four hours. Several diets have been tried at the University of Minnesota Hospitals, but the following, with minor variations has been the most adaptable.

Liquid Diet (2446 Calories)

	CARBO- HYDRATES (GM.)	PRO- TEIN (GM.)	FAT (GM.)
Whole eggs, 6		36 0	36 0
Egg whites, 2		8 0	
Skimmed milk powder 4 oz.	58 8	40 4	1 2
Lactose, 300 gm. (beet or cane sugar may be substituted)	300 0		
Skimmed milk, 1000 cc.	50 0	36 0	
Salt, 5 gm.			
	408 8	120 4	37 2

About 1.6 calories per cc.

This mixture passes readily through a 16F nasogastric catheter or a small Penrose drain. Tubes constructed of plastic are less annoying to the patient than are those of other materials particularly when they must lie in place for several days. This liquid diet can be dispensed through a drip-feeding apparatus. For this arrangement, the best functioning containers have been adapted from intravenous flasks with dependent air vents. This permits the entering bubbles as they rise, to agitate the mixture gently reducing the tendency toward sedimentation and plugging. A Murphy drip apparatus is inserted into the connecting tubing and the visible rate of flow can be regulated by the patient by means of a thumb screw at attachment. Particularly in the case of a feeble person, it is a wise precaution to have the head of the bed elevated while the patient is being drip fed. Excessive sedation should be avoided for the same reason i.e., to reduce the likelihood of overfilling the patient, which may result in regurgitation, aspiration and pneumonia.

For those who prefer to drink the above described mixture, the taste is similar to an eggnog. This flavor can be readily modified with chocolate, vanilla or other obvious choices. The diet can be used by outpatients also. Simple mimeographed directions about

its preparation the volume to be consumed daily and the refrigeration requirements should be given to the outpatient together with instructions covering the importance to the patient of drinking the prescribed amount. Whenever this liquid diet constitutes the sole source of food for the patient, he should be urged to take daily if possible about 5000 calories i.e. 3 liters. When it is used as a supplement to ordinary fare, the consumption of 1 liter may suffice. Diarrhea will sporadically occur in persons receiving this diet, but it can largely be avoided by keeping unused portions of each freshly prepared batch under refrigeration and by the use of clean or sterilized dispensing equipment. If diarrhea occurs despite these precautions, small doses of paregoric and/or Amphojel will ordinarily correct the situation. Patients with regional enteritis or ulcerative colitis are rather consistent exceptions to this rule and should be tested with small amounts to determine their individual tolerance. For a hospitalized patient, the use of this liquid with a fixed caloric value per cubic centimeter simplifies the calculation of the daily caloric intake on the basis of the volume consumed. The amount is charted, preferably as calories, along with the temperature, pulse, respiratory rate, fluid intake, output volumes and body weight. The nutritional status is thus brought into focus at the bedside while rounds are being held. This close contact with dietary management through a day-to-day accounting of progress or loss is of fundamental importance for the consistently effective preoperative care of substandard risk patients.

It is important when dietary preparation is required that it take place in the preoperative phase, because the weight of investigative evidence indicates a greater percentage of nitrogen retention than from protein ingested soon after operation. The establishment of any large nitrogen gain then offers considerable difficulty because of the catabolic phase present in the immediate postoperative period. The problem becomes even more awkward when the enteral route is no longer available, as is likely in this phase because of ileus or vomiting.

Objective measurements of the accomplishments of this hyperalimentation regimen are virtually nonexistent. Until more critical tests are available, the method to be presented offers a reasonably reliable means of calculating the duration of the special dietary regimen. First, it is important to find out the approximate amount of weight

trary the patient who has sustained a recent serious weight loss is malnourished, i.e., represents a substandard operative risk. Major operations performed on a group of these individuals will carry increased mortality and morbidity rates. This has been repeatedly demonstrated by many surgeons through and personal experience. Therefore, surgical benefits are not infrequently denied the malnourished patient. If he is accepted without special nutritional rehabilitation after appraisal and acknowledgment of the increased hazards, the operative manipulations are often poorly tolerated. The patient may react unfavorably from the outset, with an instability of blood pressure out of proportion to the actual blood loss. Numerous transfusions at the time of operation seem to provide only transient support to the blood pressure. Convalescence is slower than normal and is disturbed by a greater incidence of complications such as ileus, impaired wound healing and disruptions, or stomal obstruction. This patient, already enervated by starvation, lacks the vitality to overcome such hazards and the surgical risks are greater than for a properly nourished subject.

Those bodily derangements produced by severe starvation are hepatic dysfunction, impaired wound healing, hypoproteinemia and a contracted blood volume. These pathologic changes are reversible when appropriate dietary management is possible. The return to normal is slow and it requires many days or weeks of therapy to achieve recognizable nutritional restitution. Sudden and dramatic benefits are rarely obtained. The clinical problem is also complicated by a dearth of simple, accurate methods for precisely measuring the effects of starvation. Likewise, there is no test to indicate the quantity of nourishment required for the re-establishment of sufficient health to allow major surgery without excessive risk. Fortunately the restoration of the bodily economy to less than ideal performance suffices. After an adequate but partial nutritional restoration of the patient, the surgeon can operate and anticipate a convalescence as free from complications as in a standard risk individual.

During periods of relative starvation, the patient becomes autocannibalistic. His energy requirements are derived principally from body protein or fat and he subsists on a low calorie, low protein, high fat diet. Logic suggests, and experience confirms the characteristics of a diet most likely to cor-

rect the consequences of such starvation. In the laboratory and from clinical studies it has been found that this diet should be rich in protein and carbohydrate, high in calories and low in fat content. Of all these constituents, protein plays a leading role. The principal sources of protein in natural foods are meat, fish, eggs, certain dairy products and cereals. Yet in each of these, the protein contributes but a small fraction of the total bulk. It becomes necessary therefore, to give an enormous quantity of food when a large protein intake is desired. To achieve a protein intake of 300 gm. when beef is the source about 1400 gm. of lean meat would have to be eaten. This gargantuan feat is usually beyond the capacity of a patient who is weakened and anorexic from prolonged starvation. However reasonable quantities of tastily prepared and attractively served foods will foster the acceptance of a relatively high protein diet. Too the stimulus of a freshly devised daily menu and the cooperative appeal from bedside consultations among the surgeon, dietitian and nursing staff will add vitality to this nutritional program. In analyzing the effectiveness of this regimen, reasonably accurate calculations of the caloric intake can only be made from reappraising those quantities which the patient does not eat. An erroneous judgment is almost certain if reliance is placed on equating a prescription for a high protein and caloric diet with its consumption. Certainly every patient who willingly obtains the necessary quota from the available hospital fare should be encouraged to continue. Physical factors will, however tend to limit the accomplishments possible by this means. In such cases, liquid feedings will provide a useful means of supplementing the protein intake. Milk is an excellent vehicle for such mixtures and will hold substantial quantities of protein concentrate and carbohydrate. A wide variety of protein concentrates or digests are available. Skimmed milk powder about 38 per cent protein is among these. It is inexpensive, is widely available, stores well in bulk form and can be disguised to enhance its palatability. Its proteins are complete and possess a better than average capacity to induce plasma protein regeneration.

The use of a liquid diet to supplement the hospital fare, or as the sole source of calories is valuable and finds frequent application in patients with lesions of the esophagus, stomach and duodenum. When mild to severe degrees of obstruction complicate the pre-

other plasma volume expanders dextran and PVP. The contribution of gelatin to parenteral nitrogen therapy is dubious.

Protein hydrolysate mixtures are obtained from the acid or enzymatic hydrolysis of a variety of substrates. The resultant commercial preparations, usually after tryptophan enrichment, are widely available. Casein has frequently furnished the source of protein in these digests. The hydrolysates are readily infused as 5 per cent solutions with an equal or lesser concentration of glucose. A rate of infusion of about 400 cc. per hour is well tolerated in most cases. If the injection is much faster the incidence of unpleasant systemic reactions increases. The total nitrogen intake from parenteral use of hydrolysate mixtures is, therefore, limited to a smaller value than when the oral route is available. The total urinary nitrogen loss in twenty four hours following venoclysis of hydrolysates and glucose is greater than after the feeding of equivalent amounts of skimmed milk powder. Despite these limitations of parenteral therapy the hydrolysates occupy an important role in nitrogen replacement therapy. Through their agency it is possible to retard the steady protein losses from starvation and allied depleting mechanisms such as ulceration, sepsis and hemorrhage. Testosterone, in the Depo form as cyclopentylpropionate, has some anabolic effect and, hence, could curb the nitrogen losses. Use of this hormone unfortunately induces sodium retention. Therefore, intake of this electrolyte should be restricted (75 mEq per day) particularly in the case of the malnourished person.

The daily nitrogen intake is most substantially augmented by repeated transfusions of plasma or whole blood, a regimen which is not followed by a prompt "neutralizing" increase in the urinary nitrogen losses. Blood transfusions are preferable in the anemic patient, to be sure. Blood volume determinations, calculated for the usual weight prior to the current illness, provide a fair analysis of these requirements. Practically this is a somewhat demanding technical procedure for routine care. Daily infusions of 500 to 1000 cc. of whole blood should continue until the hemoglobin and hematocrit values reach and remain in the normal range. And as a corollary it can be expected that until the patient's normal blood volume has been reached, successive transfusions will not bring the hematocrit above a normal value. For the poorly nourished patient, as much as 2500 cc. of blood may be required. These

patients often suffer from a contracted blood volume and are poor operative risks until the blood volume is restored. A high grade correction of this hypovolemic reduced red cell mass defect can be customarily realized by this plan. The associated hypoproteïnemia is less regularly and predictably restored to normal because slight changes in these values are in "dynamic equilibrium" with quantitatively large debits in the total body protein. The plasma protein value can be raised additionally by the injection of albumin; however it is costly not always available nor frequently necessary. After this phase of blood volume replenishment, a daily transfusion of plasma and/or whole blood plus at least 3 liters of hydrolysate-glucose mixture, can be relied upon to provide the soundest possible parenteral preparation. At least a partial restoration of these various depots is essential in order to fulfill the aggressive protein demands of the healing tissues, the countless maturing phagocytes and the reticuloendothelial elements creating immune bodies in response to infectious processes.

This type of preparation represents a more complicated problem. It requires the services of a conscientious, tactful and patient house officer with sufficient skill to minimize the development of thromboses in all the cutaneous veins available to and essential for a prolonged period of intravenous therapy. In this connection, use of a forearm vessel away from the antecubital fossa avoids the distress of hyperextension at that joint. Also it permits some latitude of movement without the danger of dislodging the needle and burying it into adjacent tissue thereby causing a painful perivenous infection. In anticipation of a prolonged intravenous therapy program, sterile small bore polyethylene tubing can be inserted through the lumen of a large intravenous needle and into the blood stream. It may then be fastened with a dressing and left in place for several days. This conduit can be connected to the drip apparatus and avoids the need for frequent punctures of the patient's arm veins and frustrating of dispositions. At the conclusion of each day's infusion schedule, the tubing should be flushed with a few cubic centimeters of isotonic saline solution containing heparin and then plugged with a sterile intravenous needle of appropriate size to which has been attached a 2 cc. syringe containing a dilute solution of this anti-coagulant. It then remains patulous and ready for the next injection. When there is

that the patient has recently lost. This figure should regularly be available and recorded in the admission history. Patients often do not know the exact amount of weight lost but nearly always are able to recall a maximum weight determined for some time during the previous three to six months and this can be compared with the admission figure. Any decrease is more significant if expressed as a percentage of the total body weight. When this value becomes 25 per cent or more a period of at least two weeks of dietary preparation is desirable. Each day the patient should try to consume about 4500 to 5000 calories. If this total is obtained solely from consumption of a mixed diet, the proportions of protein, carbohydrate and fat should approximate those indicated in the liquid diet mixture. These estimates for the duration and amount of augmented caloric intake have been tested by trial and error for many patients having nutritional problems and probably are maximal rather than minimal. If the patient has had an adequate nutritional preoperative preparation, he can usually leave the hospital after the same postoperative period that a standard risk patient requires. In addition effective preoperative dietary management of substandard-risk patients will result in shorter hospitalization periods than will neglect of this consideration, performance of an early operation and the development of a complicated convalescence or a protracted interval requiring nutritional rehabilitation.

In some nutritional problem patients, the oral route is not available for protein and caloric replenishment because of alimentary tract obstruction. Ulcerative or neoplastic disease of the esophagus, stomach or duodenum is the most common cause. Ultimately a surgical procedure of considerable magnitude may be required to correct the pathologic condition. Two alternative methods are available for nutritional preparation. A jejunostomy can be made and the feeding mixtures then dripped into the intestine at its level. In many of these subjects, diarrhea, difficult to manage, develops before the quantity of material fed daily reaches the required caloric intake. This diarrhea often fails to respond despite recourse to a wide variety of dietary formulas and certain of the patient's limited reserves are depleted in the volumes of liquid stool lost.

Parenteral preparation. The other alternative is the establishment of a comprehensive parenteral feeding program which strives to attain a dual goal: (1) to meet the

daily caloric requirements, (2) to acquire the maximum possible nitrogen retention. The caloric needs are supplied by glucose* in 5 to 20 per cent solutions. At least 200 to 250 gm of carbohydrate daily is required to provide the body's energy needs. Unless this, or fat, is available, substantial quantities of either body protein or of infused nitrogenous material will continue to be catabolized for fuel. The degree of completeness of provision for this protein sparing action by carbohydrate is, therefore, an essential component of the ebb and flow of nitrogen reservoirs. Solutions for intravenous use containing fat emulsions will provide energy units which thus reduce the drain on exogenous and endogenous protein as a source of calories. Their best feature is the high (9) caloric value per gram of fat. Limiting factors in their use are instability, problems under varied climatic conditions and toxic systemic reactions, i.e., fever, chills, nausea, vomiting and headaches. In addition our knowledge to date is incomplete concerning the late consequences of prolonged use of these infusions. Instances of hepatomegaly, a hemorrhagic tendency and lowering of the plasma albumin fraction have been reported. The frequency of these side effects appears roughly proportional to the number of bottles of intravenous fat given to the patient. Therefore, prevailing opinion favors the tendency to limit this amount to ten to twelve separate half liter infusions. Meanwhile its use for a specific need which cannot otherwise be met has considerable justification and reasonable safety. It may yield some temporary benefits in the nutritional care of severely burned patients requiring high caloric intakes.

The other major control of the state of the nitrogen balance comes through the kind and the amount of the nitrogenous compounds administered parenterally. The nitrogen-containing compounds available for parenteral use are pure amino acid mixtures, gelatin, protein hydrolyzates, albumin, plasma and whole blood.

Pure amino acid mixtures are still rather costly and the limited supplies are largely allocated to investigative work.

Gelatin has some application to the emergency restoration of blood volume in shock therapy if plasma and whole blood are not immediately available, as have also those

If fructose is substituted, less will be lost in the urine even when the higher concentrations are administered, but the cost of the product is greater.

rate volumetric replacement of the measured blood lost during the operation. This is particularly important because the prevention of shock is much more successful than its treatment. Too the duration of the hypotension and the mechanism producing it are critical considerations in arriving at the prognosis for any specific case. Certainly the longer the period with a profoundly low blood pressure (< 60 mm Hg) which can be due to endotoxin shock associated with gram-negative bacteria in the blood, the poorer the outlook. This is in contrast to the situation in a patient suffering from brief hypotension due to acute hypovolemia which promptly responds to adequate transfusion.

The injudicious employment of sodium containing parenteral fluids is capable of imposing a serious cardiac overload. Unless careful attention is focused on this issue heart failure will develop in many patients with a low cardiac reserve. This sodium-retained accumulation of fluid can delay the operation or remaining unrecognized and untreated, be fatal. As a corollary of this, abrupt restoration of low sodium values to "normal" levels may also have lethal consequences in a patient previously maintained on a low sodium diet for the control of his heart disease.

Pulmonary Disease. The presence of any substantial amount of pulmonary disease is ordinarily ascertainable from the history, physical examination and admission chest film, with an assist at times from electrocardiographic findings of cor pulmonale. Routine preoperative measurement of the vital capacity is desirable before every major operation. Any serious reduction is particularly important if the patient is asthmatic, emphysematous or has advanced pulmonary hypertension. The use of the one-second vital capacity test is an even more valuable index of functional capacity in these crippled people. The capacity should be at least 80 per cent of the total expiratory volume. A simple but functionally useful test of the patient's respiratory reserve is to walk up a few flights of stairs with him and note his tolerance to this amount of physical activity that is whether he is able to converse naturally without labored breathing.

Patients with excessive tracheobronchial secretions benefit from preoperative postural drainage of the respiratory tract, the use of surface tension lowering and smooth muscle-relaxing drugs added to inhalants, avoidance of a very low environmental humidity and

differential ventilatory instructions if a pulmonary resection is contemplated. Patients with frankly purulent sputum should receive appropriate antibiotic therapy based on Gram stains, cultures and sensitivity tests for the predominant organism. Until this condition has been cleared up the patient is not a candidate for an elective operation. The prophylactic use of penicillin for several days preoperatively in such patients as well as in those with related disorders is an inexpensive precaution and appears to lower the incidence of postoperative pulmonary complications from atelectasis and pneumonia. Tobacco smokers, especially those addicted to the heavy consumption of cigarettes, should interrupt this habit for at least ten to fourteen days prior to an elective major procedure. Those patients awaiting a hernia repair or an extensive pulmonary resection or an abdominal operation likely to require a long midline incision are particularly benefited by the consequences of this action. Smokers are more likely to develop respiratory complications in the postoperative period than are nonsmokers. Even temporary preoperative discontinuance of this habit has an apparently beneficial effect.

Vitamins. The chronically malnourished patient may have either occult or manifest hypovitaminosis. If the clinical picture or laboratory tests suggest the diagnosis, specific vitamin therapy is indicated and, whenever the oral route is available, it is preferable. Intravenous preparations are more costly and their use should be reserved for those patients in whom the enteral route is unavailable because of obstruction, vomiting or diarrhea. In the absence of a deficiency requiring specific therapy, only vitamins B₁, C and K are important in the routine surgical care of patients.

Thiamine (vitamin B₁) participates in those enzyme systems which regulate carbohydrate metabolism and maintain normal gastrointestinal activity. In an nutritional preparation involving large quantities of carbohydrate, liberal amounts of thiamine (10 to 20 mg. daily) are advised. Intravenous infusions of glucose rapidly deplete the existing stores of vitamin B₁, thus aggravating any latent deficiency. Carbohydrate metabolism itself is impaired, further hampering the nutritional rehabilitation. Sulfa sulidine given orally will increase the thiamine requirements in the diet. When excess quantities of thiamine have been provided, they are either lost in the urine or stored and appear to cause no deleterious effects. The

a paucity of superficial veins, this method will help resolve that dilemma also. Caution is offered against routine use of the saphenous venous system lest one thereby provoke a high incidence of thrombophlebitis and phlebothrombosis. The saphenous vein is especially prone to those complications when it has been used for prolonged intravenous therapy.

The preparation of this group of patients by parenteral feeding should be compressed into as short a period as possible. This applies even to those with marked weight loss, anemia, hypoproteinemia, edema and other stigmata of starvation. They are prone also to those complications unique to an intubing nasal tube, *i.e.* regurgitation and aspiration, and, while accepting parenteral alimentation, more liable as well, to the effects of prolonged postural immobilization. Usually from seven to ten days can safely be devoted to achieving maximum nutritional benefits for them. Even less time may need to be spent in rehabilitating those patients who are only moderately malnourished from this same type of partial, high-alimentary tract obstruction.

The Obese Patient. The very obese patient requiring an elective operation also presents a substantial nutritional problem. To accept this patient for a major surgical procedure without due preparation is to accept an increased incidence of pulmonary cardiovascular problems and wound complications. In other words, the technical difficulties during the course of the operation are enhanced and perhaps even beyond one's remedy. The excessively fat person should be earnestly advised to lose weight beforehand. Then he should be given a diet to follow and his adherence to it must be closely supervised by the doctor. If he is to undergo the safest operation, his wholehearted participation will have to be enlisted by reasoning with him and by pointing out the life-endangering consequences of rashly disregarding advice to lose weight. Any balanced, weight reduction diet that is adhered to by the patient is satisfactory because the most important factors are a suitable protein and vitamin intake and patient cooperation. For this purpose, a simple yet effective plan allows the patient about 2 quarts of skimmed milk per day along with medical supplements to meet adequately the vitamin requirements. Obese adults will lose weight on this regimen. When the amount desired has been lost, based on standard weight and height tables the individual becomes a far better surgical risk.

Anemia and Hypoproteinemia. Chronically ill patients regularly exhibit some degree of anemia and protein depletion. The total extent of these defects is masked to a greater or lesser degree by contractions of the blood volume and/or plasma volume and protein reservoirs. As a consequence of this decrease in the red cell mass and in the total amount of circulating plasma protein, these patients are intolerant of additional blood loss during operation and display evidence of impaired wound healing and decreased resistance to infection. The term "chronic shock" has been applied in such cases.

Heart Disease. The patient with heart disease may require a major operation as urgently as does his healthier contemporary. With careful attention to certain details, his chances of successfully undergoing an abdominal or thoracic operation of magnitude can be greatly improved. An individual with historical or physical evidence of heart disease deserves to have any necessary additional studies carried out. Electrocardiography, cardiac fluoroscopy, venous pressure determination and a functional test of exercise capacity will furnish worthwhile preoperative data. Whether the findings are nearly normal or grossly altered, they represent significant base lines for comparison with later values. In the event of a complicated convalescence, a suspicion of incipient trouble gains greater credence and is easier to verify with such information available. When frank heart failure is detectable on the basis of pulmonary congestion or effusion or by hepatomegaly, ascites or peripheral edema, then preoperative medical advice and management are advisable. Meanwhile, it is best to initiate a program of salt restriction, combined with the use of diuretics, unless contraindicated by serum electrolyte or renal factors.

Hypoxia, hypercapnea, shock, overhydration with an excessive salt intake and hyperkalemia are the most common surgically induced mechanisms functioning as potential or real precipitants of cardiac disaster. The avoidance of certain of these states is customarily the responsibility of the anesthesiologist and the most knowing and effective of these specialists will cooperate as a coequal member of the surgical team.

Shock can readily precipitate a cardiac crisis in an organ which is already diseased. When the surgeon has been warned by the admission history, the physical examination and special tests, he should take certain precautions. Among these is provision for accu-

requires time. Therefore this program should be carried out in elective surgical cases on two or three occasions prior to the day of the operation. A final cleansing of the area adjacent to the incision is recommended just prior to application of the skin antiseptic in the operating room. In theory the patient should not be shaved the day or evening before an operation because any scratches or abrasions of the skin created by this can become infected. If the operative incision then crosses such an area, these multiplying bacteria will be seeded into the depths.

Gastrointestinal Preparation. Insertion of a nine-holed gastric tube, and the application of continuous gentle suction to it, the evening before any major intra-abdominal operation permits the surgeon to work with a collapsed bowel and facilitates any procedure thereon. This is especially effective in combination with a thorough cleansing bowel preparation in a patient requiring repair for a pendulous ventral hernia.

For operations upon the stomach and esophagus in the presence of obstruction, extra care should be taken to remove food debris, which can be a rich medium for pathogenic bacteria, particularly in an achlorhydric environment. Repeated washings with a large-bore tube are required upon these occasions to remove this partially digested material. This precaution effectively decreases the potential for regurgitation and aspiration of material which upon reaching the bronchioles and air sacs obstructs and necrotizes these tissues. Such a complication, assisted by any bacteria present, produces diffuse, even life-endangering, pneumonitis. The use of neomycin orally will effectively reduce the colony strength of the gastric bacterial flora.

In patients having incomplete or potential obstruction of the colon, it is highly desirable to prevent this from becoming complete. Otherwise, a preliminary decompression operation may be necessary in addition to the resection procedure later. Therefore, care should be taken not to force any substantial amount of barium proximal to a narrowed area in a patient in whom an x-ray examination seems necessary to confirm the nature of the large bowel obstruction.

The diet should be selected for its low residual content. The bulk and consistency of the stool can be effectively reduced also by giving Sulfathalidine, or Sulfasuxidine and neomycin by mouth. Mineral oil orally

several times daily will counteract a tendency to impaction above the lesion. If these measures prove ineffectual and some degree of obstruction persists though it is still incomplete the temporary use of continuous gastric suction may still obviate the necessity of colostomy. Any final nutritional preparation can then be accomplished through parenteral alimentation.

For any major abdominal procedure administration of 2 to 3 ounces of mineral oil by mouth the night before an operation, and when the nasal tube is removed will minimize the distress of passing the first postoperative, often inspissated, stool.

Final Preparatory Steps. When an operation is likely to be started late, it is wise to begin to meet the patient's fluid requirements earlier in the day with an intravenous injection of 1000 to 1500 cc. of a 5 to 10 per cent glucose solution containing 8 to 9 gm of sodium chloride together with any vitamin supplements. In addition to fulfilling some of the body's energy requirements, any tendency to preoperative dehydration is controlled. It is to be noted here that unless the patient voids or is catheterized before going to the operating room, serious vesical overdistention will develop before the patient can micturate or be catheterized postoperatively.

The availability of prepared preoperative order sheets on the surgical stations simpli-

Preoperative Order Sheet

1. Scrub _____
1. Entire patient with G-11 soap _____
2. _____ area
2. Fluids today _____
- _____ cc. 5% glucose in distilled water
- _____ cc. 5% glucose in saline
- _____ cc. 10% glucose in distilled water
- _____ cc. _____ and with _____
3. Tap water enema _____ tonight
- _____ A.M.
4. Draw blood and crossmatch _____ bottles
5. Pentobarbital, mg. _____ h.s.
- _____ A.M.
6. Weigh patient before going to O.R. in _____ A.M.
7. Nothing by mouth after _____ A.M.
- _____ P.M.
8. Insert nine-holed nasogastric tube _____ P.M.
- (Connect and check function of it)
9. Insert (connect and check function of it) urinary catheter _____ P.M.
- (Have patient void before being taken to O.R. if not catheterized) _____ A.M.
10. Wrap both limbs (toes to groin) with elastic bandages _____ A.M.
- _____ P.M.
11. On call from O.R. morphine sulfate _____ mg.
- atropine _____ mg.
- codeine sulfate _____ mg.
- Demerol _____ mg.

preferred route for administration is the oral one, because of the slower absorption and hence more prolonged systemic action. If thiamine is given intravenously with glucose solutions the daily dosage should be divided, lest an unnecessary excess of the vitamin be lost by diuresis.

The contribution of vitamin C to wound healing is clearly established. A low tissue vitamin C content is correlated with an increased incidence of wound disruption the edges of which would reveal a collagen-formation deficit. The plasma ascorbic acid content can be readily measured. Vitamin C deficiencies result from an inadequate intake, faulty intestinal absorption or an excess loss from, or sequestration by the tissues. In the instance of burns abscesses or large ulcerating areas, this mechanism can increase the daily requirements to at least 1000 mg. Tissue saturation is easily attained by means of oral preparations and parenteral solutions are available when that route can not be used. Yet no large-scale, long term storage probably exists in man. Any superfluous intake is lost in the urine causing no harmful reaction. A daily dose of 500 mg. is ample for most contingencies met with in the preparation of surgical patients.

A prolonged deficiency of vitamin K, a fat-soluble vitamin, disrupts the clotting mechanism by preventing the formation of prothrombin by the liver. The resultant hypoprothrombinemia interferes with blood clotting at the thrombin-conversion stage. The common antecedent to vitamin K deficiency is any mechanism which prevents its formation in, and absorption from, the intestinal tract. Prolonged oral use of poorly absorbed antibiotics can so reduce the bacterial flora of the intestine as to inhibit vitamin K synthesis sufficiently to cause serious hypoprothrombinemia. In some types of biliary obstruction, or with an external biliary fistula, the emulsifying action of bile is lost and consequently this naturally fat-soluble vitamin cannot be absorbed. Vitamin K should be given parenterally in such cases. Hypoprothrombinemia occurs also in association with severe hepatic disease and if the plasma prothrombin value is not correctable to around 80 per cent of the normal with therapeutic amounts of parenteral vitamin K, it becomes of grave prognostic significance.

Essential hypoprothrombinemia, a rare disease and conceivably a congenital metabolic defect, is an exception since other evidence of hepatic disease may be lacking in

these patients. For those persons with hypoprothrombinemia which is not benefited by vitamin K therapy massive transfusions of freshly drawn plasma or whole blood will temporarily elevate the plasma prothrombin content and the bleeding tendency can usually be controlled sufficiently to permit an operation. In any emergency minor defects of circulating prothrombin concentration can be so managed. With the availability of vitamin K₁ oxide for intravenous use, a more prompt restoration of prothrombin content to circulating blood is now possible even in persons who have been receiving Dicumarol like compounds.

All patients suspected of being hypoprothrombinemic should have a test for prothrombin concentration. This result is usually checked by determining the plasma prothrombin time of a normal patient's plasma used as a control for the materials and methods. Also this function can be charted as the percentage of normal prothrombin activity: the normal patient's plasma prothrombin time divided by the patient's plasma prothrombin time multiplied by 100. The prothrombin percentage can be calculated from a curve derived by plotting prothrombin times against serial dilutions of normal plasma. The curve is accurate as a standard only when the same potency of thromboplastin used in obtaining the curve is used for testing the unknowns. Prolonged bleeding is unlikely to occur unless the patient's prothrombin time is more than twice that of the control, or until the percentage of activity is lower than 50 per cent. In most instances of vitamin K deficiency considerable response is seen within twenty-four to thirty-six hours after therapy is started. A daily dose of 5 to 10 mg. of vitamin K customarily will suffice for this purpose. Quantities of the vitamin in excess are well tolerated and apparently do no harm. Water-soluble preparations for parenteral use are available whenever medication cannot be given orally.

Skin Preparation. The currently vexing difficulty with wound infections unfortunately often of staphylococcal origin, stems from multiple sources. One of these is from organisms within the patient's own dermal crypts. Some protection against this locus can be realized by repeated lathering of the patient's entire body with soapsuds containing hexachlorophene. For the ambulatory individual, this need be no more than particularly thorough sessions in the shower. The action of this surface-acting germicide

Hypoxia and Hypercarbia Avoidance of hypoxia is largely in the province of the anesthesiologist. It is his obligation to insure an open airway at all times from the moment of induction through the recovery of consciousness. Having developed a patent airway, his next commitment is to provide for adequate pulmonary ventilation with an oxygen rich mixture. Whenever the patient fails to or cannot respire deeply enough, the prevention of hypoxia is incumbent on the anesthesiologist.

Far more insidious than hypoxia and at least as detrimental to the patient's welfare is hypercapnia. The gradual accumulation of significant volumes of carbon dioxide in the anesthetized patient's blood due to inadequate ventilation, produces profound metabolic consequences when allowed to persist for any substantial time. This treacherous situation has few overt manifestations likely to alert the anesthesiologist. Direct measurements of p_{H_2O} on the circulating arterial blood or analyses of the expired breath are revealing of the problem's magnitude. Other than in those institutions actively studying some aspect of ventilatory problems, however, the carbon dioxide analyzer is not routinely used by anesthesiologists despite the fact that this instrument is readily available, clinically important and has an acceptable accuracy over a reasonable clinical range. In the absence of such precautionary devices, the only protection against hypercarbia is an unflagging attention to the maintenance of effective manual or mechanical ventilation. A failure to recognize the pile-up of carbon dioxide until the accumulation is able to produce severe acidosis, may well be an initiating factor capable of triggering abnormal vagal reflexes and cardiac standstill. Moreover, hypercarbia through its vasopressor effect may mask coexisting hypovolemia and the latter becomes disastrously apparent only when the blood carbon dioxide tension has been reduced toward normal.

POSTOPERATIVE CARE

The unconscious or semicomatose patient should be returned from the operating room with the head lower than the feet on a cart designed to be elevated at one end. As a substitute procedure, he can be positioned flat on his side with the dependent thigh and knee flexed and the upper one extended to provide some rotational stability. The head then rolls from the shoulder allowing secretions to drain away. Whatever the posi-

tion or method of transport a clear airway is the first consideration and a periodic check should be made even while the patient is en route from the operating room to his bed. The principal object of either the head down or alternative position is to prevent regurgitation and aspiration. Certainly saliva or gastrointestinal contents which are allowed to accumulate in the tracheobronchial tree postoperatively are a major cause of atelectasis, pneumonitis and pneumonia. An effective prophylaxis against this occurrence is the routine use of gastric siphonage during an operation. This arrangement will also minimize the development of distention secondary to the anesthetist's pumping of gas into the intestinal tract while he is forcefully ventilating the patient by means of a face mask. In the early postoperative phase repeated nasopharyngeal aspirations are frequently required to care for the gathering saliva when the volume exceeds the patient's capacity to handle it.

The use of a recovery ward in proximity to the operating room represents a current trend which has already earned approval at many institutions. When this very helpful arrangement exists, all anesthetized patients should be kept there during the immediate postoperative phase, until vital signs are stable and the individual responds to inquiry. This grouping is conducive to the establishment of more efficient routines. Likewise, the management of postoperative emergencies will be improved since responsibility for the patient's nursing care during the important first few hours is met by this trained organization. Too changes in, and education of this numerically more limited personnel are less troublesome than when the entire nursing surgical service must be exposed to the many individual variations occurring in the postanesthesia recovery period. In turn any newcomer to the recovery room rapidly gains concentrated experience in the recognition of most types of early postanesthetic and surgical difficulties. Intelligent and alert bedside nursing by someone well oriented in such problems is essential to the routine achievement of smooth convalescence.

Oxygen therapy equipment, tracheostomy trays, transfusion apparatus, motor-driven suction and other resuscitative equipment and drugs should be kept on hand for prompt use. A well trained group of nurses and doctors staffing a recovery ward can demonstrate teamwork in the management of an with cardiac arrest. This

fies this stage of readying the patient for his operation. It also serves as a useful check list of the customary things to be done.

Discussion of the Operation with Patient and Relatives. The preoperative preparation has not been completed until the surgeon has described carefully in language which the patient should be able to understand, the procedure contemplated. A discussion of the operation should include clear-cut explanations as to why it is necessary. It is particularly important to describe the consequences to a person about to undergo colostomy, an amputation, a visibly deforming procedure, an endocrine removal certain to require substitution therapy or to alter the patient's life-giving powers. This discourse must be carefully balanced so as to be informative but not anxiety inducing and is always to be couched in terms of interest in the person and with sympathy for his problem.

Upon completion of an operation and after ascertaining that the patient is doing satisfactorily in the immediate postanesthetic phase the thoughtful surgeon communicates with the relatives. He should devote as much time as may be required to a forthright discussion of the problems encountered and how they are being solved, and he should appraise the likelihood for a successful outcome. This can be reassuring to those in waiting who are naturally anxious.

OPERATIVE CARE

During an operation, attention should continue to be focused on the same physiologic factors which were of concern preoperatively i.e., any blood, electrolyte or fluid deficiencies, together with attention to cardiorenal and pulmonary dysfunction. The employment of a scale for accurately weighing the blood lost during the operation is of value. Throughout the dissection dry fluffs and packs are used to collect blood and, in addition, all blood suctioned from the field is collected into a calibrated trap system. Inasmuch as the tabulation is kept current by an attendant, a running account of the blood loss is available to the surgeon. The resultant figure is uniformly on the low side of the actual volume of blood lost and should be so interpreted when the amount of blood for transfusion is being decided.

Exposed viscera should be protected against dehydration by covering them with packs moistened in saline or Ringer's solution. Otherwise, various types and sizes of dry sponges are employed and when they become blood stained and have been passed

out of the sterile field to be counted by a circulating nurse their gain in weight can be tabulated. This gravimetric determination of the blood loss compares reliably with either colorimetric or isotopic estimates. The wisdom of shock prevention versus shock therapy is endorsed by providing the patient with an amount slightly in excess of those volumes which have been measured. Furthermore, a surgeon who is regularly confronted with objectively calculated blood losses larger for comparable procedures, than those his colleagues find necessary can profitably speculate about his record and his methods for hemostasis.

For children up to 20 kg in weight, use of a scale in the operating room is also a highly accurate means of measuring the total blood loss during an operation. If the child has been precisely balanced just before the operation begins, the only change at the termination of the surgical procedure must be from blood loss and the specimen removed.

The fluid losses through sweating during a prolonged operation can be considerable in an adult if the weather is hot and the operating room is not air conditioned. These fluid requirements should be met by replacement of water and electrolytes during the operation. Maintenance of adequate hydration will reduce the number of mechanisms provoking postoperative oliguria. The routine inclusion of a well functioning intravenous set up among the operating room facilities is therefore, strongly recommended for all major procedures. This equipment should be so adaptable as to permit the ready infusion of a variety of solutions and should include a cannula, or needle, suitable for rapid transfusions. Either the antecubital or the saphenous vein may be used, but the needle must be shielded from possible dislodgment during operative manipulations.

For surgical procedures of any duration, some protection should be provided for all the patient's bony prominences. A sponge rubber or air mattress is best, but air filled rings will suffice. A scrawny individual, in particular can readily acquire areas of pressure necrosis unless protected. The unrelieved weight of the patient, the drapes, sundry equipment and a tired, leaning assistant have undoubtedly caused some bedsores customarily ascribed to negligent nursing care. Enough padding around the patient's elbow to prevent pressure palsy of the ulnar nerve is always a wise precaution.

should be avoided. With this sac about the heart widely cut and one hand placed inside effective cardiac massage can be instituted. The rate (80 to 100 per minute) of manual compression is important and should be adequate to provide a detectable pulse at the wrist or over the carotid artery. Increased cardiac filling is usually obtainable by placing the patient in a limited Trendelenburg position. In those instances of cardiac arrest when, after a few minutes of vigorous massage and with restoration of a pinker hue to the myocardium, the heart fails to recapture effective tone despite a regular rhythm, then an intraventricular injection of epinephrine (0.5 cc. of 1:1000) may prove effective. Once an airway adequate for effective ventilation has been achieved, and after the establishment of a modest circulatory pressure by efficient cardiac kneading, then the next steps designed to retrieve this situation can be managed in a more leisurely fashion. The need for planned, coordinated, prompt action to achieve these two primary objectives is to be emphasized however. That state can only come about when considerable forethought has been given to the management of this recurring problem by those individuals responsible in any post-anesthesia recovery room and on the surgical service. Opportunities for practicing such steps on the medical wards are in existence, but they are even less likely to be carried out successfully because of an over-all unfamiliarity with procedural details and lack of essential equipment.

Ventricular fibrillation may be the initial presenting complex when the pericardium is opened. If so no great haste is indicated to convert this vermiform inefficient activity to a normal thrust. A more important objective during those crucial first minutes is to restore, by manual massage, an effective blood flow while the patient is simultaneously being well ventilated. Moreover in certain instances of fibrillation handled by massage, once adequate ventilation has been achieved, spontaneous reversion to a normal rhythm occurs. On the other hand, efforts to defibrillate a cyanotic, dilated heart will usually prove to have been in vain and more brain damage will result. Cardiac tone must be restored and the myocardium be well oxygenated if defibrillation is to succeed. Defibrillation requires the use of an instrument capable of delivering, ideally for brief periods (0.1 to 0.5 second), 110 to 250 volts of low amperage current which is then distributed broadly on the heart surface by

paddle-shaped electrodes applied firmly to opposite sides of it. In the better made instruments, it is possible to set rather precisely the duration of this electrical pulse, thereby minimizing the total amount of heat delivered to the already damaged heart. Since lower voltage levels might prove incapable of converting the heart to a regular rhythm it is necessary that the instrument at hand be capable of discharging up to 250 volts. In order to prevent electric shock to the individual manipulating the handles of this defibrillating instrument it is vital that he be insulated with rubber gloves free of any holes and that there be no other contact with the patient. Use of an isolation transformer prevents grounding through the operator and limits the dangerous contact to a single electrode. Compact defibrillation units are now commercially available and should be looked upon as essential equipment in modern hospital operating rooms or postanesthesia recovery areas. Adequate units should be handily available to the surgical convalescent floors.

Intracardiac injection of M/7 sodium lactate, or a solution of 7 percent sodium bicarbonate, in 10 to 50 cc. increments will facilitate subsequent electrical defibrillation of a heart recalcitrant to the establishment of a normal rhythm despite massage and previous electroshocks. It should be recalled in this regard that until the tissue pH has been restored to normal values from the state of metabolic acidosis present in hypoxia, the pharmacologic effectiveness of pressor amines is materially reduced. After correction of the pH, the heart, if still flabby in tone, will derive appreciable benefit from the judicious use of dilute solutions of epinephrine (0.5 to 1.0 cc. of 1:10,000) introduced into the blood perfusing the myocardium.

When adequate additional assistance becomes available at any stage of this rapidly moving sequence of events someone should be delegated the responsibility of placing a wide bore cannula of metal or polythene into an acceptable peripheral vein. The availability of this route for infusion is always helpful and may prove of considerable importance at any given moment. The ability to establish deftly a cut-down type venous cannulation, leading to a smooth working infusion, is a modest skill yet one to be admired and cherished by the surgeon regardless of his stage of development.

Continued observation of the cardiac behavior is indicated for at least a half hour

is as emergent a situation as can confront any doctor and must be handled promptly decisively and effectively in order to provide that patient with the best opportunity for survival despite this catastrophe.

Cardiac Arrest and Resuscitative Measures. The term "cardiac arrest" is meant to include all forms of cardiac standstill in asystole or ventricular fibrillation wherein the heart is no longer able to propel a sufficient volume of blood through the circulatory system to produce a detectable blood pressure. With either cardiac standstill or the ineffectual motions of ventricular fibrillation no more than a few minutes must be allowed to intervene before the circulation is restored, if death or irreparable brain damage is to be prevented. The cells of the cerebrum are particularly vulnerable to the metabolic consequences of circulatory stagnation associated with cardiac arrest. After an interval of no more than five minutes their destruction progresses rapidly and so even though other major organ systems may ultimately be restored, including effective cardiac activity the individual may have been made decerebrate during this period of inadequate cerebral blood flow. The suggestion to reduce the self-destructive metabolic demands of the brain by prompt utilization of general hypothermia (30 to 32 C.) has much to recommend it, once an effective cardiac beat has been restored. Any tendency to shivering or convulsions must then be eliminated with muscle-paralyzing drugs to avoid inducing thereby an increase in body temperature. It may be necessary to maintain the lowered body temperature for twenty four to seventy two hours before respiration to a euthermic state is safe.

There are many methods of preventing this grave complication, but it is to be emphasized that a definite number of such instances can clearly be identified as arising from improper anesthetic techniques, with particular reference to the management of high levels of carbon dioxide accumulation in the blood during the anesthetic interval. Hyperventilation, even with air under these circumstances can trigger a bout of ventricular fibrillation.

It is crucial to the success of these resuscitative measures that provisions be made in advance for handling this situation, firstly that the equipment required be available and, secondly that the principles involved in surgical technique be clearly understood. An airway must be promptly established and adequate ventilation achieved. A tank, valve

and conduction system capable of delivering oxygen in high concentration to a tight-fitting mask, or even better into an endotracheal tube, is quite acceptable. When this equipment is unavailable then mouth-to-mouth insufflation should be tried. Any arrangement selected must permit satisfactory ventilation of the lung after this organ collapses following the opening of the chest. A prolonged search for mask, oxygen tank, surgical instruments and related paraphernalia converts an outlook grim, at best, into a hopeless cause.

All but those experienced in handling this complication will have major qualms about opening the chest when suddenly faced with that need in a patient presumed to have developed "cardiac arrest." At this moment of decision there can be no justification for debate or procrastination while seeking a superior to accept this grave responsibility. The need must often be met by an intern or young house officer. An indecisive attitude will inevitably prejudice the outcome or even preclude any hope for success. Even an unduly long interval, while the doctor is seeking conscientiously with his stethoscope to be sure of a distant heart beat, may jeopardize the patient's slim chance. Unless one would forfeit the patient's life by further hesitancy this diagnosis must either be confirmed promptly or disproved. The best method of resolving one's misgivings is to make a limited incision over the region of the fifth intercostal space. If this cut fails to bleed in the usual vigorous fashion that any wound should, then the operator can proceed with reassurance that that patient does, indeed, have cardiac arrest. In addition to decreasing the interval left to conjecture or speculation, there are therapeutic advantages of settling this quandary. Additional moments may unfortunately be lost in a search for sterile instruments and skin antiseptics. While certainly desirable, such preparations may not be available and their lack is not likely to be critical. Thanks to man's native resistance and the protection provided by antibiotics, one can rely on the use of a clean, but not aseptic, technique. This compromise will usually prove a wiser choice than blind insistence on rigid listerian principles. When, during the course of subsequent steps, it becomes feasible to shift to sterile instruments and equipment, this should surely be done.

Once the chest has been opened and the heart exposed, the overlying pericardium should be incised, but the phrenic nerve

should be avoided. With this sac about the heart widely cut and one hand placed inside effective cardiac massage can be instituted. The rate (80 to 100 per minute) of manual compression is important and should be adequate to provide a detectable pulse at the wrist or over the carotid artery. Increased cardiac filling is usually obtainable by placing the patient in a limited Trendelenburg position. In those instances of cardiac arrest when, after a few minutes of vigorous massage and with restoration of a pinker hue to the myocardium the heart fails to recapture effective tone despite a regular rhythm then an intraventricular injection of epinephrine (0.5 cc. of 1:1000) may prove effective. Once an airway adequate for effective ventilation has been achieved, and after the establishment of a modest circulatory pressure by efficient cardiac kneading, then the next steps designed to retrieve this situation can be managed in a more leisurely fashion. The need for planned, coordinated, prompt action to achieve these two primary objectives is to be emphasized, however. That state can only come about when considerable forethought has been given to the management of this recurring problem by those individuals responsible in any post-anesthesia recovery room and on the surgical service. Opportunities for practicing such steps on the medical wards are in existence, but they are even less likely to be carried out successfully because of an overall unfamiliarity with procedural details and lack of essential equipment.

Ventricular fibrillation may be the initial presenting complex when the pericardium is opened. If so no great haste is indicated to convert this veniform, inefficient activity to a normal thrust. A more important objective during those crucial first minutes is to restore by manual massage, an effective blood flow while the patient is simultaneously being well ventilated. Moreover in certain instances of fibrillation handled by massage, once adequate ventilation has been achieved spontaneous reversion to a normal rhythm occurs. On the other hand, efforts to defibrillate a cyanotic, dilated heart will usually prove to have been in vain and more brain damage will result. Cardiac tone must be restored and the myocardium be well oxygenated if defibrillation is to succeed. Defibrillation requires the use of an instrument capable of delivering, ideally for brief periods (0.1 to 0.5 second) 110 to 250 volts of low amperage current which is then distributed broadly on the heart surface by

paddle-shaped electrodes applied firmly to opposite sides of it. In the better made instruments, it is possible to set rather precisely the duration of this electrical pulse thereby minimizing the total amount of heat delivered to the already damaged heart. Since lower voltage levels might prove incapable of converting the heart to a regular rhythm it is necessary that the instrument at hand be capable of discharging up to 250 volts. In order to prevent electric shock to the individual manipulating the handles of this defibrillating instrument, it is vital that he be insulated with rubber gloves free of any holes and that there be no other contact with the patient. Use of an isolation transformer prevents grounding through the operator and limits the dangerous contact to a single electrode. Compact defibrillation units are now commercially available and should be looked upon as essential equipment in modern hospital operating rooms or postanesthesia recovery areas. Adequate units should be handily available to the surgical convalescent floors.

Intracardiac injection of M/7 sodium lactate, or a solution of 7 percent sodium bicarbonate, in 10 to 50 cc. increments will facilitate subsequent electrical defibrillation of a heart recalcitrant to the establishment of a normal rhythm despite massage and previous electroshocks. It should be recalled in this regard that until the tissue pH has been restored to normal values from the state of metabolic acidosis present in hypoxia, the pharmacologic effectiveness of pressor amines is materially reduced. After correction of the pH, the heart, if still flabby in tone will derive appreciable benefit from the judicious use of dilute solutions of epinephrine (0.5 to 1.0 cc. of 1:10,000) introduced into the blood perfusing the myocardium.

When adequate additional assistance becomes available at any stage of this rapidly moving sequence of events, someone should be delegated the responsibility of placing a wide-bore cannula of metal or polythene into an acceptable peripheral vein. The availability of this route for infusion is always helpful and may prove of considerable importance at any given moment. The ability to establish deftly a cut-down type venous cannulation leading to a smooth working infusion, is a modest skill, yet one to be admired and cherished by the surgeon regardless of his stage of development.

Continued observation of the cardiac behavior is indicated for at least a half hour

after its condition has improved and while the general situation is being tidied up. This interval can be spent in acquiring sterile instruments and drapes to replace any unsterile equipment used in the emergency. If after this interlude, the systemic pressure is reasonably normal, assuring a respectable coronary flow the pericardium should be closed loosely with one or two sutures. In order to prevent prolapse of the heart into the pleural space. Tubes should be introduced into the thorax which will provide effective drainage for any postoperative intrapleural accumulations of plasma and blood. Any incidental air leak occasioned by a rapidly executed thoracotomy requires an anteriorly positioned catheter. If there has been obvious contamination of the pleural space, this area should be washed liberally with sterile saline solutions in order to dilute the number of organisms present and flush out any extraneous clots seeded with them. The thoracotomy closure can then be completed in any standard fashion.

The ultimate prognosis in any given case of cardiac arrest will depend upon a host of factors but the most important items probably are the duration and degree of cerebral hypoxia. True underlying myocardial disease will inevitably prejudice the end result. But, with attention to the details discussed above, dozens of patients can now be saved each year who would have previously been lost because of this abrupt and rarely anticipated disaster.

Additional Postanesthetic Care. After the patient reaches the recovery ward, or his own room, unless he is fully conscious and active it is best to elevate the foot of the bed slightly. In addition to the reasons cited previously the slope promotes evacuation of tracheobronchial secretions, discourages venous stagnation in the lower limbs and contributes to stabilization of the blood pressure. And, until the patient has resumed control of his faculties, it is important to remove excess mucous secretions regularly by the use of motor-driven suction which is applied intermittently through a catheter passed via the nose into the upper portion of the trachea. Pulmonary ventilation is promoted by turning the patient at intervals, urging him to cough and breathe deeply or through periodic hyperventilation with carbon dioxide-oxygen mixtures. These measures will contribute to a substantial lowering of the incidence of atelectasis and postoperative pneumonia.

Transfusions are given to balance losses in the operating room or recovery area and until the blood pressure and pulse rate are stabilized well outside any shock range. Sufficient fluids and electrolytes are supplied, foreseeing the total daily requirements. It is unwise to follow unqualifyingly the practice of giving only glucose in distilled water on the premise that sodium retention exists in the early postoperative period. Water intoxication as manifest by weakness, paralysis, twitchings or even convulsions, can develop. Especially in children, this syndrome may appear abruptly on the first or second postoperative day if the child receives parenteral fluids totally lacking in sodium.

The Intensive Care Unit. As suggested earlier, all patients should be kept in the recovery room until fully conscious and free from the immediate effects of the operation. Some hospitals provide recovery room convalescent care until the patient is ambulatory and has little demand for specialized nursing. Other institutions have developed a subrecovery or intensive care, unit in which specialized nursing is given to the patient after his transfer from the recovery room. Patients remain there until they are up and about and become minimal care problems. Here also the nursing staff becomes pre-eminently adept at providing a superior quality of bedside care and surgical procedures thereby are made safer for the patient. The availability in these areas of mimeographed postoperative order sheets for the doctors insures use of this list and will simplify the nursing routine and avoid overlooking important measures in this phase of postoperative care. A workable example of this arrangement is listed below.

Doctor's Postoperative Order Sheet

1. Vital signs q 15 min. x 4
q ½ hr until stable then
q 2 hr x 24 hr
2. Position _____ Low Trendelenburg
_____ Semi-Fowler's
_____ Flat
3. Hemoglobin stat. and in A.M.
4. Turn, cough, hyperventilate q. 2 hr x 24 then
q 4 hr until up and about.
5. Suction trachea and aspirate pharynx p.r.n.
6. Continuous gastric suction. Check p.r.n. (at least
q 4 hr) and irrigate as necessary with _____ cc.
of _____ solution.
7. Straight drainage to urinary catheter. Irrigate
p.r.n. with sterile water.
8. Chart urine specific gravity b.i.d. 8-6
9. Weight daily. litter _____ standing _____

10. Sedation
Morphine sulfate mg _____ h p.r.n. for pain
Codeine mg _____ q _____ h for pain
Demerol mg. _____ q _____ h for pain
Methadone mg _____ q _____ h. for pain
11. Penicillin _____ units q _____ h
Streptomycin _____ gm q _____ h
Other _____
12. Fluid order: ADD
_____ cc 5 glucose in distilled water
_____ cc 5 glucose in normal saline
_____ cc 10^{cc} glucose in distilled water
_____ cc 10^{cc} glucose in normal saline
_____ cc _____
13. Transfuse
_____ cc whole blood
_____ cc plasma
14. Blood loss _____ (chart on face sheet)
15. Privileges _____
16. Diet _____
17. Chest x-ray stat. _____ A M _____
18. Oxygen therapy
Test _____ Nasal _____

Operative and Progress Notes. An accurately written, descriptive operative note should be placed promptly on the patient's chart by the surgeon. The first surgical assistant should write a note immediately and place it upon the chart to help in guiding the postoperative care. Such a note is not designed to replace, but rather to supplement, the formal operative report dictated by the surgeon. This summary by an assistant should mention the kind of anesthetic, type and location of the incision, pertinent details of the operative findings, principles of the operation carried out, amount of blood lost and transfused, type and location of drain, whether T tube or catheter was used and method and suture materials used in the closure. Any unusual technical problems or maneuvers should be appropriately identified along with the names of those doctors participating in this operation. Progress notes should be regularly added to this report, particularly during the first few days, and always whenever any new problem arises to mar the convalescence. Any significant changes in therapy should be cited. A specific note should be made when any drain is removed or allowed to remain in place at the time of the patient's leaving the hospital.

Finally the notes should include a tabulation of the discharge instructions regarding diet, medications and recommendations for outpatient follow up visits. In all these reports the emphasis should be upon clarity and brevity without sacrificing relevant in-

formation and with minimal reliance on the use of eponyms colloquialisms initial type abbreviations and other pet symbols indecipherable to future generations seeking knowledge from these charts.

Nutritional Requirements. Granted that a reasonably satisfactory preoperative dietary preparation has been possible, the total caloric needs of the postoperative patient are of small concern if no serious complications arise to prolong this period. As soon as peristalsis has been re-established and after removal of the gastric tube on the second to fourth day the patient can be permitted a full liquid or soft diet. By the fifth to seventh day the convalescent often accepts with gusto the solid fare which is offered and is soon thereafter ready for his hospital discharge. Those persons of a leaner habitus, after gastric resection may feel the need of several small meals daily until the residual pouch and jejunum have become accommodated to the new status.* Since some of these patients tire easily from a moderate work load and are handicapped by their limited gastric capacity if they will drink 1 or 2 ounces of salad oil topped with fruit juice for increased palatability, two or three times daily energy units will be provided with but little bulk.

Wound Healing and Wound Infection. The avoidance of wound complications begins with the patient's preoperative preparation. Especially important are the nutritional aspects, including the establishment of near normal plasma albumin and hemoglobin values. A reduction in skin bacterial flora and sterilization of intestinal contents when the bowel is to be opened are also major considerations. Failure to meet these requirements may result in a seroma or hematoma, wound infection, disruption or evisceration. Wound infections occurring despite careful attention to these practices usually develop from unrecognized, gross contamination. Strict adherence to aseptic techniques by the operating personnel is essential, but it alone will not suffice. The more effective the precautions taken to control air borne, bacteria laden dust, the lower the incidence of infected wounds. The use in the operating suite of conditioned air under slightly positive pressure which has been filtered or better yet passed over an electrostatic par-

Certain annoying postgastroectomy sequelae can be minimized or even avoided if the convalescent will not indulge in eating to satiety during each meal. Rather he should fill his stomach incompletely upon seven or eight occasions daily taking care to select a diet high in calories for the volume consumed.

ticle collector or the use of a wet sorbent technique contributes to a reduction in wound seeding from air borne bacteria. Freshly laundered canvas boots placed over the street shoes of every person who enters each operating room help also in this connection. Requiring students, nurses, anesthesiologists and surgeons to provide extra foot gear to be worn only in the operating rooms will serve to decrease the amount of contamination introduced from other areas. Whenever architectural arrangements permit, the normal ebb and flow of hospital traffic should be routed so as to by pass the operating rooms.

During an operation the wound edges can be protected against excessive air borne contamination as well as against drying, by packs moistened with isotonic saline solution. Prior to closure, all wound layers should have a generous washing with this same solution to free them from the rich bacterial nutrient present in clot and tissue debris. Incisions which are so treated will then heal more kindly. Careful hemostasis throughout the operation, gentle handling of tissues, routine use of fine suture material, avoidance of mass ligation with its secondary necrosis, elimination of tissue dead spaces and anatomic dissections and repair respecting the residual blood supply all contribute to a high incidence of primary wound healing.

A wound hematoma usually arises from careless hemostasis and a seroma from failure to obliterate dead space. Therefore all incisions through a deep panniculus adiposus should be carefully approximated side to side as well as to the suture line of deeper fascia, from which fibrous layer it should not have been scraped—a practice unwisely indulged in by some. Rarely is a hematoma secondary to excessive postoperative anti-coagulant therapy or to an unrecognized preoperative bleeding tendency. When one does develop, the area of involvement should be opened widely and the clot evacuated lest it become secondarily infected. If liquefaction has occurred, and if the volume of liquid is small, aspiration under sterile precautions followed by a compression dressing will sometimes suffice.

The routine use of retention sutures is a matter of individual choice. Often they are inserted under so much tension, however, that considerable loss of effectiveness occurs from subsequent necrosis and shearing of the tissue inside each loop. The technique suggested by Price for their placement and care seems more logical than many older methods.

Various materials are used in skin closures. If metal clips are employed, their removal in two to four days is wise in order to avoid local necrosis and unsightly postoperative blemishes. Broadly placed, coapting skin stitches may remain five to seven days. The smallest cutaneous stitches (5-0 or 6-0 silk) catching merely the epithelial elements, cause no disfigurement when left fourteen to sixteen days and they support the edges during the later stages of healing.

Whenever one is required to incise the skin in a person with a known tendency towards keloid, provision should be made to give x radiation to this area unless other contraindications exist. On the other hand, incisions, and particularly vertical ones made in an area which has previously received heavy irradiation are quite likely to heal poorly. The phase of vascularization in wound healing has thereby been impaired and consequently that wound will recover strength slowly. When a reasonable alternative site exists for the opening it should be elected.

The Contaminated Wound. Certain wounds are unavoidably contaminated during operation and may become seriously infected if closely primarily closed. Hence, delayed closure is advantageous in these cases. In this technique, the deeper fascial planes are approximated in the usual manner after a particularly thorough flushing with Ringer's or saline solution. Sutures are placed in the superficial structures but are not tightened. A dry gauze pack inserted loosely into the incision holds the wound edges apart and the entire area is covered with a dressing. After forty-eight to seventy-two hours or even longer if the wound reaction and amount of drainage appear excessive, the pack can be removed and the individual sutures snugged up. A fresh external dressing is then applied. After-care is the same as for any wound. Provision for this interval of drainage averts any tendency to destructive burrowing by a pocketed infection and satisfactory healing has been assured both in the depths and at the skin edges.

Wound Disruption and Evisceration. Wound disruption is a serious sequel to impaired healing and occurs most commonly in midline abdominal incisions. Many factors predispose to this grave complication and in any one instance several may contribute. A knowledge of the more common mechanisms and how to avoid and/or overcome these hazards should help to reduce the incidence of this dangerous complication. The more

common factors contributing to wound disruption and evisceration are (1) in the preoperative phase (a) malnutrition, (b) hypoproteinemia, (c) chronic anemia (d) massive recurrent hemorrhage with or without shock and (e) vitamin C deficiency (2) in the operative period (a) improper selection of suture material and/or defective suturing technique, (b) careless hemostasis (c) an idiosyncrasy to absorbable suture material and (d) a midline or long vertical incision (3) in the postoperative stage (a) unusual abdominal wall strain from retching, persistent coughing, unrelieved hiccoughs, habitual sneezing, maniacal behavior with uncontrollable motor activity (b) marked distention and (c) uremia.

Evisceration may occur through any type of incision. Also, it may occur whether the wound is closed with absorbable or non-absorbable sutures. However much of the experimental and clinical evidence on this score suggests, but does not conclusively prove, that an oblique or transverse incision closed with some form of nonabsorbable material, is less likely to be followed by wound disruption. Whenever despite precautions to the contrary the possibilities of wound disruption become real, a tightly applied spica arrangement of tape to the abdomen extending from each groin to opposite costal margins will lessen this likelihood. That advantage is somewhat counterbalanced by the concomitant interference of such tight strapping with normal ventilation and venous return from the lower extremities.

Violent straining or retching is likely to occur at times just as the patient is recovering from the anesthetic. During such episodes if the sutures give way an audible though muffled sound can sometimes be heard and this, of course, requires prompt investigation. When wound disruption has thus been suspected, most surgeons advise a prompt return of the patient to the operating room, where he can be reanesthetized, prepared and draped. The wound is then opened and the damage corrected with suture material having greater holding power. If knots have also become untied, particular attention should be given to "squaring" their replacements. Any subsequent postoperative episodes of struggling should be controlled with a small dose of Pentothal given intravenously. With the awakening thus cushioned, it can be made less violent and any unusual pain should be obtunded with narcotics. Persistent coughing may be controlled with ample sedation,

bearing in mind the greater likelihood of atelectasis during prolonged suppression of the cough reflex.

Unrelenting, protracted hiccoughing exerts a severe strain on any abdominal wound. It may develop from a variety of harmless and inexplicable mechanisms but should suggest the possibility of such complications as gastric dilatation, peritonitis with subphrenic abscess, or uremia. Active treatment of any one of these which seems clinically likely should be given high priority for with improvement of the underlying mechanism, the singultus often abates. In the absence of any specifically treatable etiologic mechanism, brief periodic inhalations of 5 to 10 per cent carbon dioxide in oxygen will frequently interrupt the cyclic diaphragmatic spasm. Occasionally it may be necessary to block the phrenic nerve on the side involved.

Wound disruption and evisceration may occur at any time in the postoperative period, but customarily this regrettable occurrence takes place about the seventh day. The patient occasionally volunteers that "something gave away inside." If the skin remains intact, the condition is called wound disruption or dehiscence. If abdominal viscera are extruded, it is called evisceration. In cases of disruption loops of bowel can sometimes be palpated under the cutaneous layers. Intestinal obstruction may occur from knuckling of a bowel segment into the separation and this may be the first recognized sign of wound disruption if the character of the initial wound drainage has been ignored. Severe localized pain in an incision, if accompanied by other evidence of mechanical bowel obstruction, should also suggest this possibility. In fact, ileus of otherwise unexplained origin may portend the development of this condition.

Shock may be present or rapidly develop in patients with evisceration and appropriate measures must be taken to provide transfusions promptly. It is a complication of calamitous proportions, the patient's life is in jeopardy and these developments should be so viewed. Any sudden staining of the dressings with a profuse, pink, serous drainage is virtually diagnostic of wound disruption or worse and makes it mandatory to remove all dressings and examine the incision. Before doing this, however, one should anticipate the probable need for special dressings and equipment. The surgeon should don sterile rubber gloves, a cap and a mask in order to provide the patient's wound with maximum protection from contamination.

tion A sterile covering should be at hand to lay over any protruding loops of bowel. In the event no viscera are visible or palpable, a few stitches should be removed with an aseptic technique and the depths of the wound probed or visualized to confirm the fact that dissolution has taken place. It is unusual not to find some area of disruption after a profuse typical serosanguineous discharge.

Treatment for wound disruption Two methods of treatment are used for disruption and evisceration. For a patient who is a poor risk, has tenuous parietal fascia or is in profound shock, a conservative technique which can be carried out at the bedside is required. In the case of disruption of an abdominal wound, before either method is used, the passage of a nasal tube into the stomach to evacuate its contents will reduce the likelihood of regurgitation, endotracheal aspiration and probably pneumonia. Several preliminary applications of a topical anesthetic to the nasopharynx will minimize retching, and further disruption of the wound, during tube passage.

After the adjoining skin and wound edges have been prepared with an antiseptic, visible loops of bowel or other viscera should be cleansed thoroughly with liberal amounts of sterile isotonic saline solution and then returned to the peritoneal cavity. They should next be held in place temporarily with a sterile towel or a wide wad of gauze packing. If cooperation or relaxation is unsatisfactory the patient can be lightly anesthetized and given as well a muscle-paralyzing drug intravenously. Soft rubber drains 1 inch wide, should extend from the upper and lower extremities of the wound. The skin of the entire abdomen, flank and lower part of the chest is then cleansed with ether-dampened sponges and painted with compound tincture of benzoin. Long adhesive strips, which have been flamed for bactericidal purposes and to enhance their grip to the skin, are used to draw the wound edges together. These strips, stretching diagonally from gluteal fold to axilla, overlap each other from below upward. The defect is closed by developing traction toward the wound edges with each strip. With the inferior tape end stuck firmly any tendency to retraction of the incision is countered by sliding the lateral tissues toward it. When the taping has been completed, the rubber drain should jut enough beyond the tape to carry away the discharge which might otherwise weaken the effectiveness of the tapes

holding power. Care is required to prevent a bowel knuckle from becoming incarcerated in the wound during this type of closure. The appearance later of signs of mechanical intestinal obstruction and localized pain would point to this possibility. Some ileus is liable to develop in most cases. The dressing, if properly functioning as a binder should remain in place for at least two weeks or longer if the patient has been poorly nourished. Although most of these patients will recover a ventral hernia in the line of closure is likely.

A healthier patient with wound disruption will tolerate a return to the operating room where formal repair can be carried out. After a nasogastric tube has been inserted and connected to suction and arrangements made for whole blood transfusion, the patient is anesthetized and the abdominal wall is resutured under aseptic conditions. Steel wire tension sutures, tied down over a dental roll, as proposed by Price, work well. It is believed by those who favor the reoperation method that convalescence is shorter and intestinal obstruction is less likely to occur.

Drains. The use of drains after surgical procedures remains a controversial subject. Advantages can be cited on both sides of the question. The blood, plasma, bile, pancreatic juice or intestinal contents which come out denote the presence of this problem and suggest its need for treatment. Those who prefer not to use drains cite a lower incidence of such complications which they allege are due to the presence of these foreign materials. Opponents cite references also to the incompleteness of the "drainage" achievement.

Several types of simple drains can be used. The most common ones: Penrose tubing, soft rubber catheters, rubber wicks and cigarette drains, rely on capillarity, gravity or slight pressure differences to effect the removal of fluid. Intra-abdominal drains customarily seal off from the adjacent viscera within forty-eight hours. However one may continue to ooze fluid, such as bile, from a site of leakage for many days. In such a case there should be no haste about removing it.

The other common type of drain is usually a more complex apparatus. It may have the form of a sump pump (Babcock) a double-lumen catheter (Chapin) air vent suction (Wangensteen) or a multiple holed catheter. These require mechanical suction to supplement the other forces of drainage and keep the selected area dry. They too, eventually become walled off but serve to keep a site of leakage or a pocket drier and thus facilitate

tate healing. Suction, applied to a multiple-holed catheter positioned under the skin flaps in a neck dissection or a radical mastectomy is a particularly effective technique for coapting widely reflected surfaces. The principal purposes of drainage are to prevent pocketing internally and to provide for external fistulization at the site of any leak. Once the drain has ceased doing either or both of these functions it should be eased out gradually a few centimeters at a time. In this way the tract can fill in from the depths. If all the tubing is pulled away at one time, the skin can seal before the depths are firmly united by healthy tissue. Pocketing and suppuration can still develop inside and complicate the later convalescence. Pain and localized tenderness will be noticeable in that area and may require subsequent re-opening of this channel.

Drains, particularly the firmer types, have been accused of abetting, or even causing, fistula formation when alongside an intestinal anastomosis. It is felt by those opposed to using them that the suture line is weakened by its proximity to a foreign body. Those in favor suggest that if provision for such drainage had not been supplied when the suture line gave way, subsequent leakage and contamination would have dissected more widely while seeking a path to the outside. Drains serve a necessary purpose when used to maintain an evacuation track from an abscess pocket; their prophylactic, routine employment has little to recommend it.

Intestinal Fistula. The treatment of an intestinal fistula is usually easier the more distal the segment of bowel from which it arises. When the fistula develops from the colon, as after its resection or at an appendectomy site, healing is slow but steady if no obstruction is present. Constipation should be avoided. The more proximal the fistula in the small intestine, the greater the difficulties with water and electrolyte loss and the more destructive to other tissues are the bowel contents. The juices from a high intestinal fistula will rapidly digest away all tissues of the abdominal wall. These structures can be partially protected by the following plans:

1. Proximal control of the volume of intestinal contents by (a) limiting the oral intake; (b) passage of a Miller Abbott or Grafton Smith tube down the gut to the fistula site with continuous suction on this tube and sufficient inflation of the distal bal-

loon to obstruct the lumen and thereby reduce the amount of discharge.

2. Control at the external opening by means of motor or water-driven suction delivered through a multiple-holed catheter to the proximal limb of the fistula, thereby aspirating any accumulation.

3. Protection of the skin and adjacent tissues. Many kinds of paste are available for this purpose. While the dermis is intact, powdered aluminum rubbed into the area periodically will give considerable protection. Ladd's paste, brewer's yeast and tannic acid in lanolin also are sometimes effective in controlling tissue digestion.

4. Relief of any intestinal obstruction distal to the fistula which would otherwise perpetuate this complication. As soon as the patient's condition permits the obstruction should be corrected by lysis of the adhesive bands by a short-circuiting procedure, an intestinal resection or by some other means of intestinal deviation. If the fistula is located low in the intestinal tract, some of the material aspirated from the gut can be returned through a secondary gastric catheter to the stomach for partial reabsorption in the upper portion of the intestine.

Better drainage for the intra abdominal ramifications associated with a fistula-forming episode can be obtained by placing the patient face down on a Foster or Stryker frame for specified intervals, as tolerated, several times each day.

The amount and kind of fluids and electrolytes necessary in these cases can tax the judgment of an experienced clinician, over all nutritional care is an equally difficult problem.

Atelectasis. Atelectasis can arise either from pulmonary compression by intrathoracic accumulations of blood clot, fluid or tumor or from obstruction of a portion of the bronchial tree by a mucous plug. The former mechanism rarely causes systemic symptoms other than a decrease in maximum ventilatory capacity and, occasionally, osteoarthritis. The latter invariably provokes a clinical picture characterized by fever and some tachycardia. Imperfect expansion of the lung, whether massive, patchy or plate-like, occurring in the postoperative phase, is invariably due to obstruction of a bronchus or its distal ramifications by a mucous plug. Secondary factors which may contribute to the development of obstructive atelectasis include the following:

1. Hypoventilation can result from too pro-

found sedation but is more commonly secondary to pain in the operative site, which tends to limit the usual costal activity. A vertical upper abdominal incision can contribute to this mechanism. As the ribs flare out with each breath, lateral tension pulls painfully on the wound edges. When pain is a restricting factor in the patient's ventilation, administration of an anodyne or providing a local anesthetic block of the area will often increase the depth of voluntary respiration.

2. Elevation of the diaphragm. An operation which weakens or paralyzes the diaphragm is commonly followed by some ventilatory impairment. After biliary tract surgery atelectasis is not uncommon. Marked distention also interferes with normal diaphragmatic activity.

3. Aspiration of material from the intestinal tract. These contents irritate the tracheobronchial mucosa and provoke an inflammatory reaction, which may progress to pneumonia. In milder cases an increase occurs in the volume of local secretions including mucus, and this too contributes to the high incidence of atelectasis after the accidental aspiration of intestinal contents. The aspiration of coagulable fresh blood is likewise provocative of atelectasis and pneumonia. Its removal, even by bronchoscopy can be troublesome.

4. Overatropinization. The administration of large doses of atropine inspissates the mucus and makes it more tenacious, thereby increasing the difficulty in bringing up these sticky plugs.

5. Anesthetic technique. Certain gases are more rapidly absorbed from the lungs than is air. Failure to "wash out" these mixtures at the end of an operation may contribute to an early appearance of atelectasis.

6. Existing pulmonary suppuration. In the presence of bronchiectasis or a lung abscess, pus, excess mucus and other secretions are already present and available. Loss of the cough reflex during anesthesia fosters retention of secretions and their spillage into adjacent pulmonary segments. The patient with pulmonary suppuration should spend a period several times each day before his operation in the head-down position and try to cough out as much as possible of the accumulated material. In such cases, bronchoscopy should also be done after the operation in order to insure thorough evacuation of the tracheobronchial tree, thereby minimizing the chances of postoperative atelectasis. The preoperative use of penicillin for

a few days in any person requiring an operation, despite the presence of coexisting pulmonary disease will subsequently alter the tracheobronchial flora and likewise reduce the volume of secretions. The likelihood of postoperative atelectasis and bronchopneumonia will thereby be reduced.

7. Smoking. Cigarette smokers, particularly the heavy users, should be firmly urged to stop this habit for at least ten to fourteen days before any major elective surgical procedure. This interval will permit the tracheobronchial tree to recover at least partially from the irritation and excess mucus formation frequently associated with "smoker's cough."

Usually the diagnosis of obstructive atelectasis is serious enough to cause clinical symptoms is readily made from the chart and the physical findings. Its presence can be confirmed by a chest film. Though the pulse is rapid, the temperature is elevated often out of proportion both to the heart rate and to the apparent illness of the patient. The onset of fever is sudden and commonly a temperature of 102° F or higher is reached. On palpation of the trachea in the neck a deviation to the affected side can often be noted. The respiratory excursion is more limited over one side than over the other. On auscultation a few sticky rales can be heard, but either the usual breath sounds are absent or bronchial breathing is noted in the area of involvement. Cyanosis and dyspnea may be present. The x-ray picture is essentially that of an area of increased density on the affected side, with a lobar mottled or platelike distribution, in association with elevation of the diaphragm, narrowing of the rib spaces and variable degrees of displacement of the trachea, mediastinum and heart to the affected side.

The treatment of atelectasis begins with attention to the predisposing and contributing factors. A lowered incidence and milder examples of this complication will result from such prophylaxis. Early ambulation probably helps avoid this complication by increasing the patient's ventilatory efforts. Once the process has developed, it is essential to treat it until it is cured. Failure to reinflate an atelectatic lung is likely to lead to chronic pulmonary changes and irreparable damage. Recurrent atelectasis in any person should alert the clinician to the possibility of intrinsic bronchial obstruction from an adenoma, carcinoma or nonopaque foreign body. Inducing the patient to cough, turning him in bed, hyperventilation with 5

to 10 per cent carbon dioxide in oxygen and a sharp blow over the side of involvement are often effective means of dislodging a plug and expanding a collapsed segment. Auscultation of the chest should confirm the efficacy of the treatment or indicate the need for further active therapy. Endobronchial aspiration with a catheter attached to suction will make the patient cough and may pull out the offending mucus. In a particularly uncooperative or lethargic patient, translaryngeal suctioning of the trachea and bronchi can be facilitated by using a laryngoscope and an appropriately curved open tipped catheter. If these simple measures fail bronchoscopic management is indicated. The end results of treatment are uniformly good when the disorder is corrected at an early stage of the process. If atelectasis proves intractable or has been neglected for many days, re-expansion is slower and chronic, irreversible, pulmonary changes are more likely to occur.

Tracheostomy A tracheostomy often has lifesaving advantages to bring into the management of specific problems incident to the control of tracheobronchial accumulations. Almost its sole drawback is the unattractive cervical scar as an aftermath, and this is minimized with a transverse incision. Tracheostomy is at times a wise prophylactic step on other occasions, it is a well chosen supplemental method of retrieving a deteriorating situation. With regard to the former indication, it may complement the patient's convalescence when extensive surgery about the head and neck has temporarily abolished full control of the swallowing reflexes so that saliva or liquids are prone to trickle into the trachea. The tracheostomy permits easier and more effective aspiration of this airway, clogging, mucoid drainage before it can reach the pulmonary terminals. After deglutition has been recovered, this accessory vent for saliva can be abandoned. For other individuals whose prolonged efforts to rid themselves of excess secretions have led to exhaustion and a weakened coughing power the addition of a tracheostomy is of slight surgical magnitude and considerable benefit. Its establishment can prevent drowning of a patient who is comatose or profoundly debilitated, and insensate to the healthy cough-provoking mechanisms of foreign material in the respiratory tract. Moreover when a tracheostomy opening is available, it simplifies the technique of bronchoscopy and thereby insures more ready use of this means to achieve the most complete and thorough

removal of all retained secretions. The ease of handling this problem afterward and the minimal disturbance to the patient so prepared are certain to encourage more frequent recourse to endoscopy. Whenever the eventual need for a tracheostomy appears likely it is best to establish one electively a few days prior to the major surgical procedure. A technically neater operation can then be done and the patient's respiratory tract will thereby have had adequate time to adapt to the altered humidity of the air which has by passed the oronasopharynx. During this interval it is particularly important to add moisture to the ambient air preferably through cold air humidifiers. Of course if oxygen is piped directly into the tracheostomy opening, it should first be dispersed through water in an attempt to saturate it. Otherwise desiccation and excessive tracheitis are likely to complicate and needlessly prolong the recovery from a tracheostomy.

Many persons otherwise well versed in postoperative problems exhibit a surprising reluctance to do a tracheostomy. They over look or reject the possible contributions of this simple procedure until terminal and irreversible states of pulmonary congestion and pneumonia prevail. The patient is then being destroyed by infection, hypoxia and hypercapnia. Unless last-minute efforts at removing occluding plugs and pools are successful, he is doomed.

Distention. Distention may develop during the postoperative period in either of two forms: acute gastric dilatation or ileus mechanical or inhibitory.

Acute gastric dilatation can appear after almost any operation, including thoracic and cardiac procedures. At times the procedure may seem almost too inconsequential to have caused this complication. Acute gastric dilatation appears also in patients with diabetes, during uremia and after fractures. Persons with compression fractures of the spine treated with a hyperextension frame or a cast are particularly likely to have this complication, the superior mesenteric artery duodenal compression syndrome may play a role. After a variety of thoracic operations patients at times exhibit mild to moderate gastric dilatation.

The inexperienced house officer can hardly be condemned for thinking first of hemorrhage or embolism when called to see a patient who, a few days after a major operation, is obviously in a grave condition, appears slightly cyanotic, is dyspneic, has a

found sedation but is more commonly secondary to pain in the operative site which tends to limit the usual costal activity. A vertical upper-abdominal incision can contribute to this mechanism. As the ribs flare out with each breath, lateral tension pulls painfully on the wound edges. When pain is a restricting factor in the patient's ventilation, administration of an anodyne or providing a local anesthetic block of the area will often increase the depth of voluntary respiration.

2. Elevation of the diaphragm. An operation which weakens or paralyzes the diaphragm is commonly followed by some ventilatory impairment. After biliary tract surgery atelectasis is not uncommon. Marked distention also interferes with normal diaphragmatic activity.

3. Aspiration of material from the intestinal tract. These contents irritate the tracheobronchial mucosa and provoke an inflammatory reaction which may progress to pneumonia. In milder cases an increase occurs in the volume of local secretions, including mucus, and this too contributes to the high incidence of atelectasis after the accidental aspiration of intestinal contents. The aspiration of coagulable fresh blood is likewise provocative of atelectasis and pneumonia. Its removal, even by bronchoscopy can be troublesome.

4. Overatropinization. The administration of large doses of atropine inspissates the mucus and makes it more tenacious, thereby increasing the difficulty in bringing up these sticky plugs.

5. Anesthetic technique. Certain gases are more rapidly absorbed from the lungs than is air. Failure to "wash out" these mixtures at the end of an operation may contribute to an early appearance of atelectasis.

6. Existing pulmonary suppuration. In the presence of bronchiectasis or a lung abscess, pus, excess mucus and other secretions are already present and available. Loss of the cough reflex during anesthesia fosters retention of secretions and their spillage into adjacent pulmonary segments. The patient with pulmonary suppuration should spend a period several times each day before his operation in the head-down position and try to cough out as much as possible of the accumulated material. In such cases, bronchoscopy should also be done after the operation in order to insure thorough evacuation of the tracheobronchial tree, thereby minimizing the chances of postoperative atelectasis. The preoperative use of penicillin for

a few days in any person requiring an operation despite the presence of coexisting pulmonary disease, will subsequently alter the tracheobronchial flora and likewise reduce the volume of secretions. The likelihood of postoperative atelectasis and bronchopneumonia will thereby be reduced.

7. Smoking. Cigarette smokers particularly the heavy users should be firmly urged to stop this habit for at least ten to fourteen days before any major elective surgical procedure. This interval will permit the tracheobronchial tree to recover at least partially from the irritation and excess mucus formation frequently associated with "smoker's cough."

Usually the diagnosis of obstructive atelectasis is serious enough to cause clinical symptoms readily made from the chart and the physical findings. Its presence can be confirmed by a chest film. Though the pulse is rapid, the temperature is elevated often out of proportion both to the heart rate and to the apparent illness of the patient. The onset of fever is sudden, and commonly a temperature of 102° F or higher is reached. On palpation of the trachea in the neck a deviation to the affected side can often be noted. The respiratory excursion is more limited over one side than over the other. On auscultation a few sticky rales can be heard, but either the usual breath sounds are absent or bronchial breathing is noted in the area of involvement. Cyanosis and dyspnea may be present. The x-ray picture is essentially that of an area of increased density on the affected side, with a lobar mottled or platelike distribution, in association with elevation of the diaphragm, narrowing of the rib spaces and variable degrees of displacement of the trachea, mediastinum and heart to the affected side.

The treatment of atelectasis begins with attention to the predisposing and contributing factors. A lowered incidence and milder examples of this complication will result from such prophylaxis. Early ambulation probably helps avoid this complication by increasing the patient's ventilatory efforts. Once the process has developed, it is essential to treat it until it is cured. Failure to re-inflate an atelectatic lung is likely to lead to chronic pulmonary changes and irreparable damage. Recurrent atelectasis in any person should alert the clinician to the possibility of intrinsic bronchial obstruction from an adenoma, carcinoma or nonopaque foreign body. Inducing the patient to cough, turning him in bed, hyperventilation with 5

100 to 200 cc of a 10 per cent sodium chloride solution will often induce peristalsis. However in any patient with persistent intestinal atony it is imperative to exclude an intra-abdominal abscess as the precursor of this state before fruitless efforts are expended on its management by the various methods cited above. If a leak at a suture line is suspected, or an extra-enteric accumulation with a fluid level is discernible on an upright x-ray film of the abdomen, the first step, and sometimes the only one required to relieve the ileus is to drain effectively this pocket of infection. Giving Congo red by mouth and finding this stain on the wound dressings aids in diagnosing the existence of an intestinal communication.

Subphrenic Abscess. Subphrenic abscess should be an uncommon complication after uncontaminated abdominal operations. A perforated ulcer or a ruptured appendix is the usual cause when there has been no antecedent surgery. Because of its often insidious onset, exploration of one or both subdiaphragmatic areas is frequently postponed. A subphrenic abscess may be anterior, posterior or on the left or right side. Combinations of these sites are, of course, possible, and rarely all four spaces can be infected simultaneously.

In a patient who has recently undergone a gastrointestinal operation or has had an other intraperitoneal insult, fever of unexplained origin should arouse a suspicion of subphrenic abscess. The physical findings may be surprisingly meager, slow to appear or even inconsistent. To wait until all consultants agree upon the diagnosis is often to delay too long. X-ray and fluoroscopic study can contribute considerable information and support for a tentative diagnosis. At fluoroscopy the diaphragm may be found to be elevated, sluggish or paralyzed. A fluid level pocketed under this leaf and in a specific area outside the intestine, is virtually diagnostic. A small quantity of barium in the stomach with the patient in the head-down position will help demonstrate an abnormal separation between the diaphragm and the gastric pouch. The presence of pleural effusion with obscuration of the costophrenic sinus is also a helpful sign pointing at subdiaphragmatic inflammation, although this finding has many times erroneously been construed as having arisen from a pneumonic process. Plain and overexposed Bucky films in anteroposterior and lateral projections are useful to identify the true

nature of this complication and to locate the abscess as accurately as possible.

Recognition of a subphrenic abscess is at times difficult in the early postoperative period after abdominal surgery. Air is in variably present under the diaphragms following such operations and may remain easily identifiable for two weeks. Persistence, a tendency toward encapsulation and the appearance of a fluid level are significant signs pointing more to an infection than toward some benign residuum of the operation. However a subphrenic infection need never show a fluid level and may merely remain as a smoldering granulomatous process. To complicate the clinical picture also the patient may be febrile from causes other than the subphrenic process. At times it may be impossible to make the categorical diagnosis of subphrenic process. Exploration is often justifiable then on the basis that further delay fosters the complications of diaphragmatic perforation, empyema, lung abscess, bronchial fistula, rupture and generalized peritonitis with a consequent rise in the mortality rate.

The principal purpose of operative treatment for a subphrenic abscess is to achieve continuous dependent drainage, whether the patient is reclining or standing. Except in the case of infection in the right posterior space this goal is rarely realized. When the pocket has been definitely located there the best approach is through the bed of the twelfth rib, as recommended by Ochsmier. For an anterior accumulation and after an anterior drainage procedure, some residual puddling in the depths is almost inevitable whenever the patient is supine. The prone position will, therefore, provide better drainage. It should be sought by maintaining the patient on a Foster or Stryker frame for as long a period each day as tolerated. Occasionally a secondary posterior opening will be required to complete the drainage process. The preferred approach to an anterior encapsulation is that of Clairmont. Through a subcostal incision extraperitoneal dissection is carried down to the area of involvement and this is then entered. In both the anterior and the posterior approaches, extraperitoneal dissection should be utilized until the abscess is reached and evacuated. The overlying surfaces become agglutinated around the pocket and the free peritoneal cavity is sealed off by the adjacent inflammatory reaction. Transperitoneal drainage, on the other hand, is much more likely to

rapid, thready pulse and cool extremities covered with perspiration. However if percussion of the upper abdomen elicits tympanitic sounds over a wide area the presumptive diagnosis can be established by passing a gastric tube and releasing a gush of air and then a considerable volume of coffee grounds-appearing fluid. Copious vomiting may never occur as a warning sign for this complication. Rather the patient repeatedly regurgitates small amounts of hematin-tinged fluid. Meanwhile, enormous quantities, several liters, can accumulate in the stomach and the underlying problem remain unrecognized. In fact, enough gastric juice may already have become sequestered in the stomach to leave the patient hypochloremic. With decompressive relief from the gaseous distention of the stomach and restitution of adequate quantities of salt and water and occasionally after a transfusion of plasma or blood, the patient usually makes a rapid convalescence. In a few hours, it may be difficult to realize that only a short time before the patient's condition was critical. Failure to recognize and treat acute gastric dilatation can on the other hand lead to a fatal outcome. Prophylaxis is as effective as therapy and has the added advantage of reducing the opportunity for a missed diagnosis. The routine use of a continuous indwelling gastric siphonage system following major surgical operations will virtually abolish postoperative dilatation of the stomach. When the patient becomes ambulatory or intestinal activity is restored the possibility of this complication developing becomes minimal and suction should be discontinued.

Meteorism is truly the bane of surgeons and methods helpful in its therapy are worthy of emphasis. After intra-abdominal manipulation normal peristalsis is inhibited for periods of up to several days. Any extensive degree of traumatic, chemical or bacterial peritonitis will contribute to the duration of this effect. For instance, a mass ligation technique which leaves behind clumps of necrotic omentum is liable to curtail intestinal activity. Local inhibitory effects are aggravated further by any marked increase in bowel diameter. Accumulations in the gut contributing to the distention are gaseous material of which at least 70 per cent is swallowed air, the unabsorbed residue from the oral intake, plus saliva, gastric and pancreatic juices, bile, succus entericus and bacteria and their end products.

Apparently the predisposing mechanisms

for some degree of ileus are present in every abdominal procedure. The alternatives are either to interrupt the cycle leading to clinical ileus or accept a high incidence of this complication and rely on nature and the patient's durability to cope with it. A nine-holed Wangenstein gastric catheter attached to continuous suction equipment, used routinely throughout the procedure and until intestinal activity is restored two to four days afterward, will regularly avert this complication unless it is caused by peritonitis. The equipment should, of course, be properly serviced and set in working order prior to the operation.

Those with experience in the management of this problem are inclined to agree that it is easier to prevent ileus than to overcome it. In the treatment of adynamic ileus, the Miller Abbott tube, or preferably the Graf ton Smith modification, becomes really effective only after having been passed through the duodenum, a maneuver requiring patience and skill. However in some patients it may be impossible for all practical purposes, to intubate the small intestine. An other drawback occurs once distention is permitted to develop—then the differentiation between inhibitory (adynamic) and mechanical ileus must be made. If localized pain, a mass, rebound tenderness and leukocytosis are present, a strangulating obstruction may be the cause. Pain in the back of sudden onset and with the above signs and symptoms favors this diagnosis.

It can be observed that marked abdominal distention immobilizes and elevates the diaphragm, contributing thereby to a greater incidence of atelectasis and other pulmonary complications. The development of marked abdominal distention also favors venous stasis in the lower extremities. It is a reasonable presumption, therefore, that this state increases the likelihood of phlebothrombosis and, hence, embolic phenomena.

A few cases of postoperative ileus are well-nigh intractable, but most will eventually respond to one or more procedures, including intestinal siphonage, splanchnic block or spinal anesthesia and the use of Pituitrin or Mechohyl. Hypokalemia will induce intestinal atony and hence contribute to the development and perpetuation of ileus. Ambulation, when feasible, is one of the best means of inducing peristaltic activity and will usually prove helpful unless chemical or bacterial peritonitis exists. When no systemic contraindication to an augmented salt intake is present, an enema of

discharge date is perhaps as beneficial a method as we have for routine prophylaxis against these complications. Just why early ambulation appears to be of such little value in preventing venous complications is puzzling, but that this is the case has been concluded from various collected studies.

Ambulation is most effective when started within the first twenty-four hours after an operation, but this schedule should not be inflexible. Those patients who are too ill or enfeebled to get out of bed before a major operation can hardly be expected to do so shortly afterward. It is wisest to modify the regimen so that as the patient assumes an upright position he experiences no serious blood pressure change. A preliminary trial of dangling the feet for a few minutes may indicate the individual's tolerance to resumption of the vertical habitus. Any tendency toward syncope is cause for temporizing and returning the patient to bed then gradually after recovery from that episode, he may again attempt an erect posture. Once he is up he should be encouraged to exercise and then return to bed. If exercise is tolerated, he can try to walk more often and farther each day. At first, he may be unable to attempt more than a few steps in the morning and again in the afternoon.

"Ambulate" is a term meaning "to walk" and it is the surgeon's responsibility to confirm the fact that nurses so interpret this order to the patient. It is an error to consider early ambulation as synonymous with getting the patient out of bed and into a chair as soon as possible after an operation keeping him angulated there any length of time instead of having him up and about. It is an abuse of proper ambulatory management and can cause harm. An elderly patient who falls asleep in the sitting position is prone to orthostatic hypotension. Confusion, disorientation, syncope and concussion, fractures or other forms of severe trauma may result. Early ambulation, properly supervised, is decidedly beneficial and should be an integral part of any comprehensive program for postoperative management.

Fever. Although fever in the postoperative patient may arise from a variety of single factors, certain probabilities are more frequently causative and also more likely to exhibit revealing clues. The patient deserves a careful evaluation of these significant signs and, in the instance of a sharp rise of the temperature line, it should not be construed as ample treatment by the house officer merely to order doubled the dosage and

variety of antibiotics. In a patient with an elevated temperature the more likely indications of its cause will probably be found in the lungs, urinary tract, operative area, veins of extremities including the sites of intravenous therapy and in an abnormal systemic reaction to drugs. The patient with pseudomembranous enterocolitis can also exhibit fever even before the stage of diarrhea.

Fibrogenic complications are more apt to attract clinical attention at certain periods in the postoperative phase. Pulmonary problems (atelectasis, pneumonia) when they do occur are more often noticed within the first twenty-four to forty-eight hours. A bit later urinary tract infection becomes manifest, particularly if the catheter has just been removed or had to be reinserted. Leakage from a suture line is rarely apparent before the fourth or fifth postoperative day. Wound infection and disruption begin to appear on the sixth to tenth days, thrombophlebitis tends to come on about then or slightly later. Drug idiosyncrasies can be the reason for a febrile course at any time, even well into the recovery interval.

A history of burning on urination, frequency and urgency with voiding, and/or renal tenderness points strongly to involvement in the urinary tract. The microscopic examination of a centrifuged fresh specimen is then indicated; a positive urine culture from a catheterized, or midvoided, specimen will clinch that consideration. Also any male patient who is either repeatedly catheterized or wearing an indwelling tube should have a careful genitoretal examination to identify the presence of epididymitis or prostatitis as a cause of fever.

Complete examination of the operative area is awkward sometimes, but this should not serve as an excuse for failing to uncover and carefully inspect the wound itself. Clean surgical incisions should be free of the usual signs of an acute inflammatory reaction. An incision that shows signs of inflammation should be suspected of harboring an infection. Elective incisions should heal kindly and remain free of exquisite tenderness to the sterile gloved, careful, palpating finger. If in doubt, a responsible person should remove aseptically a few sutures and gently part the superficial tissues so as to reveal any purulent loculus. If an abscess is present, adequate drainage will ease the pain from the patient's incision, valuable time will be saved and the morbidity thus shortened. Should the area prove clean on inspec-

contaminate uninvolved visceral and parietal surfaces, thereby increasing the morbidity. With care, only infrequently will it be necessary to use this less desirable avenue to a subphrenic abscess.

A fact about the subphrenic abscess problem which has become increasingly evident is the relatively small contribution that antibiotics have made to a reduction in the mortality from it. The pace of the disease may be slowed and the onset masked to some extent, but few abscesses are cured by chemotherapy alone. In fact, it is questionable that the patient's recovery is materially hastened, after ample drainage, by antibiotics. In general, convalescence remains protracted, while the body slowly absorbs and heals the abscess cavity.

Parotitis. Acute inflammation of the parotid gland, except in association with mumps or secondary to sialolithiasis was more commonly seen in early times as a postoperative complication than it is now. The current liberal use of antibiotics, the giving of fluids orally after an operation and the avoidance of dehydration through better attention to the fluid and electrolyte requirements, all combine to reduce the incidence of parotitis. The most likely candidate for its occurrence is the elderly debilitated, patient undergoing a febrile convalescence, poor in oral hygiene and exhibiting a parched mucous membrane. The parotid lesion is more often initially unilateral, with a subsequent occasional appearance on the opposite side. It is characterized by local tenderness and swelling, some trismus and expressible pus from a swollen, inflamed Stensen's duct. Fluctuation is difficult to elicit because of the gland's fibrous septa and tense capsule and the infection's deep location in the gland. As a rule, early parotitis will respond to antibiotics and to correction of the predisposing mechanisms. Deep x ray or radium therapy to the area gives satisfactory results if abscess formation has not occurred. Early treatment is particularly effective. The development of portable deep x ray therapy units which can be brought to the bedside has increased the usefulness of x ray management. If abscess formation is suspected, incision and drainage parallel to the main fibers of the facial nerve are in order. In a few cases, the condition is fulminant and progresses rapidly unless actively treated. The high mortality ascribed to this condition in the past may well be related to the appearance of this complica-

tion as a terminal incident in very poor risk patients.

Ambulation. Renewed interest has developed in this recommendation made a half century ago of early ambulation. At the time his idea evoked no widespread acceptance and it was only tardily submitted to a limited practical trial by a few clinicians. Now many subscribe to a program for early ambulation. In cases of sepsis, however, conservatism is probably still indicated. In the presence of purulent foci or of widespread infection, any undue activity is more apt to disseminate the process. Rest rather than exercise is indicated until the inflammatory process is under control. Otherwise many leaders in obstetrics, gynecology, neurosurgery, thoracic surgery, orthopedics and general surgery now urge their patients to be up and about soon after major operations. Although there has been a growing acceptance of this idea it remains difficult to identify precisely the contributions of early ambulation to better patient care. Some of the good effects are unquestionably obscured by the benefits from antibiotics, superior anesthetic agents, more liberal transfusions, better nutritional preparation, the more physiologic management of water and electrolyte problems and yet other factors.

Over and above any ambiguous contributions of early ambulation, several considerations, however, appear to have merit. (1) Pain disappears more promptly from the operative site and is usually less exquisite. (2) Certain annoying postoperative sequelae occur less frequently and in a milder form. (3) The patient's morale is better and his psychologic reaction following the operation is healthier. This is of special concern and benefit in care of the elderly patient, for once he is up and about it is easier to arouse him from self pity and discourage him. (4) The incidence of serious complications is not increased. For example, the recurrence rate seems no greater if early ambulation is practiced after herniorrhaphy. (5) The duration of hospitalization after a major operation is shortened, convalescence at home is hastened and, hence the over-all economic loss to the patient is reduced.

The incidence and severity of thrombophlebitis and phlebothrombosis remain approximately the same, unfortunately wrapping the patient's legs, from foot to mid thigh, with elastic bandages before surgery and rewrapping them at least daily until the

- Varco, R. L.: Preoperative Dietary Management for Surgical Patients with Special Reference to Lesions of Stomach and Duodenum. *Surgery* 10:303 1918
- Wangensteen, O. H.: Controlled Administration of Fluid to Surgical Patients. Including Description of Gravimetric Methods of Determining Status of Hydration and Blood Loss during Operation. *Minnesota Med* 25:783, 1912.
- Wangensteen, O. H.: Intestinal Obstructions: A Physiological and Clinical Consideration with Emphasis

- on Therapy Including Description of Operative Procedures, 2nd Ed. Springfield, Ill., Charles C. Thomas, Publisher 1912.
- Wangensteen, O. H.: Care of Patient before and after Operation. *New England J. Med.* 230:121 1917
- Wangensteen, O. H.: The Surgeon and His Trust. *Surg., Gynec. & Obst.* 84:567 1917
- Williams, G. R., Jr., and Spencer F. C.: Clinical Use of Hypothermia following Cardiac Arrest. *Ann. Surg.* 143:468 1938

Management of Fluid and Electrolytes in Surgical Practice

By BERNARD ZIMMERMANN M.D.

BERNARD ZIMMERMANN was born in Minnesota and received his education at Harvard University and Medical School. He spent time in research in endocrinology and metabolism before beginning his surgical training which he received at the University of Minnesota. He has retained his interest in investigations of the relation between endocrinology and electrolyte and fluid balance in surgical patients.

Most of the ventures which have resulted in present-day surgery would not have been successful, or perhaps even attempted, had it not been for the concomitant development of an extensive body of information pertaining to the support of the physiologic needs of patients following extensive surgery. Radical visceral cancer surgery, modern thoracic surgery and cardiovascular surgery exemplify the priority of such fundamental knowledge. Many of the early steps in this field were taken by pediatricians, such as Gamble, who investigated fluid and electrolyte losses from the body in abnormal states and the chemical alterations which losses produce on the composition of the body fluids. These alterations mainly concerned sodium, chloride and bicarbonate and it took another pediatrician to indicate the importance of derangements of intracellular composition. Thus surgeons, following the work of Darrow on potassium losses in infantile diarrhea, soon appreciated the significance of this ion in the correction of postoperative electrolyte deficits. Another

aspect of the development of basic knowledge which is now applicable to the patient following major surgery is the newer understanding of those metabolic effects which accompany all major traumatic incidents to which the human body may be subjected. From the work of Cuthbertson and Albright and the observations made on animals by Selye and his colleagues, the concept has been established that the patient who has undergone major surgery regardless of the type of operation, is vastly different from the normal individual. His need for some substances is far greater, his tolerance for others is less and the problem has become much more complicated than the simple calculation of requirements from the difference between what goes into the body and what comes out.

Physiologic Characteristics of the Postoperative Patient. Trauma of a degree comparable to that represented by major surgical operations results not only in rather characteristic alterations in metabolism but also in gross changes in such tissues as the

tion, the convalescence will not have been materially prolonged the gaping wound edges can either be snugged together with flamed tape or resutured, a local anesthetic being used. Deeper-seated purulent accumulations may reveal themselves in the pelvis upon rectal or bimanual examination. Abnormal aggregates of air and fluid lying just beneath the diaphragm are quite recognizable on upright films of the abdomen. An unusually high diaphragm and pleural effusion may be clues to the existence of this process.

Displaced loops of bowel about a constant area of opacification and in association with a partial small bowel obstruction or clinical ileus are strongly suggestive signs of an intra-abdominal abscess. Localized and abnormal degrees of tenderness over this suspect area will influence one's decision to explore and drain that locus—extraperitoneally if at all feasible.

Thrombophlebitis, other than the traumatic or chemical variety secondary to a venoclysis, is likely to produce significant elevations of the patient's temperature. This complication or phlebothrombosis should be actively and carefully sought for in any person with mild to moderate fever the exact origin of which remains annoyingly obscure. Moreover promptly instituted therapeutic procedures such as decreasing the blood coagulability are in order. Certainly the appearance of a positive Homan's sign, or merely of deep calf pain on palpation and especially if associated with prominent ante-bial veins in a recumbent patient, suggests strongly this diagnosis.

Abnormal drug reactions, although uncommon, work their consequences onto the temperature chart with sufficient frequency to disrupt even the smoothest running service. Whenever the magnitude of the rise in the patient's fever is quite out of proportion to the pulse response and other more likely causes have been excluded, a medication reaction moves from the realm of a possibility to a probability.

READING REFERENCES

- Arhelger, S. W. The Advantages of Tracheotomy and the Use of a New Tracheal Tube in the Management of Intratracheal Aspiration. *Surgery* 29:260 1951.
- Cannon, P. R., Wheeler, R. W., Woodridge, R. L., and Benditt, E. P. The Relationship of Protein Deficiency to Surgical Infection. *Ann. Surg.* 120:514, 1944.
- Clark, J. H., Nelson, W., Lyons, C., and Mayerson, H. S. Chronic Shock: The Problem of Reduced Blood Volume in the Chronically Ill Patient. *Ann. Surg.* 123:610, 1947.
- Coller, F. A., and others. Postoperative Salt Intolerance. *Ann. Surg.* 119:533 1944.
- Crandon, J. H., and others. Ascorbic Acid Economy in Surgical Patients as Indicated by Blood Ascorbic Acid Levels. *New England J. Med.* 258:105 1958.
- Elsman, B., Sifen, W., Bascom, G. S., and Kowman, A. J. Fecal Enema as an Adjunct in the Treatment of Pseudomembranous Enterocolitis. *Surg.* 44:854, 1958.
- Edwards, L. C., and Dunphy, J. E.: *Wound Healing: Injury and Normal Repair*. New England J. Med. 239:224 1958.
- Elman, R. Parenteral Alimentation in Surgery. New York, Paul B. Hoeber 1947.
- Everson, T. C. Experimental Comparison of Protein and Fat Assimilation after Billroth II, Billroth I, and Segmental Types of Subtotal Gastrectomy. *Surgery* 37:525 1954.
- Gamble, J. L. *Chemical Anatomy: Physiology and Pathology of Extracellular Fluid*. Boston, Spaulding-Moss, 1939.
- Hitchcock, C. R., Smith, L., and Varco, R. L. Surgical Applications of an Intra-arterial Transfusion Apparatus. *Surgery* 32:171 1952.
- Houle, D. B., Weil, M. H., Brown, E. G., and Campbell, C. S. The Influence of Respiratory Acidosis on ECG and Pressor Responses to Epinephrine, Norepinephrine, Metaraminol. *Proc. Soc. Exp. Biol. & Med.* 94:581 1957.
- Howard, J. E., and others. Studies on Fracture Convalescence: Nitrogen Metabolism after Fracture and Skeletal Operations in Healthy Males. *Bull. Johns Hopkins Hosp.* 75:156, 1944.
- Kreem, A. J. The Problem of Parenteral Nitrogen Administration in Surgical Patients. *Surgery* 23:92, 1948.
- Lyons, C., and Mayerson, H. S. The Surgical Significance of Hemoglobin Deficiency in Protein Depletion. *J.A.M.A.* 135:910 1947.
- Machella, T. E. The Mechanism of the Post-Gastrectomy Dumping Syndrome. *Ann. Surg.* 130:145, 1949.
- Madden, S. D., and Whipple, G. H. Plasma Proteins, Their Source, Production and Utilization. *Physiol. Rev.* 20:194 1940.
- McClintock, L. S. The Shattuck Lecture: The Patient. *New England J. Med.* 256:1211 1957.
- Miller, F. A., and others. Respiratory Acidosis: Its Relationship to Cardiac Function and Other Physiologic Mechanisms. *Surgery* 32:171 1952.
- Miller, F. A., Brown, E. B., Wangersteen, O. H., and Varco, R. L. Certain Effects in Dogs of Inspiring 15 to 30 Per Cent Carbon Dioxide. *Fed. Proc.* 9:89 1950.
- Price, P. B. Stress, Strain, and Sutures. *Ann. Surg.* 128:408, 1948.
- Reifenstein, E. C. Jr. The Rationale for the Use of Anabolic Steroids in Controlling the Adverse Effects of Corticoid Hormones Upon Protein and Osseous Tissues. *South Med. J.* 49:933 1956.
- Schoenheimer, R. *Dynamic State of Body Constituents*. Cambridge, Mass., Harvard University Press, 1942.
- Smith, G. A. A Study of Intestinal Intubation Using a Flexible Stylet with Controllable Tip. *Surgery* 32:17 1952.

mains obscure. It is known that unlike the so-called glyccorticoids aldosterone does not require the presence of the pituitary for its mobilization. Nevertheless, under appropriate circumstances ACTH can cause aldosterone to be secreted so that the action of ACTH can be described as a sufficient but not necessary factor for the release of salt-regulating hormones. Sodium restriction causes a greatly increased output of aldosterone but it is probable that the effect of sodium restriction is mediated through changes in circulatory volume rather than reduction in the sodium levels. Reduction of sodium concentration by itself apparently has nothing to do with aldosterone release.

The work of Bartter would indicate that the most important single mechanism is reduction in blood volume and more specifically the volume of blood which is perfusing the arterial circulation. Further sodium retaining hormone is mobilized through the activity of some type of "volume receptors" activating hypothalamic centers which, in turn through a separate trophic mechanism, stimulate the appropriate portions of the adrenal. The resulting retention of sodium is a mechanism for protecting the circulating volume. It is not entirely clear however that all operations which are followed by an increase in aldosterone output and sodium retention involve any threat of reduction of blood volume or cardiac output. Therefore, other mechanisms must be considered. It has now been shown, for example, that a rise in serum potassium stimulates the adrenal gland to secrete aldosterone and, since potassium mobilization is a rather constant concomitant of major surgical procedures and other forms of tissue injury we must consider that this may be an important underlying mechanism which is mobilized in order to aid the kidney in clearing the circulation of an excessive potassium load.

Although it appears that the role of aldosterone must be of great importance in the regulation of mineral balance immediately following operations it is by no means clear that this substance is totally responsible for the over-all change in mineral metabolism which occurs in the postoperative course. Aldosterone, as measured in the urine of these patients, appears early in the postoperative course and almost uniformly returns to basal levels in two to three days. Positive sodium balance, on the other hand, may persist for six or seven days and almost regularly obtains after the aldosterone levels are normal.

This picture of an early rise followed by a rapid decrease in aldosterone in the face of more prolonged positive sodium balance is illustrated by the course of the patient shown in Figure 1. The investigation of other salt regulating hormonal mechanisms is therefore being actively pursued at the present time. Clearly the release of aldosterone is not caused by a single stimulus for which surgery acts as a trigger but is the result of a complicated interplay of factors involving the circulatory system and the central nervous system, as well as the changes in electrolyte concentration which result from injury to tissues.

It is likely that none of the metabolic responses to surgery are exclusively regulated by the endocrine system and it must be remembered in this connection that hormones in general only catalyze and do not originate metabolic and membrane-transfer phenomena. There is a considerable body of evidence suggesting that in both man and animals devoid of adrenal glands but maintained on constant amounts of cortical replacement, nitrogen excretion, sodium retention and potassium loss can all be induced by the imposition of nonspecific stress. This strengthens the notion that the underlying pathways for these reactions exist in the basic enzymatic machinery and do not depend exclusively on an acceleration in en-

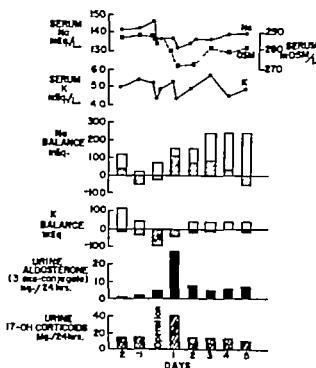


Figure 1 The course of the blood levels and balance of electrolytes as related to the urinary excretion of aldosterone and 17-hydroxycorticoids following a typical operation.

formed elements of the blood and the lymphatic system, the thymus and the adrenal glands. Nitrogen and carbohydrate balance are altered so that blood sugar tends to be elevated, glucose tolerance is decreased, glycosuria is not uncommon and, in instances in which marginal glucose tolerance exists transient or even permanent diabetes may be precipitated by a major surgical procedure. Along with this the balance of nitrogen becomes negative as measured by an increased output of nitrogen in the urine and a relative inability of the organism to utilize exogenous amino acids for the synthesis of structural body proteins. Sodium and chloride tend to be withheld from the urine and, in the immediate postoperative phase, urinary concentrations of these two ions may drop to almost zero even when the amounts given are greater than that normally necessary for daily replacement. Urinary potassium concentrations, on the other hand, are regularly elevated. Potassium balance becomes negative and that this urinary excretion of potassium is not entirely the result of tissue breakdown can be proved by demonstrating that the potassium-to-nitrogen ratio of the urine is greater than that which characterizes the interior of the cell.

The postoperative patient is frequently oliguric, even in the absence of preceding shock or other factors which might be expected to predispose to disturbances of renal function. More consistently than this, however, postoperative patients display intolerance to water. This implies that their diuretic response to a water load is impaired so that they excrete excess water sluggishly and may suffer the consequences of dilution or hydropemia before the kidneys are able to respond to an excess of administered fluid.

These metabolic alterations are associated in patients following surgery and in experimental animals following trauma, with certain morphologic changes including depression of the number of lymphocytes and the eosinophils in the blood, involution of the lymphoid tissues such as the thymus and enlargement of the adrenal cortices. The underlying factor in these morphologic changes and certain of the metabolic effects is the production of an increased amount of hormones by the adrenal cortex. It must be recognized, however, that no single mechanism is likely to be responsible for all of the metabolic phenomena which characterize the response to surgery. It is likely that these reactions have a definite protective effect on the economy of the organism

although the nature of this protection remains largely obscure. Most investigations of this subject have centered around the activation of the pituitary-adrenal system in response to stress. It is very clear that the pituitary rapidly secretes an increased amount of adrenocorticotrophic hormone to which the adrenal responds with the output of one or more steroid hormones with potent metabolic activity. Furthermore the anterior pituitary is stimulated by the activation of certain centers in the hypothalamus, the relationship of which to the hypophysis appears to be a humoral rather than a neural one. It is likely that more than one mechanism is responsible for the discharge from these hypothalamic centers.

At least three types of hormones are known to be produced by the adrenal cortex. As defined by their function rather than their chemistry these include the glucocorticoids which are responsible for regulation of carbohydrate, protein, and metabolism; the androgens which in addition to masculinizing function cause positive nitrogen balance, and mineralocorticoids which are predominantly concerned with the regulation of sodium and potassium balance. That substances capable of profoundly altering organic metabolism are mobilized by the adrenal gland following surgery has been demonstrated by the finding of increased hormones of this type in the postoperative urine as well as the presence of 11-oxo-17 hydrocorticoids in the blood. The consistent occurrence of eosinopenia following surgery also suggests the presence of this same type of hormone although less significance can now be attached to the fall in eosinophil count than was suggested by the early experiments relating eosinophil depression to adrenal cortical activity.

The endocrine mechanisms for postoperative alterations in sodium and potassium regulation are still not completely understood. Although the relationship of 11-oxo-17 hydrocorticoids to alterations in organic metabolism has been recognized for a long time, the endocrine substance responsible for regulation of sodium and potassium balance was not known until the recent discovery of aldosterone and the demonstration of its presence in various pathologic states associated with sodium retention.

The recent findings of increased amounts of this hormone in the urine of postoperative patients leaves no doubt that it is mobilized by surgery. The intermediate mechanism, however, which results in the stimulation of aldosterone production by the adrenal re-

electrolyte physiology must take into account all the major fluid spaces and the nature of the boundaries which separate them.

The extracellular fluid is composed of the blood plasma and the interstitial fluid. Values for this volume vary from 17 to 20 per cent of the body weight depending upon the method used for determination. More recent methods give lower values. Older determinations indicated that the total body water was about 70 per cent of the body weight. Measurements using deuterium oxide suggest average values of around 61 per cent for men and 52 per cent for women.

The blood plasma and interstitial fluid are essentially the same in composition except for the difference in protein concentration which results from the relative impermeability of the capillary to these large molecules. The composition of the cell fluid, on the other hand, differs radically from that of the extracellular environment. Whereas the sodium is the major extracellular cation, potassium occupies this position in the cell where phosphate is the predominant anion. Despite this, it is not true as was once believed that sodium is completely excluded from the cell. A definite amount of sodium ion constitutes a portion of the intracellular ionic structure and there is a great deal of evidence that this quantity may be increased under circumstances of injury and disease. It must also be recognized that the relative exclusion of sodium from the cell is not a matter of permeability of the cell wall. Individual sodium ions have been shown by tracer techniques readily to permeate the cell membrane, showing that maintenance of an unequal gradient is not a matter of permeability but an active process requiring the expenditure of metabolic energy. The perpetuation of these unequal concentrations is therefore, a function of normal cells and a characteristic of life as significant as the utilization of oxygen and the production of carbon dioxide. It is typical, moreover of "sick cells" that these gradients tend to break down and the intracellular concentrations approach those of the surrounding medium.

The values mentioned above for the volumes of the intracellular and extracellular compartments describe the situation in the normal individual. It is of some importance to consider the factors which regulate these volume relationships. The critical quantity in this regard is the extracellular sodium, for the intracellular volume does not depend

on the available water of the extracellular space but rather its total ionic concentration. Since sodium exists in largest concentration the level of this ion is the most important controlling factor. A decrease in sodium ion concentration results in a movement of water into the cells with consequent enlargement of the intracellular volume, and similarly an increase in sodium concentration results in cellular dehydration. Such changes in cell water have profound functional implications and the central nervous system is particularly sensitive to them. Consequently changes in tonicity underlie many of the clinical manifestations for which deviations of water and electrolyte balance are responsible.

Requirements of Surgical Patients. The initial consideration for fluid replacement is the basal requirement for the human being who for reasons of disease or surgery is denied the oral route of alimentation. The minimal quota for an adult must take account of an insensible loss through the skin and lungs ranging from 600 to 1000 cc. and an anticipated urine output of 1000 to 1500 cc. Insensible losses may of course, be far greater than this, particularly in the circumstances of fever or prolonged surgery under heavy cloth draping.

It must also be appreciated that exogenous water is not the sole source of fluid for extracellular hydration in seriously ill patients. Moore has pointed out that in severe injury or chronic illness as much as 1000 cc. of water can be mobilized daily from endogenous sources. This is made up of cell water (about 700 cc. per kg. of lean tissue) oxidation of protein (about 150 cc. per kg.) and of fat (1080 cc. per kg.). Accurate studies on patients with anuria, in whom the control of water administration must be extremely precise have shown that as little as 400 to 500 cc. of exogenous water may sometimes be required.

The factors of insensible loss and endogenous water are not only variable but highly unpredictable and this is the reason for the unique value of the body weight in assessing states of hydration. In addition, therefore to measure accurately the fluid intake and output, surgical patients should be weighed daily on a balance which is accurate to 0.1 kg. and the weight recorded on the chart along with the fluid balance data. In patients too ill to stand on a scale, this can be accomplished by a balance which utilizes a litter upon which the patient is lifted from his bed.

doctrine function. Nevertheless, the importance of the endocrines for preservation of life under circumstances of systemic stress is adequately demonstrated by the almost uniformly fatal results of even minor operations inadvertently carried out on patients with adrenal insufficiency.

A seeming paradox exists in the fact that during the period following extensive surgery when sodium and chloride are rigorously withheld from the urine plasma levels of these ions are commonly reduced. The question as to whether this reduction of plasma levels represents only the addition of salt free water which is not excreted, or must be explained in part by some presently undefined sequestration of sodium and chloride ions, remains debatable. A more significant fact, however, is that in patients following operations the administration of salt free solutions such as 5 per cent glucose, is followed by only sluggish diuresis and by reduction of plasma electrolyte concentrations. Administration of the same amount of glucose solution to normal individuals, or preoperative patients results in almost no change in the plasma concentrations. Various lines of evidence suggest that this diminution of urine output in the early postoperative course is related to antidiuretic activity of posterior pituitary origin.

In the normally hydrated patient, the surgical operation produces physiologic effects similar to those of Pitressin: urine flow decreases, urine osmolality increases and serum osmolality decreases as does the "free" water clearance (C_{H_2O}) which is defined as the difference between urine flow (V) and the osmolar clearance ($\frac{U_{osm}}{P_{osm}} V$), so that

$$C_{H_2O} = V - \frac{U_{osm}}{P_{osm}} V$$

This value is negative when water is being conserved at the expense of solute.

It is important to realize that these observations regarding water and solute output following surgery are not by themselves unequivocal evidence of antidiuretic activity. They can only be so interpreted if glomerular filtration is shown to be either constant or increasing, a circumstance which has not been met in most studies of the subject. Recent experiments have shown, for example, that even in the animal with experimental diabetes insipidus, a hypertonic urine can be produced by restriction of renal blood flow and glomerular filtration. The

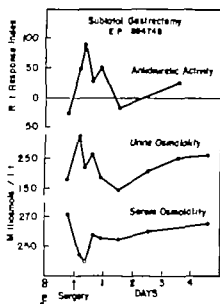


Figure 2. The course of serum and urine osmolality as related to excretion of biologically measured antidiuretic hormone excretion following a typical operation.

importance of serious alterations in renal blood flow following major surgical procedures cannot be disregarded.

The second source of evidence for antidiuretic activity derives from the measurement of the antidiuretic potency in experimental animals of polypeptides extracted from the urine. Though this biologic assay also has both practical and theoretical drawbacks, no chemical determination is presently available. Despite the limitations of both approaches, the weight of evidence indicates that even slight trauma is an important stimulus for antidiuretic activity and that this stimulus can act without regard to the normal "osmolar receptor" mechanism through which the tonicity of the extracellular fluid is normally controlled. This produces a paradoxical situation of increasing antidiuretic activity occurring while the plasma is becoming increasingly dilute. The course of biologically measured ADH excretion and the urine and plasma osmolality following a typical surgical operation is illustrated in Figure 2.

Fluid Compartments: Their Boundaries and Constituents. The tendency of clinicians to describe abnormalities of fluid and electrolyte physiology in terms of plasma values results only from the relative simplicity with which a sample can be drawn from the circulating blood. Although blood values are obviously of great importance, any rational consideration of abnormalities of fluid and

when lost from the body in significant amounts. Bile is more alkaline than plasma though its sodium-to-chloride ratio is not nearly so high as that of pancreatic juice. Intestinal fistulae, except for high ones tend also to produce acidosis because of relatively greater sodium losses. Most aspirates from tubes and fistulae are of mixed, and frequently of undetermined origin and it may be occasionally necessary to make actual measurements of solutes in the secretions in order effectively to provide for their replacement.

For common situations, average values for gastrointestinal fluids based on previous experience are adequate and Table 1 and Table 2 from the work of Lockwood and Randall present some mean concentrations of sodium, potassium and chloride for typical secretions. Reference to these tables is useful in approximating the requirements in average cases. To replace the acid secretion from a normal stomach, it is usually only necessary to use sodium chloride solutions even though this may entail giving an excess of sodium ion. Since a normal kidney excretes the sodium acid salts such as ammonium chloride are rarely necessary except when serious distortions of body chemistry have become established. However in the management of pancreatic, biliary and intestinal fistulae where sodium loss predominates replacement cannot be achieved by the use of sodium chloride alone and alkaline solutions such as sodium bicarbonate or lactate are necessary. In this connection, it will be recalled that the plasma is normally alkaline. The administration of sodium chloride alone is therefore an acidosis-producing procedure although the kidney can normally compensate for this effect.

The question of replacement of magnesium ion and the rare occurrence of magnesium deficiency syndromes are matters of considerable current interest. Although the intake of magnesium in the average diet

is said to be around 300 mg. per day the normal individual appears able to conserve this ion effectively in the presence of deficient intake. Nevertheless the magnesium content of gastric aspiration fluid is 10 to 20 mEq. per liter and there is some evidence that magnesium behaves partly like potassium with respect to adrenal function in that mineral regulating corticoids accelerate its excretion. One would expect, therefore, that major surgery particularly gastrointestinal surgery would present a threat to the magnesium stores. Unfortunately few balance studies directed toward elucidating the behavior of magnesium in response to surgery have been made, largely because available methods have not been sufficiently precise for reliable determinations on body fluids other than serum. Clinically or chemically proved cases of magnesium deficiency are rare but occasional instances are seen of clinical magnesium deficiency presenting as neuromuscular irritability, disorientation, fibrillary twitchings, tremor and even convulsions, which respond to magnesium therapy. If these clinical signs appear following long periods of parenteral maintenance and are associated with blood levels below the normal range of 1.5 to 2.5 mEq. per liter parenteral administration of magnesium ion is indicated.

Disturbances of Acid Base Equilibrium. Clinical disturbances of acid base balance may be of either metabolic or respiratory type. Whereas in the past, metabolic disturbances were considered to be vastly more frequent, the recent growth of thoracic surgery and the more intensive study of inhalation anesthesia have served to emphasize the comparative frequency with which respiratory disturbances of acid base regulation actually occur in surgical patients. Consequently it is of great importance that in evaluating cases of acidosis or alkalosis the necessary data be available to demonstrate clearly what the underlying basis for the

Table 2 Sodium, Potassium and Chloride Concentration of Bile and Pancreatic Juice
(Milliequivalents per Liter)*

		Na	K	Cl
Bile	Average	145.3	5.2	99.9
22 specimens	Range	122-164	3.2-9.7	77-127
12 patients	2/3 Cases	134-156	3.9-6.3	83-110
Pancreas	Average	141.1	4.6	76.6
3 patients	Range	113-153	2.6-7.4	54-95.2

Table 1 Values for Content of Sodium, Potassium and Chloride in Gastrointestinal Tract Losses (Milliequivalents per Liter)*

		Na	K	Cl
Gastric (Fasting) 130 specimens	Average	59.0	9.3	89.0
	Range	6.0-157	0.5-65.0	13.2-167.2
	2/3 Cases	31.0-90.0	4.3-12.0	52-124
Small bowel (Miller-Abbott suction) 89 specimens	Average	104.9	5.1	98.9
	Range	20.1-157.0	1.0-11.0	43.0-156.1
	2/3 Cases	72-128	3.5-6.8	69-127
Ileum (Miller-Abbott suction) 17 specimens 7 patients	Average	116.7	5.0	105.8
	Range	82-147	2.3-8.0	60.7-137.0
	2/3 Cases	91-140	3.0-7.5	82-125
Ileostomy (Recent) 25 specimens 7 patients	Average	129.5	16.2	109.7
	Range	92-146	3.8-98.0	66-136
	2/3 Cases	112-142	4.5-14.0	93-122
Cecostomy 20 specimens 9 patients	Average	79.6	20.6	48.2
	Range	45-135	3.7-47.3	18-88.5
	2/3 Cases	48-116	11.1-28.3	35-70

From Randall, H. T. Water and Electrolyte Balance in Surgery. S. Clin. North America 32:457, 1952.

The use of body weight in assessing fluid balance is of course based on the assumption that significant changes over short periods are largely the result of changes in hydration. This assumption is true but, of course, is not valid over periods of many days in chronically ill patients in whom cumulative decrements in tissue mass are occurring. Furthermore, it should be kept in mind that large amounts of fluid may be sequestered temporarily in areas of the body which play no role in the maintenance of circulation or the state of hydration. This so-called third space effect occurs to some extent following any operation or injury but assumes significant proportions where there are extensive areas of tissue damage or localized edema peripheral to sites of venous or lymphatic obstruction or in intestinal ileus. It would be absurd, for example, to calculate the fluid requirements for a patient with a major burn on the basis of the changes in body weight, when his fluid losses are almost entirely within his own body.

Large amounts of sodium chloride are not required for maintenance of patients who do not suffer from external losses. The normal kidney can restrict sodium excretion to much less than that contained in 1 gm. of sodium chloride (17 mEq.) and the insensible water loss excluding palpable sweat contains only traces of sodium salt. This ability

of the kidney to conserve in the absence of intake does not hold for potassium ion, since 30 to 40 mEq. of potassium may be excreted by patients receiving none by oral or parenteral route. It is necessary therefore, to include in a parenteral regimen at least 2 to 3 gm. of a salt such as potassium chloride for routine maintenance. This, however, should not be given immediately after surgery or until normal renal function has been adequately established. The losses of potassium may of course be much greater following the stress of major surgery, burns and trauma. In addition to the basal amounts of fluid to provide for urine output and to replace insensible losses parenteral administration must include volume-for-volume replacement of water lost through the abnormal routes of tubes and fistulae. The replacement of solutes lost through these channels must take into account the origin of the secretions, for the ratio of sodium to chloride in various gastrointestinal fluids differs greatly from that of plasma. Their loss will produce not only over-all electrolyte depletion but also profound disturbances in acid base equilibrium. Thus withdrawal of normal gastric juice which contains more chloride than plasma and very little sodium rapidly produces alkalosis. Conversely pancreatic juice which possesses a sodium concentration comparable to that of plasma but relatively little chloride causes acidosis.

was originally described by Darrow as a complication of infantile diarrhea. It is now seen at least as frequently in association with high intestinal obstruction and particularly in patients who have had prolonged parenteral maintenance without adequate amounts of potassium ion (Fig. 4). The increased extracellular bicarbonate under these circumstances is related to the high intracellular sodium which results from the movement of the latter ion into the cell as a mechanism for replacing the lost intracellular cation. A persistent hypochloremic alkalosis which is typical of the situation can be reversed only by the administration of potassium ion. The diagnosis is usually made merely by the presence of so-called refractory alkalosis. Serum levels of potassium are usually low although they need not always be. The characteristic electrocardiographic findings—prolongation of the Q-T interval, depression of the R-S-T segment and eventually inverted T waves—are valuable confirmatory signs.

The nature of the physiologic state which

follows surgery and the stress associated with serious surgical diseases explain the frequency of potassium deficiency as a surgical complication. Reference to Table 1 will indicate the amounts of potassium in milliequivalents per liter of drainage that may be lost from the body through gastrointestinal siphonage. Even when gastrointestinal siphonage is employed however the loss through the gastrointestinal tract is usually not the largest source of negative potassium balance for even under normal circumstances an individual receiving no potassium by mouth may put out large amounts of urinary potassium and under circumstances of stress imposed by a major surgical procedure or acute intestinal obstruction, these urinary losses may be greatly increased.

Although in discussing mechanisms we tend to distinguish metabolic alkalosis resulting from gastric chloride loss from that associated with the chronic depletion of intracellular potassium, these two conditions are peculiarly interrelated and in surgical patients probably never exist as separate entities. Not only does high intestinal obstruction result in depletion of potassium stores through vomiting or aspiration, representing a serious form of stress which results in potassium loss in the urine, but alkalosis itself causes depletion of intracellular potassium and excretion of this ion in the urine. In such circumstances metabolic alkalosis is almost always accompanied by an acid urine, the so-called paradoxical aciduria of metabolic alkalosis. This apparently results from avid retention of sodium bicarbonate by the kidney, a phenomenon which is characteristic of potassium deficiency and mineralocorticoid activity on the part of the adrenal gland.

Thus the kidney plays no role in compensation for this abnormality of acid base balance and only potentiates the defect through the retention of sodium and bicarbonate. A three-cornered self-perpetuating cycle involving alkalosis, endocrine activity and potassium loss becomes established which can only be interrupted by the administration of potassium ion. The extent of depletion of body potassium reservoirs may be relatively small or very great and is not readily measurable by the usual criteria of plasma levels or electrocardiogram. Chloride deficit is also large and, with bicarbonate retention, chloride losses in the urine persist despite continued depletion through vomiting or aspiration. Since some loss of sodium

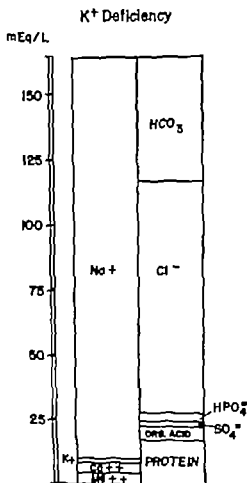


Figure 4. Hypochloremic alkalosis resulting from potassium deficiency in a patient following prolonged parenteral therapy without potassium replacement.

clinical disturbance in pH is. In most institutions, a measurement approximating the plasma bicarbonate such as the carbon dioxide combining power is used for clinical purposes. Reliance on this value alone is frequently adequate if it is kept in mind that this quantity is by itself no index of the direction of the pH change. It will be recalled in this connection that the pH maintained by the buffering system of the plasma is related to the bicarbonate of the blood and the dissolved carbon dioxide, or carbonic acid, by the familiar Henderson Hasselbalch equation

$$pH = pK + \log \frac{BHCO_3}{H_2CO_3}$$

The bicarbonate of the plasma can be altered by changes in its rate of renal excretion and the carbonic acid concentration depends on the rate at which carbon dioxide is removed by the lungs. By their individual control of these two quantities the lungs and kidneys are the organs primarily responsible for the preservation of normal pH in the face of circumstances tending to disturb it. In deviations of a primary metabolic nature, the initial effect is on the bicarbonate which is decreased in acidosis and increased in alkalosis. The respiratory apparatus responds to shift the carbonic acid in the same direction so that the ratio $\frac{BHCO_3}{H_2CO_3}$ which is

normally 20:1 is minimally altered. Respiration is therefore, accelerated in acidosis and depressed in alkalosis although the latter adjustment is minimal and usually not clinically apparent. In disturbances of a primary respiratory nature, however the initial effect involves the carbonic acid. Compensations must be made by the kidneys which retain or excrete bicarbonate. They retain bicarbonate when carbonic acid has been inadequately removed by the lungs and they excrete an excess of bicarbonate to compensate for the alkalosis which follows hyperventilation. Therefore, since the final bicarbonate value, which is the most commonly used index for acid-base disturbances, is elevated in metabolic alkalosis and respiratory acidosis and depressed in respiratory alkalosis and metabolic acidosis, it is obviously not possible to distinguish between acidosis and alkalosis on the basis of the carbon dioxide combining power alone. In most instances the clinical information points to a disturbance which is primarily of metabolic or of respiratory origin. Occasionally however the situation is not clinically

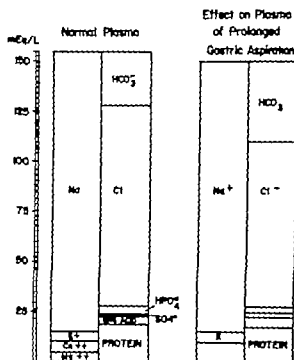


Figure 3 Hypochloremic alkalosis. Effect of several days of nasal tube suction, without adequate chloride replacement, on the composition of the plasma. Note reduction of chloride with expansion of bicarbonate and essentially normal sodium.

cally obvious and the complete acid base picture must be obtained by establishing a value of a second of the three possible variables in the Henderson-Hasselbalch equation. This is done ordinarily by determination of the blood pH and preferably with the simultaneous measurement of the total carbon dioxide content of the arterial blood.

Metabolic alkalosis is most commonly seen in surgical patients as a result of the loss of large amounts of gastric secretion, a material which has high chloride content with relatively low concentrations of sodium. It is ordinarily lost either through vomiting or continuous infusing gastric suction. Alkalosis can develop in as brief a time as twenty-four hours in the face of total diversion of gastric content. Figure 3 illustrates the ionic pattern in such a case compared with the normal concentration. As was mentioned above, this simple situation can be corrected by administration of chloride ion in the form of sodium chloride and acid salts are rarely indicated.

A more complicated situation leading to metabolic alkalosis is that associated with potassium deficiency. It is most frequently recognized under circumstances in which repeated administration of sodium chloride or even ammonium chloride results in no alleviation of metabolic alkalosis. Though it

sunken eyes and dryness of the mucous membranes. Change in body weight is the best index of the development of dehydration. Serum electrolyte concentrations can be elevated, depressed or normal and therefore give no indication of the over-all deficit of ionic components. If a patient admitted to the hospital has been vomiting or has suffered other perceptible fluid losses it must be remembered that if he has not been drinking, water loss almost invariably exceeds electrolyte loss so that it is best to begin hydration with solutions which are dilute with respect to ionic constituents. Subsequent blood chemical determinations frequently reveal the presence of electrolyte deficit as a more normal state of hydration is approached.

Edema in association with surgery was frequently seen in earlier days when "physiologic saline" was used routinely as a hydrating solution. After the recognition by Collier and his associates of the tendency for patients to retain sodium in the period after surgery, excessive use of sodium chloride was almost universally discontinued. Postoperative edema became far less common. In this situation, as with dehydration, the serum levels of ions do not, of course, give any index of the over-all excess of extracellular electrolytes which may be present. The familiar clinical signs of pulmonary and peripheral edema are obviously of the greatest importance and serial body weight determinations will demonstrate incipient fluid retention long before clinical edema is apparent.

Disturbances of Concentration. Reference has already been made to the importance of the sodium concentration of the extracellular fluid and its critical role in the distribution of water between the cells and the fluid which surrounds them. Because of the importance of this concentration, it is not surprising that the normal sodium level is maintained within rather narrow limits. Nevertheless, following surgery and in the absence of oral intake, elimination of the factor of thirst and with certain impairments in renal function, disturbances in the regulation of sodium concentration do occur. As has already been pointed out, such a deviation represents in effect an abnormality in the over-all effective osmotic properties of extracellular fluid.

It was stated above that, following major operations, the sodium concentration of the plasma tends to drop despite the fact that sodium balance inclines to be positive. This

situation results from an intolerance of the postoperative patient to administered water with subsequent dilution and possibly also from factors having to do with translocation of the sodium ion possibly into other reservoirs which are at present poorly understood. Although this frequent depression of sodium concentration is not ordinarily accompanied by symptoms, it occasionally is severe enough to cause profound functional disturbances. The most dramatic of these involve the central nervous system which reflects the depression of sodium concentration and reacts to intracellular shift of water by the physical signs of stupor, irrationality, neuromuscular phenomena and sometimes convulsions. Depending on the circumstances leading to such clinical symptoms, they are referred to as "water intoxication" or "low sodium syndrome." The most profound effects are produced when an injudicious amount of water is given to a patient within the first two to three days after surgery. Nevertheless, cases of so-called water intoxication have been observed under circumstances in which entirely appropriate amounts of water have been administered (Fig. 6). The low sodium syndrome has been particularly observed following the operation of valvuloplasty for mitral stenosis. The cardiac impairment in these individuals prior to surgery creates an even greater intolerance to administered water than the usual surgical patient exhibits. It is of great interest that the total measurable sodium in such patients actually tends to be high as does their total extracellular space. This further emphasizes the fact that it is the concentration of the sodium in the plasma which is of the greatest importance in regard to the cellular content of water.

Most patients with mitral stenosis have, of course, been prepared for surgery with low sodium diets and mercurial diuretics. Nevertheless, it does appear that they have an even greater susceptibility to postsurgical complications of dilution and hyponatremia than other patients so prepared. They behave as though under an extremely potent antidiuretic stimulus and the reason for this is not entirely clear. With low sodium levels, antidiuresis should be inhibited. However, there is a great deal of evidence that the antidiuretic response is sensitive to volume as well as osmolar stimuli and there is some evidence to indicate that the volume receptors for posterior pituitary antidiuresis are located in the region of the left atrium or pulmonary veins. It is tempting to suppose

has also occurred through gastrointestinal routes. It is frequently justifiable to initiate therapy with a combination of sodium chloride potassium chloride and ammonium chloride with subsequent adjustment in the proportions of these components as dictated by the changes in plasma electrolyte levels.

Acidosis of metabolic origin is frequently seen following the loss from the body of secretions possessing high sodium concentration. The typical case is that of pancreatic fistula. Pancreatic juice which possesses a sodium concentration comparable to that of plasma but very little chloride is almost an isotonic solution of sodium bicarbonate. Diversion of this solution from a normal path of being secreted and reabsorbed from the intestine results very rapidly in profound acidosis with a blood picture of low sodium and low bicarbonate values. The electrolyte picture resulting from a chronic pancreatic fistula is illustrated in Figure 5. A similar situation is of course produced by sodium loss through fistulae from the lower intestine where the sodium to-chloride ratio is also high. Reversal of the blood electrolyte picture in these situations cannot be ordinarily accomplished with sodium chloride, and the use of sodium solutions with labile or metabolizable anions such as sodium lactate or sodium bicarbonate is ordinarily required.

The examples of acidosis just described result from the loss of the extracellular cation or what was referred to in the older clinical terminology as fixed base. Another type is the result not of loss of base but of accumulation of abnormal acids which results in displacement of bicarbonate. An example of this is the acidosis which accompanies uremia with accumulation of phosphate, sulfate and organic acids. Another is the picture which results from the excessive reabsorption of chloride from the intestine following surgical procedures in which the ureters are transplanted into the intestinal tract. This so-called chloride acidosis frequently follows such procedures but is more likely to exist when drainage of the intestinal segment is inadequate or when poor renal function exists in addition to discharge of urine into the intestine.

Respiratory acidosis associated with anesthesia and thoracic surgery has become a matter of increasing significance in recent years. It is important to recognize that the administration of oxygen to individuals with respiratory impairment resulting from pulmonary disease or inadequate pulmonary tissue or impairment of motor respiratory

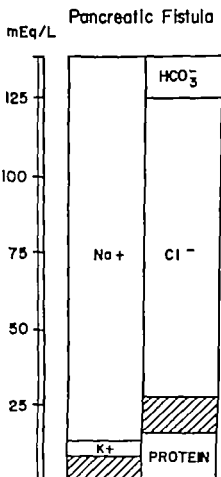


Figure 5 Hyponatremic acidosis. The result of chronic sodium loss through a pancreatic fistula. Note reduction of sodium with corresponding contraction of bicarbonate. Cross-hatch indicates normal values assumed (cations) or determined by subtraction (anions).

activity will maintain oxygen saturation of the blood but may not achieve adequate removal of carbon dioxide. The consequences of respiratory acidosis in the genesis of cardiac arrhythmia and cardiac arrest are profound.

Disturbances of Fluid Volume. A consideration of daily importance in the management of postoperative patients is the maintenance of normal extracellular fluid volume. This quantity which is very sensitively regulated in the normal individual, is readily subject to distortion when the oral intake or the effect of thirst, is removed and when renal regulations of fluid volume may be impaired. Dehydration in the strict sense refers to the effects on the body of water loss alone. It most frequently is used, however, to describe the deficiency of water in combination with electrolytes. Pure water dehydration is actually rare. The familiar signs of combined extracellular fluid deficiency include loss of turgor of the skin

Ischemia resulting in varying degrees of necrosis of renal cells is histologically most frequently demonstrable in the distal tubular epithelium. It is not at all clear, however, that those cells which can be seen to be damaged under a microscope are the only ones which are functionally impaired. The important fact is that if one follows the changes in the damaged tubular epithelium, regeneration begins to occur between eight to fourteen days and, if the patients can be maintained in adequate fluid and metabolic equilibrium until that time, the majority can survive such periods of severe reduction in renal function. In the older literature many instances of survival from periods of prolonged anuria were recorded with little specific therapy. Unfortunately, when the use of parenteral fluids and electrolytes became widespread, the overzealous use of these agents resulted in the death of many such patients. In recent years a number of methods of artificial dialysis, the most successful being the Kolff artificial kidney, have come into use. Although these have a definite field of usefulness, the mainstay of treatment in the majority of patients with acute renal insufficiency is cautious and quantitative fluid management.

Anuria is arbitrarily defined as a urine output of less than 100 cc. in twenty-four hour periods. Characteristically anuric patients have certain electrolyte deviations. In cases as they are ordinarily seen, both sodium and chloride in the extracellular fluid tend to be low (Fig. 7). The main reason for this is that no matter how early the condition is recognized the patient will have been given, or have ingested, water which has not been excreted. Consequently a certain amount of overhydration exists from the start. In chronic renal insufficiency the inability of the kidney to conserve sodium may play a role in the genesis of this diluted picture but is obviously of no importance in the completely anuric individual. In addition to the alterations in sodium and chloride, bicarbonate is almost uniformly reduced. This indicates metabolic acidosis resulting from a combination of the depressed serum sodium level and an abnormal accumulation of anionic metabolites such as sulfate, phosphate and organic acids.

Fluid administration must be limited to that which is required to replace insensible losses. In addition to accurate recording of intake and output, daily determination of the body weight is absolutely necessary because of the great variation in insensible

water loss among individuals. It will be found by such measurements that most of these patients should be given only from 500 to 750 cc. of fluid daily to replace their water loss and sometimes as little as 300 to 400 cc. Very rarely is more than 1000 cc. required. If suction tubes or fistulae are present, the loss through these channels must be added to the above. Within reason it appears definitely valuable to take what measures are necessary to maintain as normal as possible a composition of the extracellular fluid. This should be done only when it is possible to add the necessary components without exceeding the rigid quota of fluid established by the insensible water loss. Therefore particularly when the sodium level is low it is advantageous to use small amounts of sodium bicarbonate or sodium lactate to correct the metabolic acidosis. Similarly when sodium and chloride deficiencies clearly exist and are not the result of overhydration appropriate amounts of these ions should be given. Frequently this must be done by resorting to hypertonic solutions in order not to increase over all fluid intake. The only deviation which cannot be dealt with in this manner is a

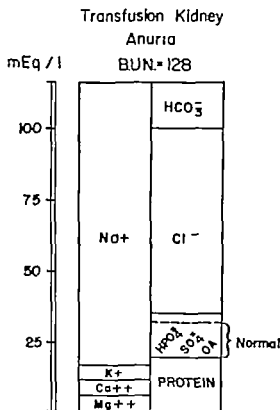


Figure 7 Effect on serum electrolytes of acute anuria. Note reduction in total base as well as chloride and bicarbonate and extension of anions normally excreted by the kidney (phosphate sulfate and organic acids).

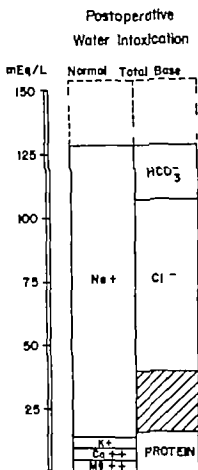


Figure 6 Water intoxication occurring thirty hours after operation (colectomy) in an elderly woman with cardiac disease who had been maintained on a low sodium regimen prior to surgery

that the posterior pituitary has become adapted to a barrage of inhibitory stimuli from a distended left atrium and that the release of left atrial pressure is followed by secretion of an extraordinary amount of anti diuretic material. This of course, is conjecture awaiting adequate experimental investigation. The practical fact is that the intolerance of these individuals to even slight excesses of water be recognized and fluid administration during and after surgery rigorously limited.

Of considerable theoretical interest and occasional practical importance is the clinical effect which results when the serum sodium is forced above its normal level. This situation is fortunately rare since the normal mechanisms for regulation of the extracellular fluid concentration rather strongly resist any elevation of the serum sodium. There are surgical situations, however wherein such hypernatremia may occasionally be observed. One is in certain types of central nervous system damage, namely those involving hypothalamus and the frontal lobes. The second follows certain types of damage

to the kidneys. Although the latter was originally described in connection with sulfathiazole poisoning, it is now recognized that it can follow almost any type of acute renal damage and, as more cases are studied, it would appear that transient hypernatremia of mild degree is a rather common part of the sequence of recovery from acute renal injury. An important factor underlying the development of hypernatremia in the phase of diuresis may be the excretion of a large load of crystalloid materials such as urea, which act as osmotic diuretics. The underlying mechanism responsible for clinical signs of hypernatremia and hyperosmolality is clearly a movement of water from the intracellular to the extracellular compartment in response to the elevation of the serum sodium. Here again the cells of the central nervous system are particularly sensitive and the signs which result are those of profound stupor, central nervous system depression, hyperpyrexia and, occasionally athetoid and choreiform movements of the extremities. Although little is known of the treatment of this disorder it is apparent that, whenever feasible, complete avoidance of parenteral salt administration must be enforced with administrations of as large amounts of salt free glucose solution as the patient can tolerate.

Management of Acute Renal Insufficiency Acute renal shutdown, being a rather common complication of surgery is one that every surgeon must be prepared to meet. The exacting nature of this problem reminds one that in most situations gross discrepancies from the ideal fluid and electrolyte management are compensated for by the kidney whose regulatory powers are normally able to correct the errors of the clinician. In the absence of renal function, however fluid therapy must be extraordinarily precise for every measure that is taken is reflected in the actual composition of the interior of the body.

Although the reactions caused by incompatible transfusion and prolonged periods of shock appear to be the cause of many cases of postsurgical or post traumatic anuria, it must be admitted that many instances of such acute renal failure are ascribable to no single cause. It would appear however that in renal shutdown which does not result clearly from transfusion reactions, or other toxic agents, the common factor is reduction of effective blood supply to the kidney. This may occur even when reduction of the systemic blood pressure does not take place.

Wangensteen, O. H. Controlled Administration of Fluid to Surgical Patients Including Description of Gravimetric Methods of Determining Status of Hydration and Blood Loss during Operation. *Minnesota Med.* 25:783 1942.

Zimmermann, B., Casey, J. H., and Bloch, H. S.,

Mechanisms of Sodium Regulation in the Surgical Patient. *Surgery* 39:161 1936.

Zimmermann, B., and Wangenstein, O. H. Observations on Water Intoxication in Surgical Patients. *Surgery* 31:654 1952.

mounting potassium level, an unfortunate but frequent complication of prolonged anuria. Within limits, this can be prevented by the avoidance of oral and parenteral administration of potassium ion and omitting blood transfusions when they are not absolutely necessary.

Since reduction of extracellular pH favors movement of potassium out of the cell, it is important to treat acidosis when possible by administration of sodium bicarbonate or lactate. Although this may require use of hypertonic solutions and occasionally the administration of more sodium ion than would otherwise be desirable, reduction of extracellular potassium to below critical levels can frequently be effected. The use of appropriate exchange resins is also of value and recent experience suggests that the most successful form of administration is by enema, the resin being suspended in 5 per cent glucose solution. Very transitory reduction in serum potassium level can be achieved by giving glucose intravenously with or without insulin, which causes a small amount of the plasma potassium to be transferred to the interior of the cell in association with glycogen. Persistent increase in potassium level over the level of 7 mEq per liter must be specifically dealt with and to date the only effective mechanism for doing this is perfusion with an artificial type of dialyzer.

With this type of treatment many instances of anuria will be carried to spontaneous diuresis within seven to ten days. At this point careful scrutiny of the chemical picture is also required, for all moieties of renal function do not recover simultaneously. In some instances, the initial diuresis will include large amounts of all the extracellular electrolytes and in the absence of complete tubular regeneration the urine may very closely approximate glomerular filtrate. Under the latter circumstances, very large amounts of water and electrolytes may be required to avoid the creation of overwhelming extracellular fluid deficiency or serious hyponatremia and hyponatremia. It has already been mentioned that under other circumstances sodium and chloride may not be excreted at all in the urine but only large amounts of water may be put out during the period of diuresis. Here as has been mentioned, hyponatremia may develop with its attendant effects on the central nervous system. When this situation is recognized, only salt free fluids should be given, frequently in very large amounts. It

is necessary therefore to follow patients carefully not only during the period of anuria but through the phase of diuresis.

READING REFERENCES

- Albright, F.: "Cushing Syndrome," Its Pathological Physiology, Its Relationship to the Adreno-genital Syndrome and Its Connection with the Problem of the Reaction of the Body to Injurious Agents. *Harvey Lect.* 38:123, 1942-43.
- Casey, J. H., Bickel, E. Y. and Zimmermann, B.: The Pattern and Significance of Aldosterone Excretion by the Postoperative Surgical Patient. *Surg. Gynec. & Obst.* 105:179, 1957.
- Coller, F. A., and others. Postoperative Salt Intolerance. *Ann. Surg.* 119:533, 1944.
- Cuthbertson, D. P. Post-Shock Metabolic Response. *Lancet* 1:433, 1942.
- Darrow, D. C. Body Fluid Physiology; the Role of Potassium in Clinical Disturbances of Body Water and Electrolyte. New England J. Med. 242:878, 1950.
- Darrow, D. C., and Yarnet, H.: The Changes in the Distribution of Body Water Accompanying Increase and Decrease in Extracellular Electrolyte. *J. Clin. Invest.* 14:266, 1935.
- Ellington, J. R., and Danowski, T. S.: The Body Fluids. Basic Physiology and Practical Therapeutics. Baltimore, Williams & Wilkins Company, 1955.
- Gamble, J. L.: Extracellular Fluid—Chemical Anatomy. Physiology and Pathology. Cambridge, Mass., Harvard University Press, 1954.
- Hayes, M. A., Williamson, R. J., and Heidenreich, W. F. Endocrine Mechanisms Involved in Water and Sodium Metabolism During Operation and Convalescence. *Surgery* 41:353, 1957.
- Howard, J. E. and Bigham, R. S., Jr. Transactions of Tenth Conference on Metabolic Aspects of Convalescence. New York, Josiah Macy Jr. Foundation, 1945.
- Little, G. W. and others. Dual Mechanism Regulating Adrenocortical Function in Man. *Am. J. Med.* 31:380, 1956.
- Moore, F. D. ed.: Symposium on Water and Electrolytes. *Metabolism* 5:367-518, 1956.
- Moore, F. D. Common Patterns of Water and Electrolyte Change in Injury, Surgery and Disease. *New England J. Med.* 258:477-525, 377-427, 1958.
- Moore, F. D. and Ball, M. R. The Metabolic Response to Surgery. Springfield, Ill., Charles C. Thomas, Publisher, 1953.
- Moyer, C. A.: Fluid Balance, A Clinical Manual. Chicago, Year Book Publishers, 1952.
- Randall, H. T.: Water and Electrolyte Balance in Surgery. S. Clin. North America 32:443, 1952.
- Selye, H. Stress. Montreal, Acta, 1950.
- Smith, H. W. Salt and Water Volume Receptors—An Exercise in Physiologic Apologetics. *Am. J. Med.* 23:623, 1957.
- Strauss, M. B. Body Water in Man. Boston, Little, Brown & Co. 1957.
- Verney, E. B.: Some Aspects of Water and Electrolyte Excretion. *Surg. Gynec. & Obst.* 106:441, 1953.
- Wacker, W. E. C., and Vallee, B. L. Magnesium Metabolism. *New England J. Med.* 259:431, 475, 1958.

because of the many regulatory functions which the adrenal steroids play in tissue metabolism. An account of endocrine changes alone brings little of usefulness to the practicing surgeon no matter how great his interest because it is the associated metabolic changes and their pathologic variants in his sick patients which are really of critical practical importance. Although the precise relationship between surgical endocrinology and the metabolic response is unknown at this time, the two occur together and are best described together as a basis for the understanding of normal convalescence and, through this, the abnormal.

It is our purpose to outline the underlying principles which should guide daily surgical care according to the chronological sequence of convalescence. First are described the early changes after injury then the period of spontaneous regrowth and, finally, certain aspects of late convalescence. A few of the major abnormalities and variations in these convalescent patterns are then discussed.

CATABOLISM: THE EARLY PERIOD OF STRESS AFTER INJURY

An intraserosus operation involving extensive dissection and anastomosis such as gastrectomy or combined abdominoperineal resection, transperitoneal nephrectomy, colectomy, splenectomy or lobectomy may be considered as the prototype "moderate trauma," a trauma occupying a midposition in the scale of injury to which the flesh falls heir. The subsequent description is based on such a midscale trauma as a "normal" with which other patterns of convalescence may be compared.

Endocrinology Adrenal cortex Endocrine changes are evident very close to the time of the incision. In occasional instances the lowering of eosinophil count prior to surgery may be evidence of preoperative adrenocortical stimulation by apprehension or medication. Shortly after the induction of an ether anesthesia, the eosinophil count (often after a transient rise) drops to zero or near zero and remains there for from two to five days. Coincident with this sudden drop in eosinophil count is a rise in the free serum 17-hydroxycorticoids from normal values of 10 to 20 micrograms per 100 cc. to stimulated values in the range of 40 to 80 micrograms per 100 cc., depending on the nature of the anesthetic and the magnitude of the trauma. The fall in the eosinophil count is usually complete two or

three hours after the induction of anesthesia. The peak in serum steroid is often not reached for an additional three or four hours characteristically in midafternoon of the day of a morning operation. These two changes are selected from amongst a host of measurable indices as indicating an increased secretion of adrenal steroids of the compound E-F group represented largely by compound F or hydrocortisone in normal human adrenal secretion.

Because of this increased secretion of adrenal substances there is an increased excretion in the urine of steroids which have largely been conjugated in the liver as glucuronides. The duration of this adrenal secretory increase varies, but normally the blood steroids have returned to normal by the next day. The eosinophil count may remain near zero for from three to five days before gradually swinging upward. The increased excretion of steroid hormones in the urine may continue for two or three days after the blood level has returned to or near normal. This suggests that adrenal secretion is still increased but that the mechanisms for clearing the blood (hepatic) and excreting the conjugates (kidney) have increased their rate so that they can keep up with the increased adrenal production.

Although many endocrine changes are involved in surgical endocrinology, more information is available on adrenal changes than any of the others. The teaching for years has been that the adrenal secretes three classes of hormones (glucocorticoids, mineralocorticoids and androgens) and that the urinary 17-ketosteroid excretion represents end products of androgen output. We now know that neither of these time-honored contentions is accurate. The adrenal secretes a variety of steroids, many of which possess in one molecule and to varying degrees several of the actions: glucose nitrogen activity, sodium activity and sex hormone activity, the latter being either estrogenic, androgenic or progestational. Indeed, progesterone is one of the adrenal secretory products being an 11-17-desoxycorticoid, and most mineralocorticoids including 11-desoxycorticosterone (DOCA) have progestational activity! The steroids which terminate their life in the body with a ketone group on carbon 17 (the 17-ketosteroids) are not only those which start with this configuration, such as the testicular androgens and adrenal androgens but also a variety of other compounds including cortisone and hydrocortisone which lose their carbon 17-side-chains in the course

ENDOCRINOLOGY AND METABOLISM IN SURGICAL CARE

By FRANCIS D. MOORE, M.D. and
RICHARD W. STEENBURG, M.D.

FRANCIS DANIELS MOORE is a Harvard College and Medical School graduate who was born in Illinois. He was trained in surgery at the Massachusetts General Hospital. He now holds the Moseley Chair of Surgery at Harvard Medical School and is the Surgeon-in-Chief of the Peter Bent Brigham Hospital. Dr. Moore represents the group of surgeons of today who have applied a basic knowledge of biochemistry and metabolism to surgical problems.

RICHARD WESLEY STEENBURG, a Nebraskan by birth, was educated at Stanford University, Harvard College and Harvard Medical School. He is now an Instructor in Surgery at the Johns Hopkins Medical School and Assistant Chief of Surgery of the Baltimore City Hospitals.

INTRODUCTION

During the past two decades it has been possible to map out in some detail a variety of endocrine alterations with associated metabolic changes which are characteristic of surgical convalescence just as other metabolic and endocrine sequences are characteristic of puberty, pregnancy, or senility. Viewed in this light, surgical convalescence is a bodily adjustment to a new set of external and internal circumstances. The circumstances involved are those of acute disease and tissue trauma; the complex response results in healing of the wound and restoration of the individual to normal physical

economic and emotional activity. These changes of convalescent endocrinology and metabolism are common to all fields of surgery and all areas of the human body, although the nature of the wound itself is of great importance in determining the depth and duration of the metabolic response. The metabolic changes produced by a fracture, for example, are different from those produced by a burn, even though certain of the endocrine responses have much in common. An understanding of this metabolism is the basis for effective care in surgery.

The role of the adrenal glands in general surgical physiology is an important one

unless they have been rendered euthyroid by the administration of thyroid substances. Yet there is no incontrovertible evidence that systematic alterations in thyroid function follow trauma and there is a wide variety of disease processes which increase oxygen consumption without demonstrable thyroid imbalance.

There is clinical evidence that gonadal function is decreased after injury. In the female, amenorrhea occurs after extensive trauma or surgery and there is often a male type hair growth observed in women who have very extensive injury such as burns. That this is not solely a nutritional factor is suggested by the observation that weight loss due to other forms of disease is not necessarily associated with such reversion to a neutral sexual pattern. Reproductive activity resumes some time during or after the phase of positive nitrogen balance.

Systematic changes in the function of the pancreatic islets and the parathyroids have not been observed after trauma. The mobilization of liver glycogen and formation of glucose from protein result in an elevated blood glucose after injury effects hastened by adrenal medullary and adrenal cortical activity. When immobilization is prolonged, there is a marked loss of body calcium.

The Wound. During the early period of post injury metabolism, the primarily sutured wound has little tensile strength. The accumulation of leukocytes and of extra cellular fluid containing the compounds found in plasma (many of which are mobilized from muscle) characterizes this period. If dead tissue and virulent organisms do not abound, the wound then commences to gain tensile strength by the conversion of protein precursors into the intercellular structure of collagen. Vitamin C is a requisite for this reaction to occur.

Metabolism. Protein and nitrogen. Protein metabolism is profoundly affected by trauma. Observations of this phenomenon formed the earliest metabolic studies of trauma and there is a wealth of information available in the literature. The characteristic change consists of an increased absolute urinary nitrogen excretion rate despite diminished intake, with negative balance as a result.

The normal adult male ingests approximately 10 to 12 gm of nitrogen a day and excretes a like amount, all but 1 gm. of which is excreted in the urine. He is therefore, referred to as in zero balance of nitrogen. After injury even though the nitrogen

intake may fall to zero as it so characteristically does, the excretion is increased and, after major injury nitrogen excretion rates in the urine of 12 to 15 gm. a day are regularly observed. After more extensive injury the urinary nitrogen loss may range as high as 25 gm per day. This amount of nitrogen (20 gm) lost daily over a period of four days represents the catabolism of about 500 gm of protein—an extreme figure but not an unusual one. This much protein would form the cellular mass of approximately 2000 gm. of wet lean tissue such as muscle. This large amount of tissue which is catabolized after major injury is mentioned to emphasize that there can be little mystery about its source. Except in most remarkable circumstances, the wound itself is a source of only a small fraction of this nitrogen. Changes in size of the liver, heart, lungs, kidney or other viscera would have to be massive of the order of magnitude of 50 per cent reduction in size, in order to account for even a fraction of this nitrogen. It is well known that these organs do not diminish in size after trauma, indeed they are apt to increase. This leaves the skeletal muscle which forms the great mass of protein solids in the body composition, as the source of the nitrogen lost after trauma. After major injury the patient feels weak and if his injury has been considerable, as in a burn or in a war wound, he is extremely weak for two or three weeks after the injury. This weakness is correlated with a visible and easily measurable reduction in size of major muscle masses. It is apparent, therefore that trauma excites the mobilization of small molecular weight nitrogen compounds from the protein of muscle, by an intense catabolic destruction. These nitrogen compounds are excreted in the urine most of them having been converted into urea en route from muscle to urine. There are interesting and important changes in excretion of certain other small nitrogen compounds such as the amino acids, but these are of quantitative minor import though of great significance in providing raw material for wound synthesis.

Potassium. A number of electrolytes exist in the cell with protein and the whole is bathed in an amount of water which comprises approximately 73 per cent of the weight of the cellular mass. The intracellular electrolytes are chiefly potassium, phosphate, sulfate, calcium and magnesium and of these substances, the most information is available with respect to potassium. After a

of metabolism and have it replaced by a keto group probably in the liver. The adrenal hormones are interconverted, inactivated by conjugation or reduction, oxidized and excreted at varying rates depending upon secretion, liver function and kidney function. Given a constant rate of secretion sudden changes in inactivation rate could give the appearance of enhanced activity—and precisely this appears to happen at least to some extent after surgical trauma, in the course of which liver function is almost always impaired.

For these reasons one must view the measurements in blood or urine of a certain steroidal configuration as indicating crudely only a small fraction of the net result of a very complex endocrine change involving secretion, degradation, interconversion and conjugation. The occurrence of such sudden and massive changes in the 17 hydroxycorticoid fraction leaves little doubt that the smooth tensor of adrenal function is drastically altered by tissue trauma.

The secretory impulse which follows surgery appears to involve hydrocortisone predominantly; the concentration of this hormone being much larger than any of the others. The increased concentration of this substance in blood and urine may be measured by a colorimetric method specific for corticoids with a hydroxyl group on carbon 17 (compounds E, F and S). It is such measurements which have been referred to in the foregoing account of postoperative changes.

Mineral activity is not prominent in hydrocortisone, though present. An extremely potent electrolyte-active steroid, aldosterone, is normally secreted by the adrenal independent to some degree of ACTH changes. The increased tendency to retain salt after surgery suggests an increased secretion of this substance. This has not as yet been established and again we must recall that changes in inactivation or excretion could alter the peripheral metabolism, as well as changes in production.

The pituitary and hypothalamus are the keystones of this endocrine response since it is through these agencies that the trauma initiates the endocrine response. The initial change is an increased secretion of ACTH by the pituitary evidently in response to stimuli originating in the periphery and mediated through the hypothalamus.

Adrenal medulla. Coincident with these measures of increased adrenal cortical activity after trauma are evidences of activity

of other endocrine glands. Increased pulse rate and narrowing of pulse pressure with increased sweating and decreased capillary circulation in the skin are evidences of adrenal medullary activity. These signs are most marked if the patient is in severe pain, is very apprehensive or approaches clinical shock due to reduced blood volume. In the absence of any of these circumstances the adrenal medullary evidences may be quite minimal except for the effects of other anesthetics which evidently are associated with a very marked production of epinephrine and related compounds by the adrenal medulla.

Antidiuresis. A normal individual given an intravenous injection of a 5 per cent solution of glucose in water rapidly increases his urine flow from normal rates of 1 ml. per minute to rates as high as 6 to 8 ml. per minute. Coincident with this there is a drop in the urine osmolarity from normal values of 500 to 800 milliosmols per liter down to values nearer 200 milliosmols per liter. By dint of this water diuresis the solute concentration of serum (as measured by sodium protein or total osmolarity) undergoes no lasting change. This is a normal diuresis in response to water infusion. After surgery, even of rather minor extent, this normal diuresis of a water load is markedly or completely inhibited. The same infusion given under these circumstances produces virtually no increase in urine volume or decrease in urine osmolarity. The water is retained with a resultant fall of serum osmolarity from normal values of 280 to 290 milliosmols per liter down to values around 260.

These are the observations in surgical patients which have demonstrated the occurrence of "postoperative antidiuresis." This is clearly of importance in surgical care and it appears to be another evidence of endocrine activity after trauma. Presumably this effect is produced by stimulation of the posterior pituitary gland to elaborate its antidiuretic hormone. Although this has not been proved, several workers have measured an increase in the urinary excretion of antidiuretic substances in surgical patients.

Other endocrines. After trauma there is an increase in oxygen utilization and an evident increase in the rate of oxidation of tissue substrates particularly fat and protein. This might appear to be thyroidal in origin. It is certain that a normal thyroid is essential for a normal trauma response; patients with hypothyroidism are notoriously intolerant to surgical trauma and medication.

zero for the obvious reason that the patient will not cannot or should not eat. The glucose administered intravenously provides a small supply of readily available calories which may be the only calories supplied until oral intake again begins. During the first five days after trauma, there is little to suggest that forcing further calories affects the patient favorably. This is in sharp contrast to the situation later on in convalescence when the administration of exogenous calories is of vital importance for recovery.

The blood sugar quite regularly rises after trauma and there is some tendency for glycosuria and decreased glucose tolerance. This "anti insulin effect" may be due to the increased amount of steroid circulating in the blood, to the mobilization of liver glycogen or to the sudden production of carbohydrate by the degradation of fat or protein.

Serum protein. During this early phase after trauma, the serum protein concentration does not change markedly. If there is acute dehydration due to loss of water or water with salt, the serum protein may rise along with the hematocrit reading. If there is accumulation of water and salt due to overadministration of these substances, the serum protein will fall. Such excesses of water and salt are the commonest causes of hypoproteinemia in surgical patients.

Clinical Management. After moderate trauma in well nourished individuals, the metabolic management of this phase of convalescence presents few problems.

The most important therapeutic step is that of a well performed operation, accurately directed to the major disease from which the patient suffers and carried out in such a way as to impose on the wound a minimum load of contamination and dead tissue and to avoid hypotension, oliguria, distention or extrarenal loss. All of these distort the normal convalescent sequence and divert the resources of the organism.

If the patient's oral intake is interrupted for only one day, he will get along well without any intravenous or other parenteral therapy. If more extensive trauma has occurred, the administration of intravenous fluids for a day or two may forestall dehydration and provide water until intake starts. The administration of 50 to 150 gm of carbohydrate by vein in these first few days forms an inevitable accompaniment of water administration and provides a small caloric ration. Enough water should be administered to cover the losses from the body by lungs, skin and urine, allowing for urine

output of between 350 and 750 cc. on the first day and somewhat more on subsequent days.

When there are no extrarenal losses of water and electrolyte the administration of electrolyte is usually unnecessary during the few days before oral diet is recommended. Small amounts of electrolyte do no harm however and many prefer to give enough to allow for small losses. As an example if enough potassium sodium and chloride are given to allow for 40 mEq of each in the urine during the early postoperative days this purpose will be accomplished without excess. Under no circumstances should the patient be given excesses of electrolyte or of water since in either case retention will result, with the production of serum dilution or hypoproteinemia or both. Furthermore quantitative replacement of all urinary losses of sodium and chloride should not be attempted.

When trauma has been extensive and is accompanied by sepsis continued interruption of oral intake, high fever continuing blood loss, wound edema, extrarenal loss or renal disease, the management of this phase of convalescence requires extremely accurate and considerably more complex therapy. The principles involved are based firmly on the endocrine and metabolic facts mentioned above for normal convalescence. The administration of fluids, electrolyte or blood should be carefully balanced to equal the loss, recalling that renal excretory patterns are keyed to conservation of extracellular water and salt. When the interruption of oral intake can be expected to last for more than three days it is advisable to commence potassium administration by vein on the day of or the day following surgery using 40 mEq per day as a general rule to cover renal losses and larger amounts when extrarenal loss is prominent.

ANABOLISM: THE PERIOD OF REGROWTH

Endocrinology. As mentioned previously, the initial burst of adrenal steroid activity which follows trauma is rather short lived, at least as judged by the increase in serum and urine steroid concentration. The eosinophil count rapidly returns to normal, often reaching values considerably higher than those observed preoperatively. It should return to normal by the fifth day after mild trauma. Evidences of a posterior pituitary like effect in the stress phase immediately after injury were found in the tendency to

surgical operation, potassium excretion is increased in the urine. The normal adult consumes about 100 mEq of potassium each day and excretes a like amount in the urine. If his intake is suddenly stopped, he excretes about 40 or 50 mEq in the urine the first day and then over a period of days, or weeks his urinary potassium excretion is gradually reduced to a minimum figure of about 10 mEq per day. After major surgery—with no potassium intake—the urinary excretion ranges from 70 to 100 mEq the first day and for the next two or three days the potassium excretion continues to be greater than one would expect in starvation alone. The amount of potassium lost from the body in the first four days is from 150 to 300 mEq as an average. For the most part, this potassium may be considered as coming out of cells along with nitrogen as the cellular structure is destroyed or atrophied following trauma. In most instances, however, there is some "excess" potassium excreted above that which would be expected to come out with the nitrogen alone. Potassium and nitrogen exist in muscle in a fairly fixed ratio at 2.7 to 3.0 mEq potassium per gram of nitrogen. After injury the potassium is lost at a rather higher potassium nitrogen ratio and by the same token it starts to return to the cell somewhat sooner than nitrogen. This "excess" potassium is presumably removed from the cell in exchange for hydrogen or sodium ions or both, and this abnormal state of affairs reverts to normal more rapidly than the resumption of true tissue anabolism. After this initial potassium loss, the body rapidly regains potassium as feeding is resumed.

Sodium chloride Both sodium and chloride are conserved during the early phase after surgery. As a general rule more sodium is retained than chloride. In terms of renal excretion the sodium conservation mechanism operates more actively than that for chloride. On the day of operation, sodium excretion may continue, but at a reduced rate with amounts from 10 to 50 mEq being lost in the urine if no sodium is given. Then, in the next two or three days, sodium conservation becomes evident and the sodium excretion in the urine is reduced sharply to from 1 to 10 mEq. In more severe injury particularly when there is an oligemic phase, the reduction in sodium excretion may commence immediately if sodium is given intravenously during this phase, there is an increase in sodium excretion over that observed when no sodium is

given. The sodium excretion will not keep pace with the infusion, however, and a strongly positive sodium balance will result. The duration of this sodium conservation phase is among the most variable features of post traumatic metabolism. Usually in well nourished individuals who have had a major injury or are undergoing surgery sodium conservation starts rapidly and passes off in three or four days with fairly clear-cut sodium diuresis during which sodium excretion is increased. In individuals who are less well nourished, sodium conservation may be a little slower to start but persists for a much longer time and diuresis may never be clear cut.

Serum electrolytes Early after trauma and coincident with these changes in metabolism is a tendency for the serum sodium concentration to fall (to 130 to 135 mEq per liter) and the potassium concentration to rise (to 4.8 to 5.8 mEq per liter). These tendencies appear to be paradoxical in view of the opposite nature of the concomitant balance changes. The explanation of the concentration changes is unknown and in normal circumstances it is of no more than passing interest clinically but in the presence of depletion, shock or heart disease these changes may become tremendously exaggerated and be a threat to survival.

Body weight Body weight tends to fall sharply after trauma, particularly if the trauma is in a previously well nourished patient, and if there is not a large area of edema accumulation as in a burn or a crush. If traumatic edema accumulates the weight loss may be obliterated or replaced by a gain because of the accumulation of water and salt. When this fluid is subsequently excreted, the patient's weight will fall sharply down to the level which would have been attained had catabolism of fat and lean tissue been the only occurrences. Following moderately severe trauma in well nourished people, one may expect a loss of approximately 3 kg. of weight in the first five days. In more extensive injury the loss may be two or three times as fast. This lost tissue is about one-half fat (the increased oxidation of which is one of the characteristic changes of convalescence) and one-half lean tissue (muscle) which is the source of nitrogen in the urine as already mentioned. This weight loss rate is gradually reduced, reaching a plateau, and later giving way to the weight gain of convalescent anabolism.

Calories glucose The caloric intake immediately after trauma is usually reduced to

of nitrogen metabolism is the positive balance of later convalescence. While we may view the initial nitrogen loss with some tolerance, not knowing of any deleterious results, we may take no such relaxed view toward failure in subsequent anabolism. Nitrogen gain and protein synthesis are absolutely essential for recovery. Without them the patient cannot return to a position of social usefulness, reproductive activity or the enjoyment of life. He will heal his wound—but there his achievements will cease.

As to the relation of nitrogen balance to wound healing, one commonly hears the statement that wounds will not heal unless the patient goes into positive nitrogen balance. This is not the case. Most wounds heal readily during a negative nitrogen balance and it is commonplace after very extensive injury or surgery for the wound to heal to a state of tensile strength which permits removal of the sutures and the resumption of function of the intestinal tract, for instance, while the patient is still in negative nitrogen balance. This is not a rarity; it is a daily occurrence. It has been hypothesized that the mobilization of nitrogen from body stores has as one of its natural purposes the provision of small molecular weight nitrogen compounds as building blocks to provide the tissue which heals the wound. Such a view is merely an interpretation of natural events; but it is based on the fact that patients accomplish the initial phases of wound healing during the negative-nitrogen phase. The most egregious failure of wound healing (manifested by wound dehiscence and the complete failure of fibrosis to occur) is not associated with an unusual degree of nitrogen loss. Indeed, it is somewhat commoner in depleted individuals with cancer in whom the post-traumatic mobilization of endogenous nitrogen is halting or scanty. And finally as mentioned above the later assumption of nitrogen anabolism achieves its importance not because of wound healing but because it permits the regrowth of muscle and resumption of normal bodily strength, vigor and visceral function.

Potassium balance becomes positive during this anabolic period and potassium loading starts significantly prior to positive nitrogen balance in most instances as described above. It remains slightly positive throughout the phase of spontaneous anabolism.

Weight loss slows down as the metabolic corner is rounded. If adequate diet cannot

begin it persists at a slower rate. As diet begins a slow upward trend commences.

Clinical Management. If the patient will eat his food normally the clinical management of this phase of convalescence poses no therapeutic problem. Recognition of the underlying requirements for exogenous calories and of the caloric nitrogen ratios required, forms the basis for the rational treatment of surgical convalescence. As mentioned above, the provision of large caloric intakes is of dubious benefit in the early period of catabolism. If food given by mouth is not absorbed or is vomited, the attempt to provide food early may actually be very harmful. There is no greater tragedy than distention or aspiration as a complication of unnecessary early diet forcing. The nutritional objective in the early period is a scaphoid abdomen.

As anabolism is achieved, peristalsis is resumed, there is anal excretion of swallowed air and the time has come for diet. As he passes through the corticoid withdrawal phase and assumes a decreased urine nitrogen excretion rate, his body becomes extremely avid for exogenous calories. This is usually manifested by an increase in appetite, an interest in food and resumption of normal peristaltic activity. The provision of exogenous calories now becomes essential to recovery and for the production of anabolism. The calories ideally should be supplied at the rate of 200 calories for each gram of assimilable nitrogen in the diet. But caloric-nitrogen ratios somewhat lower than this will support anabolism.

In normal convalescence after surgery of the abdomen or thorax or extensive surgery of the extremities the patient's initial dietary effort during the corticoid withdrawal phase will give him nitrogen intakes in the range of 3 to 7 gm. per day with caloric intakes between 500 and 1500 calories. After two or three days of such an intake in the form of semisolid food, the patient will resume a normal mixed diet having a caloric intake of approximately 2400 with a nitrogen intake in the range of 12 gm. In the case of a female, these figures are all somewhat lower.

During early anabolism, the patient's weight will remain seemingly stationary at a level of from 1 to 3 kg. below the pre-operative weight. If very careful weight measurement are made daily it will be found that weight is not stationary but is instead increasing very slowly at a rate determined by the positive nitrogen balance. Each gram of nitrogen loaded represents

retain ingested or infused water. This tendency passes off and the patient's metabolism of water returns to normal within a day or two unless complicating systemic or surgical factors are present.

The Wound. In a clean primary wound of the type considered as the "norm" here, the resumption in tensile strength occurs during the sharp upswing in nitrogen metabolism described below. This coincidence is lacking in more complex settings, however, and we estimate that, on a mixed general surgical service caring for trauma as well as elective surgery about two-thirds of the wounds gain their tensile strength during continued nitrogen negativity. Resumption of tensile strength is the result of fibroblastic activity and the formation of intercellular collagen which later becomes increased in the mature scar. The wound at seven days is a thin line appearing much as it did when originally sutured, at five to seven weeks it is a red, raised, thickened cicatrix.

Metabolism. Although the steroidal response is usually short lived, the metabolic change apparently initiated by it may outlast it by many days. In the moderate mid-scale trauma we are using as a prototype, the nitrogen reversal often occurs within a day or two after subsidence of the adrenal discharge. In other circumstances, particularly in burns and fractures, as described subsequently the association is less close. In any case, at some time after this release from steroidal influences, and only after its release, there occur two metabolic events which symbolize the changing metabolism of recovery.

The first is a reduction in the urinary nitrogen excretion rate. It is the occurrence of this reduction in urinary nitrogen excretion rate together with a rise in eosinophils and a fall in urine steroid excretion, which has caused this early reversion toward more normal metabolism to be called "the corticoid withdrawal phase." When the events happen together the picture closely resembles that produced when large doses of ACTH or cortisone are withdrawn from a normal individual. The urinary nitrogen excretion rate is suddenly reduced from rates of 14 to 20 gm. a day down to rates of from 5 to 7 gm. a day over a period of one to two days. This indicates a sweeping change in nitrogen metabolism and protein dynamics and if intake now rises with the resumption of eating, the patient rapidly assumes a positive nitrogen balance.

The second event is a release of water and

salt from the early post-traumatic conservation. Urine sodium concentrations rise and the excretion of water loads is prompt and more clearly related to serum osmolarity. In some instances these events constitute a clear-cut diuresis phase. In other cases, the changes are delayed and less clear-cut though of equal importance in signaling the normal progress of convalescence.

The positive nitrogen balance period which now ensues has been called "the spontaneous anabolic phase of convalescence" and is a period of surpassing importance to the recovery of the individual. If the patient does not go into positive nitrogen balance, he will not regain muscular mass and strength and he will not recover. In such an event, the dynamic progress of convalescence may be thought of as "stalled." The patient will develop chronic weakness and apathy will not permit mobilization, weight loss will continue and the picture of convalescent failure will ensue so common in association with late burns, sepsis and starvation. The rate at which protein is synthesized during anabolism averages in the neighborhood of 3 to 5 gm. of nitrogen per 70 kg. body weight per day. Instances have been reported in which spontaneous anabolism persisted for several weeks at a somewhat higher rate, even as high as 8 gm. of nitrogen per 70 kg. per day an unusually rapid rate.

The relationship of nitrogen metabolism to the recovery of the patient, on the one hand, and to the healing of the wound, on the other, has been the source of much misunderstanding. There has been an impression that the loss of nitrogen in the early phase of convalescence is harmful to the patient and should be combated. If the individual has long been depleted by chronic disease, a strongly negative nitrogen balance (such as that produced by sepsis) is unquestionably deleterious as further acute tissue wasting superimposed on chronic starvation results in visceral damage, particularly a fatty liver. In the well-nourished, the brisk loss of nitrogen which follows trauma is usually correlated with a satisfactory convalescence. Although the patient might be "better off" if he did not lose this nitrogen, such has never been proved. By large intravenous loads of nitrogen (as protein hydrolysate) and glucose, the total losses may be reduced, the excretion rate however is further increased. Benefit to the patient by such procedures is not readily demonstrable. Much more important in the consideration

the application of surgical skill as a whole of which metabolic care forms but one aspect.

Extrarenal Losses. Since the first chemical observations in surgical patients the subject of dehydration has occupied a great deal of attention in the surgical literature. Surgical dehydration most commonly occurs as a result of gastric or intestinal obstruction with vomiting of intestinal juices. Other causes are diarrhea and intestinal and pancreatic fistulae. These represent extrarenal losses of fluid from the body and the term is used to indicate those losses of fluid salt and protein from the body which take place outside the kidneys and outside the normal renal regulatory mechanism. Occasionally such collections as massive recurrent pleural transudate, portal vein obstruction with massive accumulation of exudate in the bowel or appendiceal peritonitis produce a picture of which dehydration forms a part. Under special circumstances abnormalities of renal function may produce severe dehydration through the kidneys themselves. The recovery phase of post-traumatic renal insufficiency furnishes an example of unregulated water and salt loss through the kidney. With this exception most dehydrating conditions in surgery involve extrarenal losses.

The term "dehydration" has come to be loosely used. Dehydration without salt loss is rare in surgery, being largely due to pulmonary loss in high ambient temperatures. When it occurs, it produces marked serum hypertonicity with hyperchloremia, hypernatremia and uremia. A similar picture is produced by injudicious tube feeding in the unconscious patient. Prompt treatment with intravenous dextrose in water is most effective in pure dehydration. The commoner situation is dehydration due to loss of water and salt, the latter largely extracellular.

In no phase of surgery is the concept of "metabolic balance" more aptly brought to the bedside than in the management of the patient with large extrarenal losses. The objective of the attending surgeon should be to achieve perfect balance. By this is not meant nitrogen balance. It is almost impossible in such a patient to achieve anything that even approaches positive nitrogen balance. In a patient with acute intestinal obstruction losing liters of salt-rich fluid each day it is folly to waste time over a consideration of calories or nitrogen balance. When the acute situation has been brought under control

and the patient is again eating normally, his nitrogen balance will take care of itself.

Rather the concept of "metabolic balance" in taking care of such patients is devoted to their balance of water and salt. The salts involved theoretically include the entire gamut of substances which are found in body fluids: sodium, chloride, potassium, magnesium phosphate, sulfate, calcium and zinc; these could all be mentioned but we have no way of measuring most of these substances. If the surgeon will devote his unstinted attention to the balances of sodium chloride and potassium avoid over administration of water maintain blood volume and colloid osmotic pressure, he can guide his patient through the most massive disorders of water and electrolyte metabolism and emerge successful.

The first step is to measure the patient's intake and output with maximum accuracy. The measurement of fluid volumes should never be incomplete or inaccurate and on a surgical service equipped with such a simple device as a hollow container there is no excuse for ignorance as to the amount of fluid which has come out of the various orifices of the patient. If there are copious exudates in the dressings, vomitus on the sheets and pillow cases or diarrheal stools in the bed, such measurement becomes impossible but an observant nurse or doctor can make an educated estimate which is better than nothing. An estimate of the salt content of the collected discharges can be made on the basis of data documented in the literature or by direct analysis. The following general rules hold for the gastrointestinal tract:

Chloride—

100 mEq /l throughout

Sodium—

High acid gastric juice: 15 mEq /l
Low acid gastric juice: 60 mEq /l
Duodenal juice: 100–140 mEq /l
Pancreatic juice: 140–180 mEq /l

Potassium—

Stomach: 15 mEq /l
Small bowel: 15 mEq /l except in small bowel obstruction or ileostomy diarrhea in which this may reach 70 mEq /l

Secondly the patient's weight should be followed closely. Weighing every day or at least three times a week, is essential. Rapid gains or losses are undesirable, indicating massive fluid shifts. In the unstressed starving patient a slow loss of about 150 gm

about 30 gm. of lean wet tissue. For this reason a positive nitrogen balance of 5 gm. per day a high rate of spontaneous anabolism will result in the accumulation of only 150 gm. of weight per day. On such a basis it takes approximately a week to gain 1 kg. Minor fluctuations in water balance and daily happenstance differences in the time or condition of weighing will mask this small rate of gain unless special measures are taken to observe it.

The most important abnormalities in convalescence during this phase are continued sepsis, continued activity of parenchymatous visceral disease, hemorrhage and disorders of the intestinal tract, particularly diarrhea, obstruction and paralytic ileus. All of these either increase the length of the period of catabolism, or postpone anabolism by lowering the ability of the gastrointestinal tract to resume its functions of ingestion and assimilation.

FAT GAIN: LATE CONVALESCENCE

Endocrinology. Little is known about the endocrinology of late convalescence. In the female, following extensive injury surgery or burns the male type hair growth characteristic of the catabolic phase disappears and the female physical appearances return. Normal menses likewise return. The relationship of this gonadal renaissance to the other metabolic changes is uncertain.

The Wound. During nitrogen anabolism the wound becomes a red, raised cicatrix, often somewhat sensitive to the touch. As the patient passes into the later phase of convalescence, the wound gradually broadens, softens, turns white and may become concave or wrinkled. The fine suture marks often are obliterated completely.

Metabolism and Clinical Management. During this time the patient is observed to have returned to zero nitrogen balance if convalescence has been normal and complete. Zero nitrogen balance is the normal state of the intact adult without gains or losses of weight.

Despite zero balance of nitrogen, potassium and sodium these being the elements which we have considered throughout, the patient continues to gain weight. Measurements of total body water demonstrate that body water is constant during this period of weight gain which indicates that the weight gain is due to accumulation of fat. When a patient has been chronically or severely ill or has undergone major surgery which has been followed by resumption of

normal gastrointestinal function no elaborate methods are needed to determine that fat is being gained during this time. Normal body contours are restored and clothes fit once again.

The patient quite regularly leaves the hospital during the spontaneous anabolic phase of convalescence. For the body to regain fat normally the caloric intake must be significantly in excess of the daily energy output. If a patient is "borderline" as to calories because of some disorder of the gastrointestinal tract premature return to work will interfere with fat gain; the patient will appear to have had a normal convalescence in every other respect, but he will not regain his normal weight. An example would be digestive disorders after subtotal gastrectomy. Therefore, if an individual is not doing well as regards appetite and caloric intake, he should be discouraged from assuming full activity until weight has been restored.

COMMON ABNORMALITIES OF CONVALESCENCE

In some of the common abnormalities of convalescence, the basis of care lies in an understanding of the variations which they present from the normal endocrinology and metabolism of convalescence. In many instances such as renal failure, dehydration from extrarenal loss, fractures and sepsis there are many details of pathogenesis, diagnosis and daily care which will not be mentioned. In all instances, the surgeon must realize that surgical judgment based upon a careful history, an accurate examination of the patient, intelligent selection and evaluation of laboratory work and x-ray examinations must take first place in his care. Sick surgical patients are problems in surgery, first. Their metabolic disorders may be of central importance in their recovery but without good surgical judgment, metabolic care is wasted. A patient with continued fever, continued endocrine activity, continued high nitrogen loss in the urine, continued conservation of sodium, all due to a subdiaphragmatic abscess, must have the abscess suspected, diagnosed and then drained. The adequacy of his metabolic care may determine survival in the end, but competent surgery comes first. If disease is overlooked or surgery clumsily performed, all else is in vain. By the same token, it avails little if body chemistry is normal while untreated thromboembolism continues its lethal course unrecognized. Patients are helped by

of ammonium chloride and potassium chloride and by the conscientious replacement of extrarenal losses of chloride and of sodium is the most important single step in the prevention of hypokalemic alkalosis. Care must be taken to avoid overadministration of sodium.

Hypokalemic alkalosis characteristically follows a period of potassium deprivation when urinary potassium loss is increased as in the postoperative state and in the presence of extrarenal potassium loss such as occurs in upper gastrointestinal disease. But the full blown syndrome can occur with the loss of only 100 mEq of potassium if alkalosis is sudden and severe. The replacement of potassium is clearly of importance. Amounts as great as 120 mEq per day may be given intravenously in treatment. *But the effect of potassium administration on serum potassium will be disappointing if the alkalosis remains uncorrected.* Fortunately potassium chloride achieves both objectives simultaneously.

Operative stress makes hypokalemic alkalosis much more severe. The syndrome is most commonly seen in postoperative patients. The administration of ACTH to alkalotic subjects produces a worsening of the alkalosis and hypokalemia. It is thus clear that tissue stress plays some role here and it is our interpretation that stress acts by blocking the urinary sodium excretion (aldosterone effect) which would otherwise help to compensate for the alkalosis. The important point again is to avoid surgery in alkalotic patients until their alkalosis is corrected. We regard any preoperative carbon dioxide concentration in plasma over 30 mM/L as dangerous in this regard.

Finally the electrocardiogram is useful and provides a check on such patients at night or at times when serum potassium concentration is hard to measure but the electrocardiographic changes are not due to changes in the serum potassium concentration alone and an electrocardiogram cannot replace quantitative analysis.

The recovery of patients with hypokalemic alkalosis does not depend solely on restoration of the chemical values of the blood or the electrocardiogram to normal, but instead on solution of the basic problem of gastrointestinal function. The patient's life is threatened by hypokalemic alkalosis and its prevention or treatment is of first rank importance, but the restoration of normal gastrointestinal function must come also to produce recovery. The repair of the hypo-

kalemia will help promote resumption of normal neuromuscular function in the gut as well as the heart.

Hypotonicity. The Low Sodium Syndromes. With the increasing use of flame photometry for the study of plasma base patterns there has been observed a large group of surgical patients with low serum sodium concentrations often as a feature of severe illness. Since the sodium ion concentration is the most important single determinant in the serum osmolality a patient with a low serum sodium usually has a hypotonic serum and extracellular fluid with an excess of water both within and without cells. Not infrequently a high serum potassium concentration is observed with the low sodium. Since it is one of the functions of the life process to provide cellular energy which maintains a high potassium concentration and low sodium concentration within the cell and the reverse outside, it is evident that one of the manifestations of cell illness and death will be the abolition of this gradient a fact histochemically established by many observers. Because of this fact a low serum sodium concentration is often associated with severe illness and its passive repair by sodium infusion quite unavailing. The repair of the primary pathologic process is followed by restoration to health and isotonicity together. Under other circumstances, an attempt to restore the sodium level may be lifesaving. It is therefore worth while to discriminate among the various hypotonic syndromes.

1. Outright *adrenal failure* involves unregulated urinary sodium loss as has been mentioned. Analysis of the urine for sodium is, therefore the first step in differential diagnosis of hyponatremia. If in the presence of a serum sodium concentration below 130 mEq/L, the urine sodium, in the absence of infusion, is over 30 to 50 mEq/L, adrenal failure or renal failure should be suspected, and suitable differential study should be undertaken. As a first step in this study the response of the renal sodium output to DOCA is useful in discriminating renal from adrenal failure.

2. Large *extrarenal losses* of sodium can lead to a low serum sodium, particularly if replaced inadvisedly by a large amount of sodium free water.

3. In the presence of uremia with a high nonprotein nitrogen or diabetes with a high sugar or possibly other situations producing high crystalloid concentrations mild hyponatremia can coexist with serum isotonicity.

a day as fat is oxidized, is to be expected until oral intake and anabolism are restored.

Thirdly do not attempt daily urinary electrolyte loss replacement. It is a great mistake to restore to the patient the following day the amount of water or sodium lost in the urine the previous day. Renal excretory mechanisms for sodium, chloride and water are in part regulatory of extracellular volume and acid base balance. The result of readministration of the sodium excreted in diuresis can readily be imagined. If the patient is having very large urine volumes, it is also unnecessary to try to restore them each day unless one of the very rare renal tubular lesions is present. In most surgical patients high volumes or concentrations in the urine indicate important renal compensation for irregularities of intake. For this reason, we allow 40 mEq of sodium, 40 of chloride, 40 of potassium and 1200 cc. of fluid each day for the urine output. This is a sound basis for treatment and will avoid serious error if renal function is normal. Insensible loss through skin and lungs totals about 750 cc. per day in a normal-sized adult male 500 cc. for the female. If there is fever, hot weather or dyspnea, this may be tremendously increased. Dyspnea with fever is the most effective combination in increasing the extrarenal loss of water through the lungs to figures as high as 2000 cc. per day.

Armed with a knowledge of what has come out of the patient, and with an estimate of the extracellular concentrations remaining, the surgeon can approach his daily intravenous therapy with accuracy and conviction. Under conditions of gastric obstruction, intestinal obstruction or diarrhea, the amount of fluids required per day may of course be massive running as high as 8 to 10 liters in certain instances. By sharp contrast, the small, chronically ill female with congestive failure who has been vomiting from digitalis toxicity may be overtreated if she receives 150 cc. of saline solution. Clarity of concept and accuracy of plan are essential.

If the patient comes upon the scene with established dehydration, the situation is much more complicated because the static debt of the patient must be made up during the course of the first few days, during which maintenance is also essential. The most important rule in a large experience with such cases has been to avoid the temptation to make up the entire static debt too suddenly or on the first day. Patients be-

come adjusted to lower levels of extracellular volume and they also become adjusted to some degree of serum hypotonicity. As has been said so aptly the acutely unbalanced patient should be "nudged," not "swept," in the right direction. If the patient is an older individual with some degree of heart disease the attempt to restore all past losses in one day may well result in a fatal outcome. As in all things in medicine, a careful history of intake and loss will set the stage for intelligent treatment. A weight gain for three to four days is to be expected as the debt is restored. This is the only exception to the basic rule of parenteral therapy: avoid weight gain.

Potassium Loss, Alkalosis, Hypokalemia. In the past ten years there has become recognized a syndrome usually in post-operative patients consisting of distention, ileus, lethargy, fever, weakness, dehydration and electrocardiographic evidences of altered neuromuscular excitability. With this there is found a remarkable chemical situation consisting of metabolic alkalosis (a high carbon dioxide with normal or high sodium and often a low chloride), hypokalemia (a low plasma potassium concentration in the range of 2.5 to 3.5 mEq per liter) and an acid urine seemingly paradoxical. Some degree of azotemia may coexist if dehydration is severe.

A considerable controversy has turned on this matter particularly on the question of whether the potassium loss produces the alkalosis or the alkalosis lowers the potassium concentration and, indeed, on the central question of the role of potassium loss itself in the syndrome. There are several important facts which have emerged and which permit an understanding of the situation and, better still, effective prevention and treatment.

Hypokalemic alkalosis usually occurs in patients who have been alkalotic prior to surgery or in those who have had an extrarenal loss pattern after surgery which could be expected to produce alkalosis. Examples are patients with obstructed duodenal ulcer with preoperative alkalosis or those having upper gastrointestinal surgery (pancreas, stomach) in whom postoperative gastrointestinal loss has been predominantly gastric juice. Alkalosis can be demonstrated to lower the plasma potassium concentration experimentally. In most surgical patients with this disorder the alkalosis appears to be the important primary event. The prevention of alkalosis by the preoperative use

intestinal cancer and chronic congestive heart failure. Though these three entities are seemingly of far different etiology, their effects on body composition are similar. There is a loss of weight due to loss of both fat and lean tissue. After a few months of illness most of the available body fat has been mobilized and oxidized. There is therefore a relative increase in body water, largely extracellular, because of the loss of fat which occupies the nonaqueous phase of body composition. In addition the chronically depleted patient shows a marked tendency to retain salt and water. This is responsible for starvation edema. If there is disease of heart or liver, this water and salt retention is greatly accentuated as has been known for centuries. The chronically depleted patient approaching surgery is therefore a patient who has too little fat, too little lean tissue, too much body water, too much of which is extracellular water, and too much extracellular salt (sodium and chloride) in his body. His response to trauma is quite characteristic. The depression of eosinophils and rise in blood and urine steroids are quite active but they are very transient. The quantitative steroid changes are not great and they rapidly revert to normal. The loss of nitrogen in the urine is not great and the patient will return to positive balance with low calorie-nitrogen ratios. If he is managed "gently" and water and salt overloading avoided, the chronically depleted patient will pass through his surgical experience quite uneventfully and wound healing will usually be quite normal.

Although such careful management will usually produce a well healed wound and a successful convalescence, the over-all incidence of wound dehiscence, or primary failure of fibroblastic union, is slightly higher in this group than in previously well nourished subjects. This is especially true of patients with cancer, particularly if the disease has reached the inoperable stage. Nonetheless, the incidence of dehiscence in the group as a whole is low in absolute terms—about 2 per cent. The remarkable thing is that given good technique in closing such a wound, adequate vitamins, avoidance of salt overloading, the great majority of these wounds do heal well despite advanced tissue wasting—an example of the meaning of the high biologic priority of the wound in the early phase of convalescence.

If blood loss has been a part of the depleting disease process, the blood volume will be low, particularly in the red cell fraction.

If blood loss has not been prominent, these wasted patients commonly have a normal or high blood volume with a definitely elevated plasma volume and a slightly low hematocrit reading. Massive transfusion of these patients preoperatively is based on the impression, often erroneous, that they have a chronic blood volume deficit. This impression is based on the slightly low hematocrit reading. The transfusion will load down the circulation with excess fluid, only to embarrass anesthetic and postoperative management. If these patients' depleting process has been accompanied by true hemorrhage, their red cell mass may be very low and the judicious use of whole blood transfusions or of packed cells may be quite valuable. Beware of sudden increases in blood volume in depleted elderly patients! Again "nudge," don't "swamp" the depleted individual.

Depleted patients have lost their "metabolic reserve" and are extremely sensitive to several metabolic challenges. First among these is infection. Such patients have little ability to withstand the catabolic ravages of sepsis and if they are overtaken by invasive sepsis they will frequently very rapidly succumb. Second is massive injury. Such patients can withstand gentle management and surgery which is not too extensive; their wounds heal nicely. If the tissue insult is too great, such patients do not rally well, will develop renal, hepatic or pulmonary failure and failure of wound union, and will succumb. Thirdly, these patients are very vulnerable to overadministration of water and salt; serious positive balances are quickly established, with resultant hemodilution, hypoproteinemia and pulmonary edema. Cough reflexes are poor, effort small and aspiration common. The cause of death observed at postmortem examination usually includes "bronchopneumonia" or interstitial pneumonia. There may also be pulmonary edema and a mixed renal lesion. In some instances the pathologist can cast little light on the mechanism of death. It is for these reasons that staging of procedures and dexterous gentle surgery with careful metabolic management are of such importance in chronically depleted individuals.

It should be emphasized that patients who have lost one-tenth to one-eighth of body weight, largely as fat, and who still have strength to walk vigorously around the ward with good vital capacity and normal extracellular chemical balance are excellent risks for extensive surgery. They are in many ways a better risk for surgery than is the

The abnormal crystalloid fills up the solute total to normal

4 After *trauma* it is a common phenomenon to find the sodium concentration low and the potassium high. The range here is not great.

5 If none of the above four obtains (adrenal renal failure, extrarenal loss, isotonic hyponatremia due to crystalloid excess normal post traumatic "shift") one is probably dealing with hyponatremia as a manifestation of diffuse cellular illness and the problem therapeutically is whether or not the use of concentrated salt solution is advisable.

If the patient is *edematous* and suffering from disease of heart liver or kidneys the use of concentrated salt is very dangerous. There is a wealth of evidence that such patients have an excess of body sodium already. Their basic defect is an inability to excrete water and an inability to exclude sodium from cells.

If the patient is *not edematous* a cautious trial of 300 cc. of a 3 per cent solution of sodium chloride daily for three days, with observation of clinical and chemical effects is justified. Particular attention should be paid not only to the serum sodium response and urine output but also to the early signs of pulmonary edema and to the hypoproteinemia which may result. If the patient has been on a low sodium diet and mercurial diuretics, the therapeutic trial is especially worth while; it should be realized that some degree of body deficit may be present, though rare.

In any case it is of singular importance to bear in mind that general nutritional care is often of greater effectiveness than an exclusive interest in the serum sodium concentration alone. Repair of anemia, avitaminosis and hypoproteinemia and, above all, attention to caloric intake in the chronically ill may be the most effective steps in restoring the serum sodium to normal and the patient to normal health.

Prolonged Intravenous Feeding. If the patient's sepsis or extrarenal losses are controlled but surgical convalescence still can not proceed because of gastrointestinal difficulty requiring prolonged intravenous feeding, a special nutritional setting is introduced. An example is to be found in such a situation as foreshortening of the bowel, malfunctioning gastrointestinal anastomosis, esophageal obstruction or chronic peritoneal infection with ileus.

Total intravenous feeding is never wholly

successful in the sense of providing sufficient calories and nitrogen to achieve anabolism, resumption of weight and strength. It appears that introduction of food into the gastrointestinal tract, as planned by nature is still necessary. It is important to emphasize that, in certain surgical conditions, introduction of food into the gastrointestinal tract may be done by means of gastrostomy or jejunostomy making prolonged intravenous feeding unnecessary. Occasional failure to recall the importance of this procedure or inability to perform this operation with the accuracy and gentleness required has produced tragic results.

When such an operation is inadvisable because of more distal disease in the gastrointestinal tract, prolonged intravenous feeding may be undertaken with the following general rules in mind.

Water, sodium, chloride and potassium should be provided daily.

The administration of water and salt each day should be carefully gauged by measured observation of the patient's weight and of extrarenal losses. The patient should not gain or lose weight rapidly. He should be expected to lose about 150 to 200 gm a day as his fat stores are burned.

One whole blood transfusion should be given approximately each week to provide trace minerals, electrolytes and erythrocytes not found in pharmacologic solutions.

Adequate intake of vitamins is vital.

If the patient is unstressed, he will anabolize from intravenous protein hydrolysate if adequate calories are also supplied. The provision of amino acids in the form of protein hydrolysates finds its maximal usefulness here.

The provision of adequate calories is a challenge. Concentrated glucose solution given into a caval catheter and the use of alcohol and fat emulsion may all be useful.

But, above all the patient should begin enteral ingestion of food as soon as possible. During the transition phase one should remember that parenteral provision of glucose "kills appetite." In a chronically ill patient whose stomach is not accustomed to accepting food and who has lost the habit of appetite, it is essential to omit the provision of intravenous calories for a few days so as to stimulate appetite as the enteric channel is reopened.

Preoperative Bodily Depletion. The surgeon frequently must operate upon patients depleted by chronic disease. Commonest among these are chronic sepsis gastro-

intracapsular subcapital femoral neck fracture with its minimal metabolic response is less apt to heal to good union.

The extensive wound of war may take any anatomic form. Very typical of a large number of the seriously wounded is a wound due to multiple penetrating fragments of artillery, mortar or mine missiles and involving injuries to the skeleton, the body cavities and hollow viscera. The result is massive blood loss, contamination of injured tissues and large cross-sectional trauma.

From the evidences available, the endocrine response appears also to be massive. The amount of adrenal medullary activity observed in shocked soldiers is great and this would lead us to believe that the cortical steroid output also is imposing although fewer data are available. Eosinophil counts are low for many days and when they gradually resume a higher value they may then be observed to be at extremely high levels (3000 to 4000 cells per cu. mm.) for many days or weeks. The amount of surgery involved is great as fractures must be set, bleeding stopped, devitalized tissue debrided and hollow viscera either repaired or exteriorized. The actual extent of the trauma, viewed as a physiologic insult to the patient, is the product of the initial cross-sectional trauma involved, the total blood lost, the duration of time which ensues between the initial injury and the completion of definitive surgery and the details of the definitive surgery itself. The latter includes débridement and immobilization.

The metabolic response which ensues is, therefore, doubly large, not only because the trauma is large but because it characteristically occurs in the athletic, healthy and well-nourished young adult male. Remarkable degrees of body wasting are observed. The patient may lose 25 per cent of his body weight in the first month. If sepsis can be controlled and visceral function (particularly renal function) maintained, the patient will convalesce. If he finally has access to adequate oral nutrition. Only through this medium can convalescence be assured.

Special problems of shock, the blood volume and renal failure present themselves regularly among the severely wounded. The three are interrelated for the clear reason that an accurate knowledge of blood volume physiology is essential to the treatment of shock, renal failure usually follows shock and the post-trauma metabolism is a

vicious cause of hyperkalemia in this setting.

Continued oligemia is the commonest cause of continued shock. The term "irreversible shock" should not be used in the case of these patients. More often than not it merely hides ignorance of other factors such as pneumothorax, hemothorax or continued uncontrolled hemorrhage all of which are susceptible to treatment whereas the concept of irreversible shock only connotes hopelessness.

Once shock has developed the amount of transfusion needed to restore the circulation is greater than the established deficit be it estimated by history or measured directly.

If oliguria persists the steps outlined should be followed with care to avoid overloading the patient with intravenous therapy.

Burns. The metabolic variants of a burn are most clearly seen in two respects. First, the water and salt picture is different from that associated with any other form of trauma. A major burn produces a large accumulation of edema under the area of thermal trauma, owing to thermal damage to capillaries. This segregates a large volume of water and salt which is derived from the plasma volume, reducing the blood volume with a resulting high hematocrit reading. Oligemic shock supervenes unless adequate plasma volume support is carried out with some sort of colloid-containing fluid, of which plasma or one of the plasma expanders is by far the most commonly used. When given into the plasma to maintain plasma volume these in turn leak out in part into the burn. The result is a large accumulation of water and salt which produces an early weight gain and strongly positive water and salt balances. Positive sodium balances in patients with major burns, early and adequately treated, may range between 500 and 1500 mEq in the first forty-eight hours depending on details of therapy. The weight gain which results is accordingly unique. It averages, in our experience, very close to 10 per cent of the initial body weight of the patient and this correlates with the fact that the extracellular fluid space is expanded to approximately twice normal. After two to five days, this fluid is excreted, weight rapidly falls and a convalescent pattern demanding caloric intake and more reminiscent of other forms of trauma is resumed. If renal insufficiency occurs and diuresis does not take place, convalescence is interrupted and recovery is rare.

overfed, obese man or woman with a tremendous omentum full of fat, decreased diaphragmatic excursion and flabby muscles. It is rightly emphasized by the nutritionists that the great hazard of the American population is not malnutrition but overnutrition and this is very true in surgery.

Infection. Acute sepsis without trauma has in itself many of the metabolic and endocrine aspects of acute trauma. There is a speeding up of the metabolic wheel, resulting in rapid loss of weight due to oxidation of fat and loss of nitrogen. There is an adrenal secretory increase and eosinopenia. There is also a clear-cut tendency to retain water and sodium. If sepsis complicates surgery and is rapid in onset after surgery there is a marked intensification of the clinical and metabolic effects of trauma. The patient looks much sicker, his fever is higher, the nitrogen loss is increased and body wasting is even more rapid. If the infection is quickly brought under control, as by drainage of an abscess, exteriorization of damaged bowel or effective administration of antibiotics, the patient will then pass into a recovery phase quite normally.

When the sepsis exists for a longer period, as in unresolved peritonitis, infected open wounds, or osteomyelitis after fracture, the continuation of sepsis amounts to an indefinite prolongation of the catabolic phase of metabolism. With continued sepsis and purulent discharge, even though the temperature be normal, the pulse is elevated and measurements of metabolism show that the eosinophil count is down, the serum steroid level remains elevated, wound losses may be considerable and the urinary nitrogen loss persists. The presence of sepsis thus prolongs the catabolic phase after trauma and, more importantly, postpones for as long as sepsis is active the corticoid withdrawal phase and resumption of normal anabolism.

This fact has several extremely important corollaries in clinical management. The obligatory catabolism of sepsis in its acute febrile phases seems to be a counterpart of the obligatory catabolism following injury in that it is difficult to affect either the clinical course of the disease or the rate of nitrogen loss in the urine by the provision of large intakes. But as the acute infection is initially controlled, the patient quickly passes into a period in which intake is of critical importance. If chronic infection is present, the provision of this intake may be difficult and threatening nutritional deficits develop rapidly. A ready example is found

in the late burn with infected open wounds. After only a week or two of thoughtless neglect, such a patient will show anemia, avitaminosis, hypoproteinemia and edema. Under such circumstances the administration of large intakes may be extremely beneficial in reducing the rate of body wasting, even though anabolism has not yet been produced. Patients with chronic septic processes furnish the chief examples in general surgery of the usefulness of forced feeding, an indwelling gastric tube being used when needed.

Fractures and Extensive Wounds. Certain endocrine and metabolic features of fractures, extensive wounds and burns are worthy of mention because each has endocrine and metabolic characteristics which set it apart from the stress of primary elective anesthetized soft tissue trauma. All three are examples of the importance of the wound in determining the depth, duration and details of early catabolism despite the similarity of their initial endocrinology.

Midshaft fractures in young individuals produce a very intense nitrogen catabolism which far outlasts the early period of endocrine activity or the period of starvation. Changes in eosinophils and blood and urine steroids may be no more prolonged than after soft tissue trauma. The prolonged loss of nitrogen, calcium and phosphorus is quite remarkable. The loss of calcium and phosphorus is traceable in part to the skeletal immobilization which is intrinsic in treatment and in part to release of these electrolytes from the fracture site itself. The more prolonged nature of the wound healing process required to restore the injured part to tensile integrity is apparently associated with a much more prolonged phase of nitrogen loss. It is for this reason that body wasting is more noticeable and that a much longer late phase of refeeding is essential. There is also good evidence that if intakes can be maintained at a maximal level after the first week or so the net loss from the body is reduced even though the absolute nitrogen excretion rate is increased.

Fractures which produce little systemic response are most clearly seen in the intracapsular subcapital femoral neck fracture. Here a fracture which occupies as much bone cross section as a midshaft fracture is associated with very little muscle disorganization or hematoma because of its intracapsular position. The relatively minor blood supply of the region is noteworthy. It is possibly a significant correlation that the

limited time not to exceed five days. The patient should be kept on antibiotics and given suitable gastric antacid therapy to avoid the commonest complications which are sepsis and bleeding peptic ulcer. At the end of that time the hormone should be "tapered off" and the patient's own adrenal response tested by the response to ACTH and by appropriate urine steroid measurements. Precise localization and assessment of the defect is then possible. Prolonged administration of ACTH or cortisone results in relative adrenal insufficiency when the drug is stopped. This must be taken into consideration in all patients operated upon after prolonged administration of these hormones.

Renal Failure The subject of metabolism in surgical patients with acute or chronic renal disease is one of considerable importance in present-day surgery. Lower nephron nephrosis otherwise referred to as ischemic nephrosis post-traumatic renal insufficiency pigment nephrosis the crush syndrome or tubular degeneration, is most commonly observed in patients after wounds or operations. All these terms are to some extent synonymous and when applied to the severely injured patient or the patient who has been in shock after surgery they indicate minor clinical variations in a renal picture most simply referred to as acute renal failure. Over two-thirds of the incidences of occurrence of this syndrome arise from either surgical or obstetric episodes. The commonest causes are massive injury with hemoglobinuria, myoglobinuria and shock, surgical shock itself mismatched transfusions and obstetric complications involving hemorrhage such as premature separation of the placenta.

The treatment of such patients centers on early diagnosis. The diagnosis that the patient is passing into a phase of renal disease, rather than oliguria due to hypotension and dehydration, rests upon the finding of a small and falling urine output at fixed specific gravity despite the restoration of normal blood pressure and hydration, with a rising blood urea and serum potassium value.

Once this diagnosis is suspected the patient should be treated by "balance" avoiding any sudden weight gains or losses which indicate sudden shifts in body water volume. Early recognition of the problem and adherence to this principle during the first forty-eight hours may be lifesaving. Those in charge of the patient all too frequently

suffer under the delusion that large water or salt infusions will "open" or "flush out" the kidneys. If hydration, blood volume and blood pressure are normal the kidneys will resume function when they can large infusions (over 1000 cc. per day) only threaten life by overburdening the circulation.

Conservative therapy, avoiding overload of the patient with water and salt, maintaining water balance, maintaining normal blood volume and avoiding the prolongation of sepsis will be followed by spontaneous diuresis and recovery in a substantial fraction of the patients.

Increase in the blood level of urea and related compounds does not seem to do the patient a great deal of harm. The accumulation of potassium in the extracellular phase is the chief threat to life in this disease. The accumulation of metabolic acids producing acidosis is also extremely hazardous. The rate of accumulation of both is greatly accelerated after injury and they act synergistically. When these occur and the patient continues to be oliguric, treatment by ordinary measures is often unavailing. The patient should be referred or transferred to a center where renal insufficiency is frequently dealt with and where methods of artificially dialyzing these substances out of the blood are available. Early transfer is safe or feasible; procrastination based on false hope is fatal.

Summary. No single summarizing statement is possible. Normal endocrine and metabolic processes underlie surgical convalescence. Their understanding as normal phenomena is the key to a rational and effective treatment of the metabolic abnormalities which are so common in all fields of surgery.

READING REFERENCES

- Bland, J. H.. *Clinical Recognition and Disturbances of Body Fluids*. Philadelphia, W. B. Saunders Company, 1956.
- Bliss, E. L., Sandberg, A. A., Nelson, D. H., and Elk Nes, K.. The Normal Levels of 17 Hydroxycorticosteroids in the Peripheral Blood of Man. *J. Clin. Invest.* 32:818, 1953.
- Blount, H. C., Jr., and Hardy, J. D. Thyroid Function and Surgical Trauma, as Evaluated by Iodine Conversion Ratio. *Am. J. M. Sc.* 234:112, 1952.
- Bonner, C. D. Eosinophile Levels as an Index of Adrenal Responsiveness. Factors That Affect Value of Eosinophile Counts. *J. A.M.A.* 148:634, 1952.
- Browne, J. S. L., Schenker, V., and Stevenson, J. A. F.. Some Metabolic Aspects of Damage and Convalescence (Abstract). *J. Clin. Invest.* 23:632, 1944.
- Cannon, W. B.. *Bodily Changes in Pain, Hunger*

Second, the nitrogen-calorie relation resembles fractures in that there is a very prolonged period of nitrogen loss loosely correlated with the duration of the unhealed wound. It long outlasts the period of steroidal activity. It lasts the length of time that the wound would ordinarily be expected to take to heal. If the open wound persists long after this time as small unhealed areas it will be extremely difficult to heal. Here as in the fracture, the provision of nutrition as soon as gastrointestinal acceptance occurs minimizes the body wasting.

Endocrine Disease. Shortly after the description of the importance of the adrenal steroids in postoperative endocrinology the concept became widespread that adrenal failure would turn out to be a common cause of difficulties in surgical convalescence. Such has not been borne out by subsequent experience. Outright adrenal failure in patients previously unsuspected of endocrine disease is quite rare, but its importance cannot be overestimated for the individual patient in whom it occurs and there are lesser degrees of abnormality which are more common.

Adrenal failure after operation is manifested by high fever, tachycardia, hypotension, oliguria, shock and coma. Administration of large amounts of blood, plasma, antibiotics, water and salt is of some slight assistance but results are remarkably nonspecific. If the patient has some degree of visceral congestion initially this massive administration of parenteral fluids will ultimately be fatal. By contrast, the response to intravenous hydrocortisone is so dramatic as to constitute one of the spectacular experiences of a surgical career.

The diagnosis of adrenal failure must, therefore, be considered in any patient who is not doing well after surgery. The eosinophil count should be made and, whenever possible, measurements of the blood and urine steroids should be carried out. If within three days of surgery and with an extremely ill patient, the eosinophil count is over 50 per cu. mm., a very high degree of suspicion should be aroused.

Whether or not the eosinophil count is elevated, and if there is reasonable suspicion that other forms of hypotension and oliguria have been adequately considered and treated, a specific therapeutic trial with hydrocortisone given intravenously is indicated. It is always unfortunate when "blind" therapy is given for any condition in medicine or surgery. There are certain circumstances however in which the hazard is so

small, and the possible benefit so great if the diagnosis turns out to be correct, that blind therapy is justified. In the postoperative patient who has been accurately transfused, whose water balance has been carefully considered and adequately handled, whose airway and pulmonary and cardiac function show no important defects and who is bordering on shock or is in shock, the administration of hydrocortisone intravenously in a dose of 100 mg. in 500 cc. of a 5 per cent solution of dextrose in water is a justified procedure. A positive response is of tremendous significance.

In an acute emergency the employment of cortisone or hydrocortisone is more rational than ACTH since the latter is unavailable if there is significant adrenal disease.

When the administration of hormone to surgical patients is used as a disguise for inadequate diagnosis, it is to be deplored. When intelligently applied it is of course useful. The cause of postoperative adrenal failure is most commonly subclinical Addison's disease or acute adrenal hemorrhage; the latter often associated with the use of anticoagulants.

When the adrenals are removed surgically as in the treatment of carcinoma, one deals with postoperative hypoadrenocorticism which is treated with specific administration of adrenocortical hormones; the therapeutic pattern of which should mimic the normal metabolic and endocrine response to surgery.

Myxedema and diseases of the pituitary also are accompanied by postoperative "metabolic decompensation." If these are present, administration of appropriate hormones either thyroid, ACTH or cortisone, should be carried out.

Chronically ill patients, such as the patient with the late burn or the patient in the later phases of peritonitis with localized abscess formation will frequently carry a resting high eosinophil count and, when operated upon, the eosinophil count will rise to extremely high levels such as 2000 to 3000 per cu. mm. This does not indicate adrenal failure. Adrenal failure is manifested not only by a high eosinophil count, which is an isolated laboratory observation also producible by a variety of other mechanisms including allergy, but by actual metabolic changes the most striking of which are failure to maintain blood pressure, unregulated loss of salt in the urine and fever.

Once the administration of hydrocortisone is started, it should be maintained for a

ANESTHESIOLOGY

By JOHN ADRIANI M D

JOHN ADRIANI went from his home in Bridgeport Connecticut to Columbia University for his college and medical school degrees. A surgical internship and a fellowship in physiology paved the way for his training in anesthesiology. His interest has a broad base and recognizes the mutual cooperation necessary between surgeon and anesthesiologist for the best care of the patient. He has spent his professional life thus far in New Orleans where he directs anesthesiology in the medical schools of Tulane and Louisiana State University.

Anesthesia is an adjunct to patient care. Surgery as it is now practiced, would hardly be possible without it. Although physical agents such as pressure, cold and electric currents may be used to induce anesthesia, the practical methods for its production entail the use of chemical substances which depress the activity of nervous tissue. These chemical agents either act systemically and depress the central nuclei or they act locally and block the activity of a nerve fiber when applied directly to its surface. Thus, anesthesia is recognized as *general* or *systemic* and *local*. More precisely local anesthesia is referred to as *regional* or *conduction* anesthesia. Regional anesthesia is subdivided into types named according to the site of application of the drug used to produce it, namely *spinal*, *epidural*, *paravertebral*, *nerve block*, *field block*, *infiltration* and *topical*.

To be surgically useful, an anesthetic drug or method of producing anesthesia must fulfill two purposes: (1) it must abolish reflex activity and other responses to stimuli and (2) it must provide muscle relaxation. A third requirement, loss of consciousness is desirable but is not always necessary.

GENERAL ANESTHETICS

The general anesthetics are volatile substances which are administered by inhalation or nonvolatile drugs which are administered by routes other than inhalation. The volatile drugs differ in pharmacologic characteristics from the nonvolatile. The members in each group are similar pharmacologically and are used for the same purposes.

The volatile drugs are complete anesthetics. They cause a blockade along the path from the periphery to the pain perception centers. The loss of sensibility is accompanied by a loss of consciousness. Loss of muscle tone of varying degrees is obtained, depending upon the potency of the drug. Volatile anesthetics are inert; that is, they are not altered by the cells. They are eliminated unchanged by exhalation. They are gases, or highly volatile liquids, which boil below 60° C. With the exception of nitrous oxide, currently used drugs are hydrocarbons, ethers or halogenated hydrocarbons. Three gases, nitrous oxide, ethylene and cyclopropane, and six liquids, ether, vinyl ether, chloroform, ethyl chloride, Fluothane and trichlorethylene, are used.

The nonvolatile drugs, on the other hand,

- Fear and Rage. New York, D Appleton & Company 1915
- Collier F. A., Campbell, K. N., Vaughan, W. H. and Job L. V. Postoperative Salt Intolerance. *Ann. Surg.* 119:533, 1944.
- Cope, O. and others: Metabolic Rate and Thyroid Function Following Acute Thermal Trauma in Man. *Ann. Surg.* 137 165 1953.
- Cuthbertson, D. P.: Observations on the Disturbance of Metabolism Produced by Injury to the Limbs. *Quart. J. Med. N.S.* 1 233, 1933.
- Davenport, Horace W. The ABC of Acid-Base Chemistry Chicago The University of Chicago Press, 1950
- Dudley H. A., Boling, E. A., LeQueune, L. P., and Moore, F. D. Studies on Antidiuretics in Surgery: Effects of Anesthesia, Surgery and Posterior Pituitary Antidiuretic Hormone on Water Metabolism in Man. *Ann. Surg.* 140 354, 1954.
- Frankson, C., Gemzell, C. A., and von Euler U. S.: Cortical and Medullary Adrenal Activity in Surgical and Allied Conditions. *J. Clin. Endocrinol.* 14 608, 1954
- Hardy James D.: Surgery and the Endocrine System. Philadelphia, W B Saunders Company 1952.
- Howard, J. E. Protein Metabolism during Convalescence after Trauma. *Recent Studies. Arch. Surg.* 59:106, 1945.
- Howard, J. M., and others: Studies of Adrenal Function in Combat and Wounded Soldiers. *Ann. Surg.* 141:304 1955.
- Hume, D. M. The Role of the Hypothalamus in the Pituitary Adrenal Cortical Response to Stress. *J. Clin. Invest.* 28 790 1949
- Hume, D. M., and Nelson, D. H.: Corticoid Output in Adrenal Venous Blood of the Intact Dog. *Fed. Proc.* 13:73, 1954.
- Ingle, D. J. Ward, E. O., and Kutzenau, M. H. The Relationship of the Adrenal Glands to Changes in Urinary Non-protein Nitrogen following Multiple Fractures in the Force-Fed Rat. *Am. J. Physiol.* 149:510 1947
- LeQueune, L. P. Fluid Balance in Surgical Practice. London, Lloyd Luke, 1954, 128 pp.
- Lockwood, J. S. and Randall, H. T. The Place of Electrolyte Studies in Surgical Patients. *Bull. New York Acad. Med.* 25:228, 1949
- Merrill, J. P., Levine, H. D. Somerville, W. and Smith, S., III. Clinical Recognition and Treatment of Acute Potassium Intoxication. *Ann. Int. Med.* 33:797 1950.
- Moore, F. D. The Adaptation of Supportive Treatment to the Needs of the Surgical Patient. *J.A.M.A.* 141:646, 1949.
- Moore, F. D.: Burns. An Annotated Outline for Practical Treatment. *Symposium on Specific Methods of Treatment. M. Clin. North America* 36:1201 1953.
- Moore, F. D.: Bodily Changes in Surgical Convalescence. I. The Normal Sequence—Observations and Interpretations. *Ann. Surg.* 137 289 1953.
- Moore, F. D. Low Sodium Syndromes of Surgery Outline for Practical Management. *J.A.M.A.* 154 879 1954.
- Moore F. D.: The Significance of Weight Changes after Trauma (Editorial). *Ann. Surg.* 141:141, 1955
- Moore, F. D. and Ball, M. R.: The Metabolic Response to Surgery Springfield, Ill., Charles C Thomas, Publisher 1952.
- Moore, F. D., and others: Discussion on the Endocrine Response to Trauma. *Proc. Roy. Soc. Med.* 48 817 1955.
- Moore, F. D. and others: Studies in Surgical Endocrinology I. The Urinary Excretion of 17 Hydroxycorticoids and Associated Metabolic Changes, in Cases of Soft Tissue Trauma of Varying Severity and in Bone Trauma. *Ann. Surg.* 141 145 1955
- Nicola, J. A., Wilson, P. D., and Umberger C. J.: Observations on Adrenocortical Function in Patients Undergoing Operations upon the Bones and Joints. *Surg., Gynec. & Obst.* 99 1 1954
- Roberts, K. E., Randall, H. T., Philbin, P., and Lipston, R.: Changes in Extracellular Water and Electrolytes and the Renal Compensation in Chronic Alkalosis, as Compared to Those Occurring in Acute Alkalosis. *Surgery* 38:590 1954
- Selye, H. "Conditioning" Various "Permissive" Actions of Hormones. *J. Clin. Endocrinol.* 14 123, 1954
- Simpson, S. A., and others: Konstitution des Aldosterons, des neuen Mineralocorticoids. *Experientia* 10 132, 1954
- Steenburg, R. W. A Study of the Free 17 Hydroxycorticosteroids on the Peripheral Blood of Surgical Patients. *Surgical Forum, American College of Surgeons*, 1954. Philadelphia, W. B. Saunders Company 1955, p. 593.
- Steenburg, R. W., Ganong, W. F. and Moore, F. D.: The Effect of Extra-adrenal Factors on the Free Serum 17 Hydroxycorticoids in Surgically Stressed Experimental Animals. *Surgery* 38:92, 1955.
- Steenburg, R. W., Lennihan, R., and Moore, F. D. Studies in Surgical Endocrinology II. The Free Blood 17 Hydroxycorticoids in Surgical Patients; Their Relation to Urine Steroids, Metabolism and Convalescence. *Ann. Surg.* 143 180, 1956.
- Thorn, G. W., Jenkins, D., and Lakflaw J. C. The Adrenal Response to Stress in Man. In *Recent Progress in Hormone Research. Proc. of the Laurentian Hormone Conference. G. Picus, Editor* New York, Academic Press, Inc., 1953, Vol. 8, p. 171
- Tyler F. H. and others: The Role of the Liver and the Adrenal in Producing Elevated Plasma 17 Hydroxycorticosteroid Levels in Surgery. *J. Clin. Invest.* 33:1507 1954
- Wangensteen, O. H., and Zimmerman, B. Observations on Water Intoxication in Surgical Patients. *Surgery* 51:654, 1953.
- Wilkinson, A. W., Billing, B. H. Nagy G., and Stewart, C. P.: Excretion of Chloride and Sodium after Surgical Operations. *Lancet* 1:540, 1949
- Wilson, G. M. and others: Metabolic Changes Associated with Mitral Valvuloplasty. *Circulation* 9: 196, 1954.

reservoir and the mask prevents return of expired gases and obviates rebreathing.

4 The closed method (Fig. 3) permits total rebreathing of gases and vapors. An inhaler composed of a mask and breathing bag is provided with a filter containing an absorbent (soda lime) for carbon dioxide. Oxygen to meet the metabolic requirements of the subject is supplied from a reservoir. Both the semiclosed and closed types of apparatus must be provided with metering devices (flow meters) to supply gases and vapors in desired proportions. The closed system is almost universally used for the administration of inhalation anesthetics in present-day practice.

Nonvolatile drugs are administered by routes other than inhalation. No satisfactory substances exist which can be administered orally which induce satisfactory surgical anesthesia. Most nonvolatile drugs used in conjunction with surgical anesthesia are administered rectally or intravenously. The narcotics and certain of the hypnotic drugs are administered subcutaneously or intramuscularly.

Anesthetics are protoplasmic poisons with three notable characteristics they have a special predilection for nervous tissue, they ultimately affect all protoplasm as concentrations are increased and their action is reversible within limits. Once the drug is removed, the physiologic state of the cell

reverts to normal. This reversibility of action is extremely important. Overdosage of central nervous system depressants paralyzes the medullary centers. Death results from respiratory failure. The nervous system is depressed from above downward, that is the cortex first, the midbrain next and then the medulla. If artificial respiration is promptly instituted and maintained after medullary paralysis occurs the circulatory system remains intact. A dose exists for each drug above that which causes paralysis of the medullary centers which is lethal even though effective artificial respiration is performed. These doses vary from one drug to the other. What they are for man is not known. The concentration of ethyl ether which depresses the heart is several times that which causes medullary paralysis. On the other hand, concentrations of chloroform which depress the myocardium are close to those which paralyze the respiratory centers.

The progressive depression of the nervous system gives rise to a succession of reflex changes which are clinically useful in estimating the concentration of the drug in the nervous system. The disappearance and re appearance of these reflexes as the concentration is varied, serve as a useful guide for the administration of anesthetics. The manner in which volatile drugs influence these reflex changes differs considerably from that of nonvolatile drugs. The changes caused by

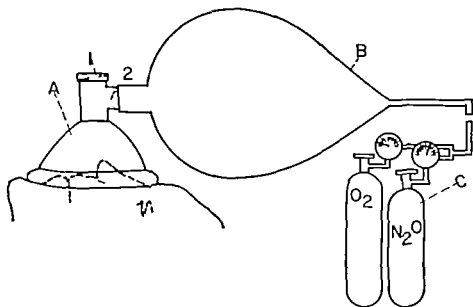


Figure 2. Semiclosed technique. The semiclosed apparatus consists of (A) a mask and (B) a rubber breathing bag of 4 or 5-liter capacity and a source of anesthetic gas and oxygen (C). An exhalation valve (1) on top the mask allows exhaled gases to pass to the outside atmosphere. A valve (2) may be interposed between the mask and the reservoir to prevent rebreathing. Under these circumstances the minute volume exchange of the patient must be supplied continuously without interruption. This second valve is omitted in most apparatus used for anesthesia.

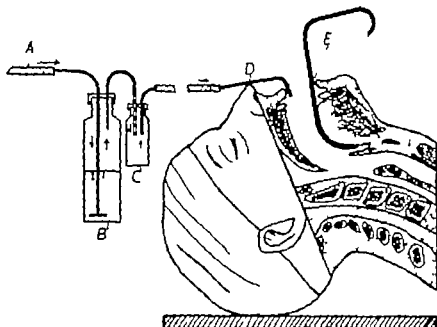


Figure 1 Insufflation technique. The volatile liquid is vaporized by bubbling air, oxygen or a mixture of an anesthetic gas and oxygen (A) through the container (B) and the mixture is conducted into the upper respiratory passages. In oral and pharyngeal surgery a cannula (D) conducts the anesthetic mixture into the mouth. A catheter may be used for nasopharyngeal or direct insufflation into an intratracheal catheter or cannula. (From Adriani, *Techniques and Procedures of Anesthesia*. Courtesy Charles C Thomas, Publisher.)

are incomplete anesthetics. None completely blocks the passage of impulses from the periphery to the thalamic nuclei. A hypnotic state, from which the patient may be roused, is obtained with therapeutic doses. Larger doses produce a more profound state of depression accompanied by amnesia and partial obtunding of reflex activity. Doses which abolish reflex activity and produce anesthesia in a surgical sense depress the medullary centers unduly and give rise to circulatory and respiratory depression. The nonvolatile drugs, unlike the volatile can not be used as sole agents for surgical anesthesia. In present-day practice it is common to combine nonvolatile with volatile drugs. The former provide sedation or basal narcosis; the latter analgesia and suppression of reflex activity. One complements the other. The nonvolatile drugs are liquids whose boiling points are too high to permit adequate vaporization for inhalation, or they are solids. Chemically the majority are aliphatic compounds, derivatives of urea or alkaloids. The aliphatic compounds are alcohols, halogenated alcohols or halogenated aldehydes. Numerous derivatives of urea are available, the most useful of which are the barbiturates. The alkaloids are narcotic substances that is, they possess both analgesic and hypnotic properties. The analgesic power however is not sufficient to abolish reflexes and relieve sharp pain. Narcotics

thus possess little usefulness as surgical anesthetics. They do however induce a euphoric state which is utilized for preoperative sedation. An additive effect is obtained when narcotics are combined with less potent anesthetics such as nitrous oxide, which thereby facilitates their use. The narcotics are almost universally used for control of pain preoperatively or postoperatively.

METHODS OF ADMINISTERING GENERAL ANESTHETICS

Volatile anesthetics are administered by one or a combination of four different methods.

1. The open or drop method is adaptable for liquids. The liquid is vaporized on a gauze mesh, becomes mixed with air which serves as a vehicle and source of oxygen, and is inhaled.

2. The insufflation technique (Fig. 1) is suitable for both gases and vapors. A stream of gas or vapor mixed with air or oxygen, is conducted into the oropharynx, nasopharynx or trachea by means of a catheter or a tube.

3. The semiclosed method (Fig. 2) is used for gases or vapors mixed with air or oxygen. A preformed mixture of gases and vapors is inhaled from a closed mask which communicates with a reservoir (breathing bag). The exhalations pass through a valve on top of the mask provided for the purpose. A directional valve interposed between the

2 because the smaller muscles throughout the body including those of the eye, lose their tone. The pupils are, then centrally fixed. Respiration is altered little in planes 1 and 2 because both the intercostal muscles and the diaphragm remain active. As plane 3 is reached, intercostal activity is lost and respiration becomes diaphragmatic in quality. Musculo tone is diminished throughout the body. The pupils increase in size but are not widely dilated. Corneal reflexes are obtunded or abolished. There may be some elevation in pulse rate and a slight fall in blood pressure. Surgery necessitating relaxation of the large muscles may be performed in plane 3. In plane 4 complete relaxation of the large muscles and loss of all reflexes occur. Diaphragmatic activity is diminished the pupils are widely dilated. Respiration is diaphragmatic, inspiration is quick, short and jerky and expiration is prolonged. The minute volume exchange is diminished.

In stage IV the medullary centers are in active. The respiratory center is the first of the medullary centers to become completely depressed and apnea results. Unless artificial respiration is instituted promptly death results from the ensuing asphyxia. With most of the anesthetic drugs employed, the heart continues to beat after the onset of this stage if artificial respiration is instituted and maintained as soon as apnea occurs.

The cough reflex disappears as stage III is reached. The vomiting or gag reflex disappears at the same time. Tracheal and bronchial reflexes disappear in planes 2 and 3. Reflexes initiated by traction on the abdominal viscera, pleura, hilum, trachea or bronchil or by stimulation of the perineal structures disappear in plane 3 but may persist even into plane 4. When the anesthetic is discontinued, reflex activity returns in the reverse sequence. After the administration of ether for a long period, however the order of return of reflexes is variable. Anoxia, carbon dioxide excess, heavy sedation and basal narcosis with nonvolatile drugs modify and invalidate the signs. The signs tend to vary with the extremes of age.

The electrical activity of the cerebral cortex is suppressed by anesthetics and hypnotics. The reduction in the cortical potentials is in proportion to the degree of depression. The variations in potentials are demonstrable on the electroencephalograph. During ether anesthesia, seven typical wave and voltage patterns are discernible in descent from consciousness to overdosage. The pattern is different for each drug. The

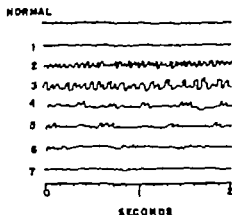


Figure 4 Changes (EEG) in electrical activity of the cerebral cortex during ether anesthesia. Seven levels of activity are consistently apparent. As anesthesia deepens the normal fast rhythm is converted to a slower one with increased voltage. Then periods of suppression with intervening bursts of activity are seen. Finally in deep anesthesia, there is a loss of activity characterized by flattening of the wave.

electroencephalogram may be used to guide the anesthetist in determining depth of anesthesia. Variations in activity caused by non-volatile drugs and combinations of volatile and nonvolatile drugs may be detected by the electroencephalograph. There are however pitfalls which may befall one in the use of the electroencephalograph for determining depth of anesthesia. Artifacts, technical difficulties with the apparatus, skill in interpretation of the record and the lag between the time of establishment of the central and of the circulatory effects of the drug limit the usefulness of the encephalogram for routine purposes (Fig 4).

ABSORPTION AND ELIMINATION OF ANESTHETICS

The absorption and elimination of volatile anesthetics are influenced by the following factors (1) the tension of concentration in the inspired mixture, (2) the tidal exchange (3) the minute volume exchange (4) the functional residual air volume (5) the solubility coefficient of the drug in blood, (6) the rate of diffusion through the alveolar membrane (7) the minute volume blood flow through the lungs (8) the blood flow through the tissues and (9) solubility in the tissues.

A pressure gradient is established from the inspired gases to the blood and thence to the cells. Obviously the higher the gas tension in the blood, the greater the pressure gradient between it and the cells and the more rapid the saturation of the tissues. An adequate tidal exchange is necessary to allow proper mixing of the gas or vapor with

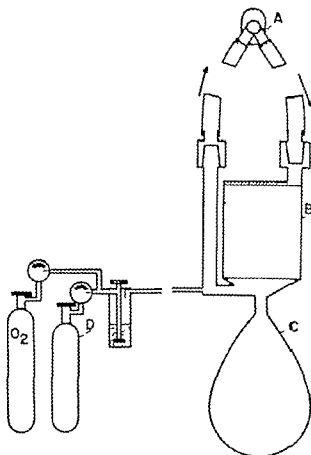


Figure 3 Closed or rebreathing technique. The closed type apparatus consists of a mask (A) and a rubber breathing bag of 4- or 5-liter capacity (C). A canister (B) containing soda lime is interposed between the mask and breathing bag. In the circle filter the gases are conducted through the canisters by corrugated tubes. Valves at the inlet and outlet of the canister permit a unidirectional flow. Gases and vapors (D) are admitted through an inlet as required.

volatile drugs are more labile and more pronounced. Presumably this is due to the inertness of volatile drugs. The intracellular concentrations are closely correlated with blood concentrations. Variations in blood concentrations are quickly reflected in the cellular concentration. This does not appear to be the case with nonvolatile drugs.

SIGNS OF ANESTHESIA

Anesthetic drugs affect all physiologic systems to some degree. However the changes in the nervous system, respiratory system and circulatory system are the most apparent. These three systems are under the constant surveillance of the anesthetist. The character, rate and depth of respiration, the pulse and blood pressure, the reflex changes and the variations in muscle tone are all observed closely.

Four stages of anesthesia are recognized in administering volatile drugs. One stage

merges into the next as the cellular concentrations increase. There is no abrupt line of demarcation between stages.

In stage I referred to as the *stage of analgesia* the patient remains conscious but is unable to perceive painful stimuli. Reflexes remain active and the subject attempts to withdraw from the noxious stimuli, even though he does not interpret them as pain. The sensorium remains clear and the subject is aware of his surroundings. This stage is of limited usefulness because the patient is uncooperative.

Stage II is called the *stage of delirium*. The cerebral cortex is depressed and consciousness is lost. Cortical inhibitions are released as control of various centers is lost. The lower, more primitive centers then exert their influence. This stage is characterized by exaggerated reflexes, increased muscle tone, irregular respiration, incoordination, struggling and disorientation. Stimulation of any sort aggravates these undesirable manifestations. Excitement is frequent and signs of sympathetic stimulation are present. This stage is not only of no value, but is one which is avoided and transcended as soon as possible. Fear and apprehension must be allayed and all manipulations and external stimulation held in abeyance until this stage has been passed.

Stage III is the stage of *surgical anesthesia*. Anesthesia is completely established and superficial reflexes are abolished. Deep reflexes and those associated with vital functions remain active. Voluntary movements are absent. Tapping the eyelid no longer evokes the so-called lid reflex. The lid reflex must not be confused with the corneal reflex commonly employed by physiologists as a guide to depth of anesthesia in laboratory animals. The corneal reflex is objectionable as a guide for depth of anesthesia because ulcers result from repeated touching of the cornea. The medullary centers remain active. Respiration is automatic, rhythmical and resembles that noted in normal sleep because cortical and subcortical influences are abolished and it is under sole control of the respiratory centers. Pulse and blood pressure which are usually elevated in stage II return to preanesthetic levels. Stage III, since it is broad, is subdivided into sub-stages called planes. Stages are indicated by roman numerals, planes by arabic numerals. In the first plane there is no loss of muscle tone. Ocular movements continue when the lids are retracted and light enters the pupil. These ocular movements disappear in plane

tics are due to asphyxia. As is the case with nitrous oxide narcotics basal narcotics ether vinyl ether chloroform Fluothane or trichlorethylene is used as a fortifying agent. Alone, it is suitable for operations requiring pain relief without muscle relaxation. Ethylene is nonirritating to the respiratory tract and, like nitrous oxide, causes no significant physiologic disturbances if adequate oxygenation is maintained. It is used for induction of ether anesthesia. The chief objection is that it is flammable. The technique of administration is the same as that used for nitrous oxide. It is somewhat more potent than nitrous oxide in comparable circumstances.

Cyclopropane. Cyclopropane (trimethylene) is a stable flammable pleasant-smelling easily inhaled, nonirritating, gaseous hydrocarbon. Induction of anesthesia is rapid, requiring three or four minutes. Most of the gas, irrespective of the length of operation, is eliminated within ten minutes. Recovery of reflex activity occurs promptly in the operating room. All stages are traversed. When overdosage occurs respiratory failure precedes circulatory failure. Muscle relaxation adequate for abdominal surgery may be obtained without the use of adjuncts if one chooses muscle relaxants, such as curare and the synthetic relaxants, permit use of the upper planes of anesthesia. Cyclopropane is more potent than ethylene and nitrous oxide. A tension of 150 to 175 mm. Hg (20 to 25 per cent) in the alveoli is necessary for surgical anesthesia. Respiratory failure results when the tension is about 300 mm. Hg (40 per cent). The margin of safety therefore is wide. Anoxia is no problem because 20 per cent or more oxygen may be used at all times. The closed system with total rebreathing is used to administer the gas since it is highly potent and nitrogen need not be eliminated from the system. This is an advantage in view of the high cost and flammability. Anesthesia may be lightened and deepened quickly and at will. This is a distinct advantage in management of poor surgical risks.

Metabolic processes are not significantly disturbed. A slight insignificant elevation of blood glucose occurs, but there is no effect on the carbon dioxide combining power, liver or renal function.

Respiration is depressed, as a rule. The minute volume exchange is reduced, but in spite of this the arterial and venous blood are normally saturated with oxygen. Respiratory acidosis results from carbon dioxide

retention due to this hypoventilation. The most objectionable feature of the drug is the increase in cardiac irritability which causes arrhythmias. These may be ventricular in origin. Ventricular fibrillation may occur if epinephrine is used in conjunction with cyclopropane, because both drugs increase cardiac irritability. Myocardial depression does not occur and cardiac output is decreased only during deep anesthesia. An elevation in blood pressure is often noted during operation which is due to carbon dioxide retention. Postanesthetic nausea and vomiting are less frequent than with ether but they do occur. At the conclusion of anesthesia, hypotension is sometimes noted which is associated with the respiratory acidosis. At times excessive oozing of blood in the wound occurs. This is due to the elevation in blood pressure and local peripheral vascular dilatation. Bleeding and clotting times are not disturbed.

Vinyl Ether. Vinyl ether (Vinethene) is a highly volatile flammable liquid which boils at room temperature (28° C.). The vapor is nonirritating, easily inhaled and quickly produces surgical anesthesia. The drug is the unsaturated counterpart of ethyl ether. It is less stable than ether. Approximately 30 to 40 mm. Hg (4 per cent) are necessary in the alveoli for anesthesia, 60 mm. Hg (8 per cent) for respiratory failure. The drug is used as an induction agent for ether anesthesia by the open mask technique and for analgesia for minor surgical procedures. It is also used as a complementary agent to fortify nitrous oxide or ethylene. Vinyl ether is administered by the open drop method in short surgical procedures or in combination with nitrous oxide by the semiclosed method. The closed method is not satisfactory because difficulty is encountered in maintaining an even plane of anesthesia. Even though stage IV can be attained, relaxation is not adequate for major surgery. Induction and recovery are rapid (two to three minutes) and pleasant. Postanesthetic nausea and vomiting are infrequent. Vinyl ether is not chemically stable unless an inhibitor is added and the drug is protected from light, heat and air.

No clinically significant physiologic and metabolic disturbances occur during anesthesia. Hepatic or renal damage does not occur unless this agent is administered repeatedly or if administration is accompanied by anoxia. Vinyl ether is not used for procedures of over thirty minutes, or for those requiring muscle relaxation. The drug is not

the functional residual air. The functional residual air volume is important because it represents the total gas volume which comes into contact with the alveoli and which mixes with tidal air. When the functional residual air volume and the tidal volume are about the same the changes in alveolar concentration with each respiration are abrupt. This situation is encountered in children. A momentary increase or decrease in concentration is immediately reflected in the blood level and anesthesia lightens or deepens. If the functional residual air volume increases out of proportion to the tidal exchange mixing does not occur. Saturation and desaturation are impaired and take more than the usual time. An increase in minute volume exchange facilitates tissue saturation by increasing the total quantity of drug carried to the lungs.

The degree of depression depends upon the concentration of the drug in the brain. This in turn depends upon the alveolar tension. An equilibrium is usually established between the tension in the brain and the arterial blood before it is in the rest of the mass of body tissue. The passage of the drug from the blood to the brain is influenced by the cerebral blood flow, the cerebral vascular resistance and the ease of penetration of the drug through the blood brain barrier. Once equilibrium is established between the alveolar air and the brain anesthesia would be maintained at a given level were it not for the absorption of the drug by the other tissues. This necessitates the intermittent addition of drug to the inhaled mixture to replace the amount being drawn from the arterial blood by the muscles, fat and other non-nervous tissue.

Volatile anesthetics diffuse through the alveolar membrane in the same manner as do other gases and vapors. The drug does not alter the membrane otherwise it would be unsuitable clinically. Volatile anesthetics are often referred to as irritating, the inference being that the alveolar membrane is in some way injured. This is not so. Most volatile drugs particularly ether stimulate the vagal nerve endings in the pulmonary tissues and cause exaggerated breathing. Also the mucous glands in the respiratory tract are stimulated and excessive secretions accumulate. Both responses are mistakenly interpreted as "irritation."

INHALATION ANESTHETICS

Nitrous Oxide. Nitrous oxide often referred to as "gas," is a nonirritating, sweet

smelling, nonflammable gas of mild narcotic potency. It rarely yields anesthesia deeper than the first plane. The alveolar tension for surgical anesthesia varies between 625 and 650 mm. Hg (80 to 85 per cent). Induction and recovery are rapid—approximately three minutes. Nitrous oxide is not suitable for operations requiring profound anesthesia or muscle relaxation. Its effectiveness is increased when combined with morphine or other narcotics. basal narcotics induced with Avertin or thiopental, ether, vinyl ether, chloroform, Fluothane, trichlorethylene or the muscle relaxants. Unless thus fortified, suboxygation invariably occurs. Overdosage does not occur if the gas is administered at atmospheric pressure with more than 20 per cent oxygen.

Nitrous oxide is an effective analgesic. An alveolar tension of 365 to 380 mm. Hg (35 to 50 per cent) causes a high degree of analgesia. Its use as an analgesic is limited because little surgery can be performed effectively with analgesia. Nitrous oxide is used as an induction agent for ether anesthesia and in combination with basal narcotics in situations in which cautery, endotherms, endoscopes or other equipment which may be a fire hazard are required. It is also used for analgesia for obstetric or dental surgery. Nitrous oxide is administered by means of semiclosed inhalers in order to permit the elimination and replacement of nitrogen from the blood and tissues by the gas. Without anoxia, the drug disrupts none of the important vital functions. Desirable features are that it is nonflammable, nonirritating to the respiratory tract, relatively inexpensive and rapidly absorbed and eliminated. Postanesthetic nausea and emesis are infrequent. There is no justification in using the drug if anesthesia cannot be obtained without anoxia. Most fatalities caused by nitrous oxide are due to asphyxia. When administered in nonanesthetic concentrations there are no contraindications to its use.

Ethylene. Ethylene is a nonirritating, flammable, gaseous hydrocarbon with an ethereal odor and a mild narcotic potency. Pharmacologically it is similar to nitrous oxide. Anesthesia is characterized by rapid induction and recovery. The depth attained is rarely lower than plane 1 or the upper limits of plane 2. An alveolar tension of approximately 540 mm. Hg (75 per cent) is necessary for anesthesia. Well premedicated patients require less. As is the case with nitrous oxide, respiratory failure occurs only when anesthesia is complicated by anoxia. Fatal

trations and in situations in which a fire hazard exists. Surgical anesthesia results with alveolar tensions of 5 to 8 mm. Hg (1.5 per cent) and respiratory failure with 15 to 16 mm. Hg (2 per cent). The margin of safety is narrow. Induction of anesthesia is rapid. Preliminary agents, such as nitrous oxide or ethylene, are not required. Chloroform has been used in tropical areas because it is less volatile than are other drugs. As in the case of ether, simple equipment (open masks) may be used for administration. Exaggerated breathing results but to a lesser extent than that caused by ether. The extreme potency and low partial pressure necessary for anesthesia permit the use of air as a source of oxygen and as a vehicle in open methods.

Chloroform exerts a dual effect on the heart by depressing the myocardium and increasing irritability of the conductive tissues. Cardiac output is reduced as anesthesia deepens. Ventricular fibrillation occurs if epinephrine is given during chloroform anesthesia. At the point of respiratory failure myocardial depression may proceed to asystole and one is then confronted by two emergencies in such an event. Liver function is depressed more with chloroform than with any other volatile agent. Besides, hepatitis may occur postoperatively from a direct toxic effect on liver cells. Elevation of blood sugar, alteration in acid base balance, and dehydration occur. The prothrombin time is prolonged. Phosgene forms when vapors are exposed to flames or cautery. Postanesthetic nausea and vomiting are frequent. Diseases of the heart, hypertension, hypotension, liver and renal derangement and diseases of the respiratory tract are contraindications to the use of chloroform.

Fluothane. Fluothane is a two carbon compound having three fluorine atoms on one carbon and a bromine, chlorine and hydrogen atom on the other. The compound is thus a halogenated hydrocarbon. Fluothane is the most potent inhalation anesthetic known. Interest in the derivative was aroused by its potency and nonflammable and relatively nonirritating qualities. The odor resembles that of chloroform, though it is less pungent. Like related halogenated hydrocarbons, it manifests cardiotoxic and hepatotoxic qualities. Immediately after induction, hypotension develops which is due to a combination of several factors, among which are a decrease in cardiac output and varying degrees of ganglionic blocking ac-

tion. The myocardium is depressed and cardiac irritability is enhanced, particularly during deep anesthesia. Spontaneous arrhythmias are common when anesthesia is deep. Ventricular tachycardia and fibrillation occur when the drug is used simultaneously with epinephrine and closely allied amines. The drug is used largely in combination with nitrous oxide, since it is only in this manner that an unvarying concentration may be maintained. Induction is rapid (2 to 3 minutes). Recovery likewise, is rapid. With nitrous oxide, concentrations varying from 0.1 to 0.5 per cent are adequate for surgical anesthesia. Liver function is depressed but not to the extent it is with chloroform.

Trichlorethylene. Trichlorethylene (Trilene) is a colorless heavy liquid. It is an unsaturated halogenated hydrocarbon-ethylene with three hydrogen atoms replaced by chlorine. The odor resembles that of chloroform. Waxoline blue is added to avoid mistaking one for the other in localities where both drugs are used. Oxidation converts the drug to phosgene. The open or semiclosed techniques are used for its administration. The closed system cannot be used because soda lime converts the drug to dichloroacetylene which is neurotoxic. Mixtures of trichlorethylene and oxygen (10 per cent) are combustible but in the concentration used for surgical anesthesia are not flammable.

Alveolar tensions of 25 to 30 mm. Hg (4 per cent) are anesthetic. The tension necessary for respiratory failure has not been established. Respiratory failure precedes circulatory failure when overdosage occurs. Tensions of 2 to 8 mm. Hg (0.25 to 1.0 per cent) are analgesic. Most of the drug is exhaled unchanged, but some is transformed to trichloroacetic acid by the liver and excreted into the urine.

Induction is slow and resembles that of ether. Analgesic concentrations are not irritating but anesthetic concentrations cause excessive salivation and secretion of mucus. Trichlorethylene causes tachypnea by stimulating the vagus nerve endings. The respiratory rate may exceed 60 per minute at times. Trichlorethylene depresses the myocardium and increases cardiac irritability. It is used for analgesia but is not recommended for surgical anesthesia. Besides being toxic, it produces relaxation of skeletal muscle that is not adequate for major surgery. It is important to differentiate between analgesia and anesthesia. Inhalers are available for self-administration of the drug to provide

used when hepatic or renal disease exists. Excess secretions may help disseminate and thereby aggravate an acute upper respiratory infection.

Ether When doubt exists concerning selection of an anesthetic, ether in spite of alleged shortcomings, still remains the safe and reliable choice. Ethyl ether is a volatile, flammable liquid whose vapor possesses marked narcotic potency. The vapor is heavier than air pungent and induces coughing when inhaled in anesthetic concentrations without previous acclimatization. Ether is suitable for major surgery requiring muscular relaxation. It possesses a wide margin of safety. The alveolar tension necessary for surgical anesthesia is approximately 30 mm. Hg (4 per cent) for respiratory failure 60 mm. Hg (8 per cent). This wide margin of safety is one of the desirable features of ether.

Ether is administered by open masks using air as a vehicle, by the insufflation technique using air or oxygen as a vehicle by the semi-open inhaler with nitrous oxide or by means of closed inhalers with oxygen. The induction period is unusually long because of the pungent irritating effects and because of the marked capacity of the body for the drug. Ether compared to other volatile anesthetics, is water soluble and the body is capable of absorbing a considerable quantity. Besides, it has a high blood air coefficient (15:1) so that partition between blood and alveolar air is slow. The elimination requires many hours on this account. The time for complete elimination thus varies with the duration and depth of anesthesia. To simplify and shorten induction a non-irritating, rapid acting drug, referred to as the induction agent, is first used. The drug used for induction is nitrous oxide, ethylene, cyclopropane, vinyl ether, chloroform or ethyl chloride. As soon as anesthesia is induced with the preliminary agent, the anesthetic concentration of ether is delivered. This can be done more rapidly than would be possible if the ether were given directly. The first and second stages thereby are shortened.

Ether is relatively inexpensive, is chemically stable, easily preserved and is administered by simple apparatus when necessary. The assertion that the open drop technique is the best for ether is fallacious. In an emergency it is the safest technique to use by inexperienced individuals. The closed methods are far more satisfactory. Air may be used as a diluent and as the source of

oxygen because the tension required for an esthesia is low. At ordinary levels of an esthesia, ether exerts few deleterious effects upon the circulatory system. Respiratory movements are exaggerated owing to stimulation of the alveolar vagal nerve endings by the vapor. Nausea and vomiting are common in the postanesthetic period. Liver function, acid base balance and carbohydrate metabolism are temporarily disturbed. Acidosis from any cause is enhanced by ether anesthesia. Ether should be avoided in an acute respiratory infection. It is contraindicated in syndromes of the brain accompanied by increased intracranial pressure because it often causes a further increase in pressure.

Ethyl Chloride. Ethyl chloride is a highly volatile flammable liquid whose vapor is pleasant smelling, easily inhaled and produces surgical anesthesia. Chemically it is a stable halogenated hydrocarbon. Analgesia results when the alveolar tension is 15 to 20 mm. Hg (2 to 3 per cent) anesthesia at 30 to 35 mm. Hg. Induction and recovery are rapid, usually requiring two to three minutes. It is used as an induction agent to shorten the first and second stage of ether anesthesia administered by the open mask techniques and for anesthesia and analgesia in operations for minor surgical procedures of not more than several minutes' duration. Ethyl chloride ordinarily is administered by the open drop method. Air acts as both the vehicle and as a source of oxygen.

Ethyl chloride does not cause respiratory depression. Cardiac arrest may result from direct depression of the myocardium. Besides, it increases cardiac irritability and causes serious arrhythmias. Ventricular fibrillation may occur early in anesthesia. Asystole may occur before medullary paralysis. For this reason, the use of the drug is not advised for any procedure. Anesthesia at times is accompanied by stridor or muscle rigidity. The depth of anesthesia is difficult to maintain at a constant level because of the high volatility. Postanesthetic nausea and vomiting are uncommon.

Chloroform. Chloroform (trichloromethane) is a colorless volatile liquid whose vapor is sweet smelling, easily inhaled and nonflammable. All types of surgery requiring muscle relaxation may be performed with chloroform anesthesia. Chloroform is unsafe because it is cardiotoxic and hepatotoxic. It has been used as an induction agent for shortening stage II of ether anesthesia, as an analgesic for obstetrics in dilute concen-

analgesia for minor procedures. Trichlor ethylene is used to fortify nitrous oxide.

ENDOTRACHEAL ANESTHESIA

Endotracheal anesthesia consists of introducing a catheter into the trachea through the mouth or nose and conducting the gases and vapors directly from the apparatus to the lungs. Actually the catheter extends or prolongs the trachea to the lips or nares. During general anesthesia, the muscles of the neck, tongue, pharynx and jaw relax and the tongue sags backward. As they do so the epiglottis swings posteriorly and occludes the pathway into the trachea and respiratory obstruction results. A properly placed catheter provides an unimpeded airway. In addition, it permits the anesthetist to employ positive pressure and artificial respiration when indicated.

A catheter of smaller diameter may be passed into the tracheal catheter to aspirate secretions. The endotracheal catheter prevents laryngeal spasm by keeping the cords abducted. Certain surgical manipulations such as incising the peritoneum making traction upon the organs in the abdomen and thorax, dilating the rectum or stimulating the perineal structures excites reflexes which cause laryngeal spasm. The catheter prevents the spasm. A properly introduced endotracheal catheter prevents aspiration of vomitus. The catheter assures an airway for patients undergoing surgery in the prone or lateral positions as in the cases of operations upon the vertebral column, back and head or thorax. In surgery of the mouth, pharynx and larynx an endotracheal tube allows the anesthetist to be out of the operative field and still have control of the airway. Endotracheal anesthesia is necessary for obese individuals and others in whom an open airway is maintained with difficulty.

The catheter is introduced by direct laryngoscopy that is the larynx is visualized directly with a laryngoscope. When the catheter is introduced through the mouth, the procedure is called *oral endotracheal intubation*. In nasal intubations the catheter is introduced through the nostril which is most patent and free from obstruction. The curvature of the catheter and the anatomic position of the larynx favor passage into the trachea without laryngoscopy. This procedure is called *blind nasal intubation*. When the catheter cannot be passed blindly the larynx is exposed with the laryngoscope and the tube is introduced under direct vision with a forceps.

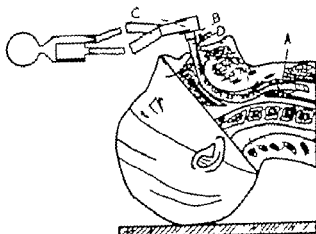


Figure 5 Closed oral endotracheal anesthesia is accomplished by connecting an intratracheal catheter (A) equipped with an inflatable cuff to an adaptor which replaces the mask of a circle filter. An entirely closed system is obtained. Anesthesia is conducted in the same manner as it would be if a mask were used. (From Adams, *Techniques and Procedures of Anesthesia*. Courtesy Charles C Thomas, Publisher.)

If in the oral technique, the mask is replaced over the mouth after intubation the technique is called *open oral endotracheal anesthesia*. If the catheter is introduced nasally and the mask is replaced over the nose and mouth, the technique is known as *open nasal endotracheal intubation*. The catheter may be connected directly to a closed or semiclosed inhaler. When this is done, the technique is called *closed endotracheal anesthesia* (Fig 5). Sometimes an inflatable cuff or a gauze pack is used to seal the space between the wall of the trachea and the catheter and provide a leak proof closed system. Catheters are made of rubber plastic or metal. Wire coils are often incorporated in the wall of rubber catheters to prevent kinking.

Endobronchial anesthesia consists of introducing a longer special catheter into the bronchi. Endobronchial catheterization is usually employed for partial or complete pneumonectomy for suppurative diseases of the lungs to prevent secretions from passing from the diseased to the healthy lung. Catheters used for differential bronchosprometry are also suitable in these circumstances. Each division passes into the right and left bronchus respectively. Anesthesia is conducted and aspiration accomplished in each lung separately.

For intubation, general anesthesia is induced in the usual manner with cyclopropane ether or other drug until the neck and jaw muscles relax. The larynx is then exposed and the catheter inserted. A muscle

relaxant facilitates the intubation. Unless performed by skilled operators considerable trauma may result to the pharynx, larynx and trachea which may be followed by serious consequences. The advantages of endotracheal anesthesia outweigh most disadvantages. Difficulties encountered, as a rule, are due to poor technique.

In certain surgical procedures apnea is deliberately induced and respiration is maintained artificially to provide a quiet operative field. Such operating conditions facilitate intrathoracic and upper abdominal operations. Ventilation referred to as controlled respiration, is maintained by rhythmic compression of the breathing bag of the anesthetic apparatus approximately twenty times per minute. Apnea is induced by using a drug which depresses the respiratory center usually a nonvolatile drug and cyclopropane. The overdistention of the alveoli to excite the Hering Breuer reflex to inhibit inspiration and hypocapnia caused by hyperventilation also contributes to the development of the apnea. Some anesthetists cause the apnea by curarizing the patient to the point of complete muscle paralysis. The effect of increased pressure on the airway for long periods may bring about a reduction in cardiac output and circulatory deterioration during controlled respiration no matter how it is induced.

With the exception of ether most anesthetic drugs cause hypoventilation. Ether causes exaggerated breathing and in increased minute volume exchange. Both oxygenation and carbon dioxide elimination are adequate. With other drugs oxygenation may be adequate, but carbon dioxide elimination is not. In order to prevent respiratory acidosis, the minute volume exchange must be augmented by compression of the breathing bag manually. This is referred to as assisted respiration. Automatic ventilating machines are available which either assist patients who, though making respiratory efforts, do not have an adequate minute volume exchange or breathe artificially for patients who are apneic. These devices are useful for specialized surgical procedures (intrathoracic) but are superfluous for routine use.

BASAL NARCOTICS

Ethyl Alcohol. Ethyl alcohol is not satisfactory for surgical anesthesia because it does not completely abolish the reflexes and it possesses a narrow margin of safety. The concentration necessary for anesthesia, in a

surgical sense approximates that which causes medullary depression. The systemic effects are the same regardless of the route of administration. Ethyl alcohol is administered intravenously (5 per cent with 5 per cent dextrose in distilled water) for analgesia postoperatively or for sedation preliminary to anesthesia for addicts or patients who cannot be given narcotics for other reasons.

Paraldehyde. Paraldehyde is a colorless mobile liquid possessing a pungent odor which appears to cling to surrounding objects for days. The drug is a polymer of acetaldehyde. It is used as a hypnotic for alcohol addicts and mentally disturbed patients or as a basal narcotic preliminary to surgery. Its use as an amnesic agent for obstetrics has also been described but is not advised. Paraldehyde possesses anticonvulsant properties. The drug is administered rectally or intravenously. Like other nonvolatile substances it lacks analgesic properties and the ability to abolish reflexes completely. It is characterized by a variability of action. The drug causes irritation of the mucosa of the mouth, stomach and rectum. Basal narcotic doses disturb metabolic processes. An elevation in blood sugar and lowering of carbon dioxide combining power are frequently observed. Some of the drug is exhaled through the lungs, but the bulk of it is detoxified by the liver. Paraldehyde is not used when pulmonary diseases or hepatic or renal insufficiency exists.

Tribromethanol (Avertin). Tribromethanol, a halogenated derivative of ethyl alcohol, is a white crystalline substance with an ethereal odor. The drug is poorly soluble in water but very soluble in amylene hydrate (tertiary amyl alcohol). The latter is used as a vehicle to prepare a solution used in clinical medicine known as Avertin fluid. One cubic centimeter of Avertin fluid contains 1000 mg. of the drug and 500 mg. of the solvent. A 3 per cent solution in water (27 to 36 mg. per pound of body weight) is administered rectally forty-five minutes before induction of anesthesia. The drug is ineffective orally; its action intravenously is variable. Following rectal administration, a deep stage of hypnosis accompanied by amnesia develops within five minutes. Reflexes are not completely abolished, however, and surgery cannot be performed without a supplemental anesthetic such as a nerve block, cyclopropane, nitrous oxide or ether. The drug is conjugated with glycuronic acid by the liver and the product is

excreted by the kidneys. Avertin is used to control convulsions and other hyperirritable states of the nervous system such as are encountered in tetanus rabies or drug reactions. Recovery from hypnosis is gradual over a period of several hours. Prolonged amnesia results. Avertin is administered at the bedside to allay apprehension preoperatively. It thus eliminates excitement during induction of inhalation anesthesia. Tribrom ethanol is nonirritating to the respiratory tract but does depress respiration. Postoperative nausea and vomiting are minimized and the amount of supplemental anesthetic necessary is reduced. It is uncontrollable however once a dose is administered it can not be retrieved. The exact dose is difficult to estimate owing to variations in susceptibility of the individual and absorption from the rectum. Hypotension due to depression of the vasomotor center is frequently observed. Repeated doses result in cumulative effects. Laryngeal and pharyngeal reflexes are partly obtunded and are reactivated by stimulation. Constant attendance of the patient is required from the moment the drug is administered until narcosis has receded. Proctitis and colitis may result from use of deteriorated solutions. The drug is not stable and deteriorates if solutions are heated or allowed to stand. Hepatic and renal diseases, "toxemia, sepsis, acidosis shock or other forms of hypotension, dehydration, low metabolic rate chronic pulmonary disease disease of the gastrointestinal tract, amnesia and chronic alcohol addiction are contraindications to the use of the drug.

Barbiturates. One of the most useful groups of drugs in medicine is the barbiturates. The barbiturates possess no significant analgesic properties and cannot, therefore be used alone for pain relief. Barbiturates are placed into four groups namely the long acting, the intermediate acting, the short acting and the ultra-short acting. The long and intermediate acting derivatives are used for sedation. The short acting and ultra-short acting are used in conjunction with surgical anesthesia as basal narcotics. Barbiturates are usually given intravenously when used for basal narcosis. The rectal route is used for children. The ultra-short acting derivatives are more potent than the short acting. All barbiturates manifest a latent period from the moment of injection until the peak effect is attained. The latent period is briefest and the peak effect most intense with the ultra-short acting drug. Thiopental (Pentothal) thiosecobarbital

(Surital) and hexobarbital (Evipal) are the most important currently used derivatives. A dilute solution of any of these injected intravenously causes unconsciousness within thirty five or forty seconds. These compounds pass quickly from the blood and are stored in the lipid and other tissues. Recovery occurs when the blood level falls. Fractional doses are administered at frequent intervals in order to maintain basal narcosis. In due time enough of the drug accumulates in the adipose tissue to maintain a sustained depressed state. Reflex activity is not completely abolished during barbiturate narcosis. The patient does not respond, however unless stimulated. Painful stimuli often cause the patient to become partly roused into a delirious unruly state. It is customary in present-day practice to combine barbiturates with an analgesic usually nitrous oxide. Neither the barbiturate nor the nitrous oxide yields satisfactory muscle relaxation. This is obtained by adding a muscle relaxant.

Barbiturates cause depression of the medullary centers. Hypotension due to depression of the vasomotor center is not uncommon after intravenous administration. A cumulative action invariably occurs when fractional doses are administered over a long time. The barbiturates are therefore, used at the beginning of anesthesia to facilitate induction or for short procedures. The laryngeal, pharyngeal and bronchial reflexes remain active. In fact, the thiobarbiturates enhance the activity of these reflexes and intense bronchial and laryngeal spasms are initiated by stimulation of the upper respiratory passages. Mucus, blood, or surgical manipulation in the respiratory tract may give rise to serious difficulties. The effective dose varies with the susceptibility of the individual. As a rule, 10 gm is the average dose for an adult. Recovery after short procedures is prompt. Detoxification occurs in the liver. The rate depends on the total amount injected and the metabolic state of the patient.

Aqueous solutions of salts of barbiturates are alkaline. Phlebitis and thrombosis may occur after intravenous administration. Induction of basal narcosis is rapid and pleasant with intravenous barbiturates. Secretions are absent in contradistinction to the volatile anesthetic drugs. The barbiturates are the only agents which may be used in areas where a fire hazard exists particularly when the anesthetic is to be combined with nitrous oxide. Nausea and vomiting are uncommon

following the use of barbiturates. The ultra short acting barbiturates are not desirable for patients at the extremes of age in hypotensive states (shock) or for those who have any of the anemias or any disease of the respiratory tract characterized by dyspnea, hypoventilation, excessive secretions due to suppurative, or reduction in pulmonary function.

NARCOTICS

The narcotics are nonvolatile drugs which possess both analgesic and hypnotic properties. Most narcotics induce a psychic response often termed, "a sense of well being" or "euphoria." Narcotics obtund dull aching types of pain more effectively than the sharp, lacerating types. Unless large doses are used, reflex activity is not abolished. The narcotics, therefore, are suitable only for relieving pain preoperatively or postoperatively or after trauma, but not for producing surgical anesthesia. The feeling of well being and the air of indifference induced by narcotics make them superior to other drugs for preanesthetic medication. Barbiturates and other hypnotics do not as a rule induce this response and are, therefore, used less frequently for psychic sedation preliminary to surgery.

Until recent years the only useful narcotics were opium derivatives. Synthetic narcotics are now available. Qualitatively, they are similar to opium derivatives but quantitatively they are different. Nalorphine (Demerol), methadone, methyl morphinan (Dromoran), amiloridine (Leritine) and alpha-prodine (Nisentil) are suitable analgesics but less effective as preanesthetic medicaments. The opium alkaloids are derived from phenanthrene. The most important of these, of course, is morphine. Modifications of the morphine molecule yield codeine and dihydromorphine (Dilaudid). Tolerance and habituation quickly result when narcotics are administered repeatedly for a time. After several weeks physical dependence (addiction) develops. In addition, the drug is required to maintain necessary cellular functions and withdrawal results in the abstinence syndrome, the symptoms of which suggest an organic disease.

MUSCLE RELAXANTS

Muscle relaxants are nonanesthetic substances used as adjuncts to anesthesia. They act by interfering with the transmission of impulses from peripheral nerves to striated muscle. They do not block the nerve or

depress the muscle fibers. They are nonhypnotic and nonanalgesic. Two types are recognized: those which prevent acetylcholine from acting at the receptor organ of the muscle fiber and those which cause a persistent depolarization of the membrane at the receptor substance.

Curare was at one time, the most widely used muscle relaxant. The active principle in curare is the alkaloid tubocurarine. Synthetic substances are now used, the most important of which are decamethonium and succinylcholine. Curare and tubocurarine inhibit acetylcholine-decamethonium and succinylcholine cause persistent depolarization. Succinylcholine is rapidly hydrolyzed by cholinesterase. Its action, therefore, is fleeting and sustained relaxation can only be obtained by continuous infusion.

When relaxation is desired, the drug is administered intravenously. Complete muscle paralysis may be secured during which respiration must be sustained artificially. By controlling dosage, paresis of large muscles without apnea results. The action of curare lasts fifteen to twenty minutes after which time the dose must be repeated. The combination of nitrous oxide, a muscle relaxant and basal narcosis, using an ultra short acting barbiturate (thiopental) is widely used.

The most widely used and the safest of the muscle relaxants is succinylcholine. Its transient and fleeting action and ready destruction in the body make it controllable. Difficulties do arise, however, after prolonged use or after the use of excessive quantities. The most vexing of these are the prolonged apneas lasting many hours. These are seen when the drug is administered by the continuous infusion technique and given to the point of complete paralysis which necessitates controlled respiration. Difficulties are uncommon when the drug is used in intermittent fractional doses.

LOCAL AND REGIONAL ANESTHESIA

Regional or conduction, anesthesia is employed when one wishes to obviate the risks, disadvantages and discomforts of general anesthesia. Regional anesthesia is subdivided into (1) spinal block in which the drug is introduced in the subarachnoid space and exerts its effect on the spinal nerve roots, (2) peridural block in which the drug is applied in the peridural space, (3) nerve block in which the drug is applied directly to a nerve, (4) field block in which a group of nerves is blocked as each branches from a main trunk, (5) infiltration, in which the

excreted by the kidneys. Avertin is used to control convulsions and other hyperirritable states of the nervous system such as are encountered in tetanus, rabies or drug reactions. Recovery from hypnosis is gradual over a period of several hours. Prolonged amnesia results. Avertin is administered at the bedside to allay apprehension preoperatively. It thus eliminates excitement during induction of inhalation anesthesia. Tribrom ethanol is nonirritating to the respiratory tract but does depress respiration. Postoperative nausea and vomiting are minimized and the amount of supplemental anesthetic necessary is reduced. It is noncontrollable, however, once a dose is administered it can not be retrieved. The exact dose is difficult to estimate owing to variations in susceptibility of the individual and absorption from the rectum. Hypotension due to depression of the vasomotor center is frequently observed. Repeated doses result in cumulative effects. Laryngeal and pharyngeal reflexes are partly obtunded and are reactivated by stimulation. Constant attendance of the patient is required from the moment the drug is administered until narcosis has receded. Proctitis and colitis may result from use of deteriorated solutions. The drug is not stable and deteriorates if solutions are heated or allowed to stand. Hepatic and renal diseases, "toxemia," sepsis, acidosis, shock or other forms of hypotension, dehydration, low metabolic rate, chronic pulmonary disease, disease of the gastrointestinal tract, amnesia and chronic alcohol addiction are contraindications to the use of the drug.

Barbiturates. One of the most useful groups of drugs in medicine is the barbiturates. The barbiturates possess no significant analgesic properties and cannot, therefore, be used alone for pain relief. Barbiturates are placed into four groups, namely, the long acting, the intermediate acting, the short acting and the ultra-short acting. The long and intermediate acting derivatives are used for sedation. The short acting and ultra-short acting are used in conjunction with surgical anesthesia as basal narcotics. Barbiturates are usually given intravenously when used for basal narcosis. The rectal route is used for children. The ultra-short acting derivatives are more potent than the short acting. All barbiturates manifest a latent period from the moment of injection until the peak effect is attained. The latent period is briefest and the peak effect most intense with the ultra-short acting drug, Thiopental (Pentothal). thiobarbital

(Surital) and hexobarbital (Evipal) are the most important currently used derivatives. A dilute solution of any of these injected intravenously causes unconsciousness within thirty-five or forty seconds. These compounds pass quickly from the blood and are stored in the lipoid and other tissues. Recovery occurs when the blood level falls. Fractional doses are administered at frequent intervals in order to maintain basal narcosis. In due time enough of the drug accumulates in the adipose tissue to maintain a sustained depressed state. Reflex activity is not completely abolished during barbiturate narcosis. The patient does not respond, however, unless stimulated. Painful stimuli often cause the patient to become partly roused into a delirious, unruly state. It is customary in present-day practice to combine barbiturates with an analgesic, usually nitrous oxide. Neither the barbiturate nor the nitrous oxide yields satisfactory muscle relaxation. This is obtained by adding a muscle relaxant.

Barbiturates cause depression of the medullary centers. Hypotension, due to depression of the vasomotor center is not uncommon after intravenous administration. A cumulative action invariably occurs when fractional doses are administered over a long time. The barbiturates are therefore, used at the beginning of anesthesia to facilitate induction or for short procedures. The laryngeal, pharyngeal and bronchial reflexes remain active. In fact, the thiobarbiturates enhance the activity of these reflexes and intense bronchial and laryngeal spasms are initiated by stimulation of the upper respiratory passages. Mucus, blood, or surgical manipulation in the respiratory tract may give rise to serious difficulties. The effective dose varies with the susceptibility of the individual. As a rule 1.0 gm. is the average dose for an adult. Recovery after short procedures is prompt. Detoxification occurs in the liver. The rate depends on the total amount injected and the metabolic state of the patient.

Aqueous solutions of salts of barbiturates are alkaline. Phlebitis and thrombosis may occur after intravenous administration. Induction of basal narcosis is rapid and pleasant with intravenous barbiturates. Secretions are absent in contradistinction to the volatile anesthetic drugs. The barbiturates are the only agents which may be used in areas where a fire hazard exists, particularly when the anesthetic is to be combined with nitrous oxide. Nausea and vomiting are uncommon

used to anesthetize mucous membranes. The duration of action of procaine in a nerve block averages forty five minutes. Various modifications of procaine are available. One of these *chloroprocaine* (*Nesacaine*) is equally as potent but less toxic than procaine. It is used for the same purposes and in the same general manner as procaine.

Tetracaine (*Pontocaine*) is more potent and more toxic than procaine. It is second in importance to procaine for surgical anesthesia. One milligram of tetracaine is equivalent to 10 mg. of procaine as regards its anesthetic potency. The duration of action of tetracaine is almost twice that of procaine. It is possible if epinephrine is added to solutions of tetracaine to obtain a block lasting from two to four hours. Large nerve trunks are blocked with 0.15 per cent solutions, smaller trunks with 0.10 per cent, and nerve endings (infiltration) by 0.05 per cent to 0.075 per cent solutions. Tetracaine is used extensively for spinal anesthesia.

Symptoms of toxicity following the use of tetracaine are more frequent and serious than those of procaine. Reactions are characterized by syncope and circulatory collapse. As a rule, few if any prodromal symptoms precede or forewarn of impending catastrophe. The use of tetracaine for other than spinal block should be reserved for those experienced in the technique of regional anesthesia.

Piperocaine (*Netycaine*) is closely allied to cocaine chemically being an ester of benzoic acid. It is similar to procaine in most respects. However it is somewhat more potent and longer lasting. One and one-half per cent solutions are suitable for blocking large and medium sized nerve trunks. A one per cent solution should be used for smaller nerve trunks and for infiltration.

Lidocaine is a newer anesthetic drug. It is a xylidide (not an ester) but it does possess the general chemical configuration common to other local anesthetics. It has a very brief latent period and produces almost immediate anesthesia and, when infiltrated, it produces an effect lasting one hour without epinephrine and two to three hours with epinephrine. The volumes and strength of solutions recommended are approximately one-half to two-thirds of those for procaine in similar situations. Lidocaine possesses striking ability to diffuse through tissues. Thus, if perchance the needle is introduced some distance from the nerve an effective block is still obtained because the drug diffuses over a wide area and reaches the

nerve. The incidence of failures is reduced noticeably in comparison to procaine when one adopts the drug. Blocks in which motor effects are partial or absent when procaine is used yield complete flaccidity with lidocaine.

Dibucaine (*Nupercaine*) is approximately fifteen times more potent and toxic than is procaine. Its effect is longer lasting than that of any of the currently used drugs. Nerve blocks may be performed with a 0.075 per cent solution and infiltration with a 0.05 per cent. Sloughs and severe systemic manifestations of toxicity have been reported. The drug is not recommended for infiltration and nerve blocking but is suitable for spinal anesthesia.

Commercially prepared, ready mixed and sterilized solutions of local anesthetic drugs are available in sealed glass ampules or multiple-dose vials. Such preparations are preferred, from the standpoint of safety, stability, asepsis and ease of handling, by the operator to those made up from tablets and powders. Accidents due to the use of concentrated solutions and the possibility of contamination are thus avoided.

When these preparations are not available, the powder may be dissolved in the appropriate solvent and sterilized by autoclaving or boiling. Epinephrine is added at the time the solution is used because it is not stable and cannot be boiled. Solutions as nearly isotonic as possible are used in order to prevent swelling and edema of the tissues during operation and local irritation afterwards. Potent drugs such as tetracaine or dibucaine are dissolved directly in physiologic saline, since the amount necessary for effectiveness is, comparatively speaking, minute and toxicity is not altered. Two per cent procaine must be prepared with 0.45 per cent sodium chloride to be isotonic.

The most efficient vasoconstrictor for local anesthesia is epinephrine. It is never used for patients with cardiovascular disease, peripheral vascular disease, hyperthyroidism or those who have had a sympathectomy. Injections of solutions containing epinephrine into the fingers and toes, particularly when peripheral vascular disease is present, may cause gangrene and slough.

REACTIONS TO LOCAL ANESTHETIC DRUGS

When local anesthetic drug gains access to the blood stream, either by injection or by absorption, a train of symptoms commonly referred to as a "reaction" results. Two types of reactions are recognized the

tissues at the surgical site are infiltrated so that each individual nerve ending is blocked by disseminating a solution over a wide area and (6) topical, in which the drug is applied on a mucous membrane through which it penetrates and anesthetizes the nerve ending beneath it.

Local Anesthetic Drugs. Numerous drugs are available for conduction anesthesia. In order to be of practical value for blocking or conduction anesthesia, a drug must possess the following qualities: (1) the ability to produce an adequate intensity of anesthesia within several minutes, (2) the capacity to give an effect lasting long enough to permit successful completion of the operation, (3) the inability to cause local irritation immediately after injection or in the postoperative period, (4) the inability to produce local tissue damage or to retard healing, (5) low systemic toxicity and (6) compatibility with vasoconstrictors such as epinephrine and similar drugs. The vasoconstrictors are added to retard absorption, which in turn decreases systemic toxicity and prolongs the effect.

Local anesthetics are basic organic substances which form salts with mineral and organic acids. The majority of injectable drugs are esters. The salts are soluble in water and insoluble in organic solvents. The bases are soluble in organic solvents, oils and vehicles used for ointments but poorly soluble in water. Alkalis precipitate the basic form from aqueous solutions of salts.

Detoxification of local anesthetics occurs in the liver. The rate varies with the chemical nature of the drug. Some are completely hydrolyzed and the by products are excreted into the urine. Procaine for example is converted to para aminobenzoic acid and diethylaminoethanol. Others are partly detoxified and partly eliminated unchanged in the urine (cocaine). Still others are almost entirely eliminated unchanged. Greater toxicity is usually associated with slowly eliminated or detoxified drugs.

The effective concentration of a local anesthetic depends upon the size of the nerve fiber. Autonomic and sensory fibers of a mixed nerve are smaller than the motor fibers. If a nerve containing all three components is exposed to a dilute solution of a local anesthetic, a blockade of the sensory and autonomic fibers develops first. Conduction remains in motor components for a variable period. Later the motor components are affected. This apparent selectivity depends upon the ease of penetration of the

drug into the fibers. Drugs penetrate non-myelinated fibers more easily than myelinated. Penetration into the large nerve trunks, since they have a denser perineural sheath than do the small, occurs at a slower rate. More concentrated solutions are necessary for successful blockade of larger centrally located nerve trunks than of the smaller peripherally located branches. The nerve endings in the skin and subcutaneous areas are anesthetized with still more dilute solutions.

When nerve trunks are large, fifteen to twenty minutes may elapse before a satisfactory block is established. This lapse in time, from the moment of injection until the blockade is established, is the sum of the time required for the drug to diffuse through the soft tissues, the time to penetrate and diffuse through the neural sheaths and the time required to diffuse through the perineural membrane and into the nerve fiber. The ease of penetration of a drug through the membrane of a nerve fiber is a physical factor which varies according to the configuration of the molecule of the drug. This latent period, during which nothing seems to be happening, is greater for the longer lasting drugs. Presumably the longer lasting drug diffuses slowly through the membrane. The outward diffusion, when the concentration of drug in the medium surrounding the nerve fiber is reduced below the equilibrium level, also is slow. These are the reasons for the differences in onset and duration of action of various drugs.

Besides the chemical nature of a drug, duration of action is influenced also by the site of injection, blood supply in the area injected, concentration and ability to detoxify the drug. In the scalp where the blood supply is excellent, duration is almost half that of the back where it is less abundant.

Procaine is the best known and the safest of the local anesthetics. It is almost universally the agent of choice for local and regional anesthesia. It is rapidly detoxified and eliminated. Reactions are mild when they occur and local damage to tissues is uncommon. The conventional concentrations and volumes for average adults are as follows:

2 per cent—not more than 50 cc. in one hour (1 gm.)
1 per cent—not more than 100 cc. in one hour (1 gm.)
0.5 per cent—not more than 200 cc. in one hour

As a general rule not more than 1 gm. should be used at any one sitting. Procaine possesses no topical action and cannot be

varying the dosage does not. The extent or level is controlled by dosage, specific gravity of solutions, positioning and rate of injection. The height or level of anesthesia is controlled by using solutions which are heavier or lighter than spinal fluid. Solutions heavier than spinal fluid are referred to as *hyperbaric*; lighter solutions as *hypobaric*; those of the same specific gravity as *isobaric*.

The effects of gravity are utilized in directing the drug to the desired segments. In the head-down supine position a hyperbaric solution migrates caudad, a hyperbaric one gravitates cephalad. In the head up position the reverse is true. Blocks induced with procaine, intracaine and piperocaine last approximately one hour; with tetracaine and hexylcaine two hours and with dibucaine three hours. Epinephrine added to the solution increases the duration approximately 60 per cent. Long lasting anesthesia is obtained by using dibucaine or tetracaine combined with epinephrine by the single injection method or by the continuous spinal technique. In the latter technique, a catheter of small bore is introduced intrathecally and the drug is injected at intervals as often as necessary.

Spinal anesthesia provides muscle relaxation superior to other methods of anesthesia. Surgeons prefer spinal anesthesia for the relaxation it affords.

Certain physiologic disturbances occur during spinal anesthesia of far reaching importance. In high spinal anesthesia, a blockade of sensory and motor fibers occurs in the sacral, lumbar and lower thoracic segments. However the autonomic fibers are not all blocked. The sympathetic and parasympathetic fibers in the lower spinal segments are inactivated. In the thoracic segments, the majority of the sympathetic fibers are blocked, but the parasympathetic fibers, since they arise in the cranial nerves, remain intact. This partial denervation of the autonomic nervous system in the upper part of the body causes hypotension, bradycardia, nausea, vomiting, contraction of the bowel and other changes.

Some of the advantages of spinal anesthesia are that it provides excellent muscle relaxation. The reflex arc is interrupted and muscles are completely paralyzed. The block is accompanied by little or no disturbances of metabolic processes if there is no hypotension. Loss of consciousness, excessive secretions, excitement, postanesthetic nausea, somnolence and other disagreeable features of general anesthesia are avoided. Caution

or electrical equipment may be used without fear of explosions. The operator may administer the anesthetic himself.

One disadvantage of spinal anesthesia is that it is noncontrollable. Once anesthesia has been instituted, it cannot be terminated. Its duration although predictable, is usually uncertain. The operation may outlast anesthesia and supplementary general anesthesia is required which subjects the patient to the hazards of two anesthetics. Failures, due to technical errors cannot be wholly excluded even in most skilled hands. The motor paralysis at high levels causes respiratory depression from the resulting intercostal and diaphragmatic paresis. Hypotension is common. Paralysis of the muscles and the autonomic denervation contribute to the peripheral circulatory failure. Vasopressor drugs are usually effective in overcoming circulatory failure in healthy subjects but those with a diseased vascular system do not always respond. Postoperative neurologic complications though infrequent, are a possibility. The effects of the drug on the nervous tissue, trauma from the needle infections and the use of contaminated or deteriorated solutions may be causative factors. One objection which is often offered is that the patient remains conscious throughout the operation. All patients are not cooperative and, therefore, are not psychically suited for spinal anesthesia. The vagal pathways from the viscera are not blocked during the time of operation, causing impulses to pass to the medulla. This may initiate vomiting, retching, nausea and vomiting, particularly when traction is made on the viscera. Impulses pass along the sympathetic chain in a retrograde manner and thence into the cord above the level of the block. The patient experiences pain in the thorax when traction is made upon the viscera.

Because circulatory depression is a prominent and common disturbance in spinal anesthesia, this technique is usually contraindicated when cardiovascular diseases are present. Severe hypertension, disturbances in cardiac rhythm, myocardial disease and cardiac failure are contraindications to spinal anesthesia. Hypotension and hypovolemia from any cause are contraindications to spinal anesthesia for the obvious reason that the vascular bed is increased in size and circulatory depression results. Neurologic diseases, whether degenerative or suppurative, are contraindications to this method of anesthesia. Although no evidence exists that these diseases are aggravated by spinal anes-

depressant or cardiovascular type and the stimulating or convulsive type.

The *depressant* type of reaction is due to circulatory collapse, which is the result of depression of the myocardium, dilatation of the vascular bed or a combination of both. The onset is usually abrupt, sometimes after the use of a minute amount of the drug. Sudden pallor appears followed by syncope and respiratory failure and death. In mild reactions the onset is slower and circulatory failure supervenes gradually. The patient becomes drowsy and passes into a coma-like state. Manifestations such as these have been ascribed to idiosyncrasy or sensitivity. It is generally conceded that they are due to overdosage when precautions outlined for using local anesthetics have not been observed. Artificial respiration must be instituted if respiratory failure has occurred. If asystole is suspected, both cardiac massage and artificial respiration must be instituted *simultaneously* without delay.

The *central nervous stimulation* type of reaction is ushered in by apprehension, excitement, disorientation and is then followed by convulsions and other manifestations of intense central nervous stimulation. The circulatory type of reaction occurs less frequently than does the central nervous system type. Pallor, yawning, nausea and vomiting may precede the convulsions. The prodromal signs of the nervous system type are often dismissed as hysteria. The severity and duration of the stimulation depend upon the pharmacologic nature and rapidity of absorption of the drug. The convulsive manifestations may be fleeting and be followed by a paralytic phase if large amounts of the drug are absorbed or injected. The subject is then comatose, completely depressed and in a state of circulatory collapse.

The convulsions are controlled by the intravenous administration of an ultra-short acting barbiturate (thiopental or hexobarbital). If neither of these is available, one of the short acting barbiturates such as secobarbital or pentobarbital may be used. The barbiturate is given intravenously in a quantity sufficient to control the convulsions. The ultra-short acting barbiturates are more suitable for this purpose because they act quickly and are more potent. Barbiturates merely antagonize the stimulating action. They do not hasten detoxification or elimination of local anesthetics, they do not overcome the depressant effect on the circulatory system and they do not antagonize the

paralytic phase of a reaction. They are suitable only to control convulsions.

Fatalities from local anesthetics are most frequent after topical application. The drugs are rapidly absorbed from the mucous membranes of the pharynx, trachea, bronchi and urethra. Blood levels after topical application to the mucous membranes are comparable to those obtained after intravenous administration.

Regional anesthesia should not be attempted without having available an ultra-short acting barbiturate, a vasopressor drug, a syringe and needle and some effective method of administering artificial respiration. The pallor, tachycardia, tremor and excitement caused by epinephrine used with the local anesthetic drug may easily be confused with the prodromal phase of a reaction. However, disorientation, convulsions and coma are uncommon symptoms.

The use of the intradermal wheal to determine sensitivity to local anesthetic drugs is a traditional procedure of doubtful value. Patients presenting a history of developing loss of consciousness or coma, following the injection of small quantities of local anesthetic drugs, should be regarded with suspicion and studied further before a block is attempted.

SPINAL ANESTHESIA

In spinal anesthesia the anterior and posterior roots of the spinal nerves are blocked as they arise from the spinal cord. The neurons and tracts on the surface may be involved, but the descending and ascending tracts deep in the cord substance are not blocked. The extent of the block, referred to as height or level, depends upon the number of spinal roots bathed by the solution. The perineal or saddle area is anesthetized when the sacral segments only are involved. The block is often called *saddle block*. The lower extremities are anesthetized when both the sacral and lower lumbar segments are involved. The block is referred to as *low spinal block*. When the sacral, lumbar and lower thoracic segments are blocked, the block is referred to as *medium*, or simply a *spinal block*. If the drug is forced into the upper thoracic area, the block is called a *high spinal block*.

The spinal cord ends at the level of the second lumbar vertebra. Lumbar puncture is always performed below this site to avoid trauma to the cord. The drug used determines the duration of spinal anesthesia,

injection exists because the epidural space is lined with a plexus of veins.

INFILTRATION ANESTHESIA

As a rule smaller surgical procedures may be performed after infiltration of the operative site with the desired local anesthetic drug. Procaine (1 per cent) is the most widely used agent for this purpose.

NERVE AND FIELD BLOCKS

Direct nerve blocking is adopted when the operative site is in an area supplied by easily accessible nerves. In some areas as for example the upper thorax, overlapping of nerves occurs so that blocking would have to be extensive. The anesthetic procedure might be of a greater magnitude than the operation itself. Nerve blocks thus are more practical for operations in the extremities or about the head than for those on the thorax and trunk. A field block may be considered as intermediate between a nerve block and infiltration. The anesthetic is deposited at the point of division of a nerve into branches so that the area distal to this point is anesthetized. Infiltration along the costal margin from the xiphoid to tenth rib blocks the branches of the lower intercostals to produce an abdominal field block. Anesthesia of the lower abdominal area is obtained by extending the line of infiltration along the outer border of the rectus muscle.

Brachial plexus block is widely used for anesthesia of the arm and forearm. The trunk divisions or cords of the plexus are infiltrated with a local anesthetic solution above the clavicle or in the axilla. In the supraclavicular approach, the nerves are blocked by depositing the solution along the first rib. Pneumothorax and trauma to the great vessels are possibilities. In the axillary route the injection is made in the medial aspect at the midline at the level of insertion of the pectoralis major. The median nerve is also blocked at the elbow or the wrist. When block is attempted at the elbow the landmarks are the brachial artery and the tendon of the biceps at the antecubital fossa. The radial nerve is also blocked at the elbow and wrist. When block at the elbow is attempted, an intradermal wheal is raised lateral to the biceps tendon and the needle introduced perpendicular to the skin and advanced until the lateral condyle of the humerus is encountered. The ulnar nerve is blocked at the elbow or wrist also. The landmarks for block at the elbow are the groove of the internal condyle of

the humerus and the olecranon process. By combining a block of these three nerves at the elbow anesthesia of the forearm and hand may be obtained. At the wrist, the needle is introduced between the tendons of the palmaris longus and flexor carpi radialis muscles at the level of the ulnar styloid to block the median nerve. The ulnar nerve likewise may be blocked in the same area by introducing a needle on the radial side of the tendon of flexor carpi ulnaris muscle at the level of the ulnar styloid. The radial nerve may be anesthetized at the wrist in the anatomic snuffbox.

The digital nerves are blocked on the lateral and medial aspects of the digits at the proximal phalanges. Gangrene may result from ischemia due to the injection of large volumes of solution. Brachial plexus block or blocks at the elbow or wrist are preferred.

The intercostal nerves are blocked as they course the intercostal spaces at a point in the midaxillary line on the inferior border of the rib. The femoral nerve is blocked below Poupart's ligament lateral to the femoral artery. Used alone femoral nerve block is satisfactory for operations on the anteromedial aspect of the leg. Combined with sciatic nerve block, it is satisfactory for operations below the knee. The sciatic nerve is blocked 3 cm inferior to the midpoint of a line drawn between the iliotrochanteric crest and the posterosuperior iliac spine. The anterior and posterior tibial nerves are blocked at the ankle for operations on the foot. The spinal nerves are blocked as they emerge from the intravertebral foramina along the bodies of the vertebrae by the technique known as paravertebral block. In the cervical area, block of the spinal nerves is referred to as cervical plexus block, in the thoracic area, as thoracic paravertebral block and in the lumbar area, as lumbar paravertebral block. Transsacral block, also a paravertebral block, is performed by passing needles into the sacral foramina through the dorsal aspect of the sacrum.

REFRIGERATION ANESTHESIA

At a temperature of 4° C. nerve conduction is retarded. An extremity which is devitalized and requires removal may be packed in ice for several hours and quickly amputated while still cold. The anesthesia rarely lasts more than thirty minutes. A tourniquet may be applied and the limb packed with ice for many hours until conditions for operation are more favorable in

thetia, one must remember that patients, at a later date, may ascribe symptoms of the disease to the spinal anesthetic and create a medicolegal problem. Spinal anesthesia should not be used when diseases of the respiratory system accompanied by severe pulmonary insufficiency are present. Inter costal paralysis accompanies high spinal anesthesia, decreases tidal exchange and causes a reduction in vital capacity. Anemia is a contraindication to spinal anesthesia because the oxygen-carrying power of the blood is reduced and tissue anoxia may occur. Diseases characterized by increased intra abdominal pressure due to distention from gas ascites or large tumors are contraindications to spinal anesthesia because a severe irreversible fall in blood pressure may follow induction of the block in these subjects. Septicemia also is a contraindication because the organisms may be carried into the spinal canal by the needle. Spinal anesthesia should not be used for operations above the diaphragm. Infections about the vertebral column at the site of lumbar puncture are contraindications for the same reason. Psychically disturbed subjects are not good candidates for spinal anesthesia. Patients of advanced age usually develop circulatory disturbances more frequently than younger subjects because the vasomotor compensatory mechanisms are not as effective. Children are not good subjects for spinal anesthesia because they are psychically unsuited. Distortions and bony changes in the vertebral column often preclude the use of spinal anesthesia because lumbar puncture is difficult to perform.

Neurologic complications may follow spinal anesthesia postoperatively. The most vexing and annoying of these is postlumbar puncture headache which is believed to be due to the leakage of spinal fluid from the perforation made by the needle in the dura. It is transient and leaves no sequelae fortunately. Of more serious consequence are the neurologic symptoms which appear after spinal anesthesia. The least common and most serious of these is paraplegia, the exact cause of which is not known. Myelitis and arachnoiditis have been found in subsequent examinations after this syndrome has appeared. Pre-existing neurologic diseases and errors in technique are believed to play a role. Palsies of the cranial nerves, particularly the sixth, also appear after spinal anesthesia on rare occasions. These are related to the loss of cerebrospinal fluid and are associated with headache. Infection from

contamination results in meningitis, arachnoiditis or abscess. Backache follows lumbar puncture at times. This may be due to trauma to the periosteum, the ligaments, the intervertebral disk or to the aggravation of pre-existing skeletal muscular disorders.

EPIDURAL ANESTHESIA

In epidural or peridural, anesthesia the spinal nerves are blocked as they pass through the epidural space. A needle is introduced at the lumbar area in the same manner as it is for lumbar puncture as far as the dura. Neither the dura nor the arachnoid is pierced. A modified form of epidural anesthesia is obtained by introducing a needle through the sacrococcygeal membrane into the caudal canal and injecting a large volume of a local anesthetic solution at this site. This is referred to as caudal or sacral anesthesia.

Epidural block is more difficult, hazardous and cumbersome to induce than caudal block or spinal block, because the needle may inadvertently be introduced into the subarachnoid space and a lethal amount of solution deposited there. The spinal nerves acquire a sheath as they pass through the epidural space. More concentrated and larger volumes of solution are necessary for penetration than are used for spinal anesthesia. Epidural anesthesia is used for the same purposes as spinal anesthesia. The indications for lumbar peridural block are few. The caudal technique, which is safer and is more easily performed, is used for rectal and other perineal operations. The possibility of headache, meningitis, encephalitis or other neurologic complications is less compared to spinal anesthesia. The drug migrates from the epidural space through the intervertebral foramen along the spinal nerves. Some evidence exists that it diffuses into the subarachnoid space. The drug does not contact the naked nerve roots or the cord, as in spinal anesthesia.

The disadvantages of peridural block are that the needle may be placed in the subarachnoid space instead of the epidural and an overdose of local anesthetic drug deposited there. Muscle relaxation is not always adequate. The level of analgesia is unpredictable and difficult to control. Drug reactions may occur because large amounts are needed for effective blocking. The drug does not always easily penetrate each of the spinal nerves and anesthesia is incomplete or segmental. The possibility of intravascular

death. Aspiration of vomitus and other material accounts for many cases of obstruction. The patient with a full stomach is a poor risk from an anesthetic standpoint. Irrespective of the type of anesthesia administered, whether it be local, intravenous, inhalation or spinal, the patient who has recently partaken of food or drink vomits during or after operation. Solid particles from the stomach are drawn into the trachea and bronchi and cause obstruction. The supine position, irrespective of the activity of the cough reflex, favors aspiration if vomiting occurs. Operations should be postponed if the patient has recently partaken of food or drink. Gastric lavage and emetics are of little value in completely emptying the stomach. Excess secretion of mucus results when premedication with an anticholinergic drug is inadequate. Obstruction may occur if the anesthetist is unable to maintain a proper airway because the patient is in the lateral or prone position or because the tissues in the supralaryngeal passages are relaxed or excessive. Endotracheal anesthesia obviates these difficulties. Intolerance to nonvolatile drugs such as barbiturates causes some fatalities. The use of a combination of a nonvolatile drug, a relaxing agent and an inhalation anesthetic multiplies the hazard of each drug.

Most fatalities occur during induction or at the conclusion of anesthesia, less frequently during maintenance. Nonchalance on the part of the anesthetist and carelessness or thoughtlessness may be underlying factors. In patients whose airways become easily obstructed, obstruction takes place early and the patient is asphyxiated at this time. Vomiting occurs during light anesthesia. This happens most often during induction or at the conclusion of the procedure. Irrespective of the drug and method of administration, as long as there is loss of consciousness, danger from asphyxia exists.

Deaths from spinal anesthesia are caused by respiratory paralysis or circulatory failure. Respiratory paralysis results if the drug is forced or diffuses into the upper thoracic and cervical portion of the spinal canal and causes paralysis of the intercostal muscles in the diaphragm. If immediately recognized and artificial respiration is commenced promptly the complication is not serious. Peripheral circulatory collapse during spinal anesthesia is due to failure of neurogenic control of the vascular system. It is readily overcome by vasopressor drugs (ephedrine, desoxyephedrine or Neo-Synephrine). When

it occurs suddenly as it does in poor risk subjects, and cannot be promptly corrected or if it is disregarded, cardiac arrest results. Pre-existing cardiovascular disease increases the hazard of spinal anesthesia.

PREPARATION OF PATIENT FOR ANESTHESIA

The proper preparation of the patient for anesthesia is important. The patient is hospitalized at the latest, the evening prior to operation. Preoperative examination consists of taking a history and performing a general physical examination with special attention to the cardiovascular and respiratory systems. A urine examination rules out or confirms the presence of diabetes or renal disease. Hemoglobin determinations are important because the oxygen-carrying power of the blood is of especial interest to the anesthetist. Patients with anemia do not tolerate even mild disturbances in ventilation. A barbiturate of the short acting type or other hypnotic drug is administered at bed time to assure adequate rest and allay apprehension. Food is withheld after the evening meal. Proper preoperative medication is important to allay apprehension and to obtain psychic sedation. A patient who is extremely apprehensive is difficult to anesthetize with an inhalation anesthetic. When nitrous oxide is used, the task is almost impossible.

Premedication is also necessary to obtain an additive effect in order to fortify a drug of low potency. Anticholinergic drugs are necessary to minimize secretions. Premedication may also be administered prophylactically to overcome undesirable side effects caused by anesthetic drugs, such as vagal stimulation, hypotension and cardiac arrhythmias.

The narcotics are the most suitable drugs for psychic sedation. They are superior to the barbiturates. Morphine is still the most suitable of the narcotics. Dilaudid, methadone, or meperidine is used for patients who cannot tolerate opium derivatives. Methadone and meperidine are satisfactory as analgesics but possess less hypnotic activity than does morphine and are, therefore, not as effective.

There has been an increasing tendency to combine ataractics with narcotics and barbiturates to enhance the sedative and anesthetic effects. The most effective drugs for this purpose are the phenothiazine derivatives such as chlorpromazine (Thorazine) and promazine (Sparine). These drugs are far from innocuous however because they cause a ganglionic blockade and have a

serious cases. This is tantamount to a physiologic amputation.

INTENTIONAL HYPOTENSION DURING OPERATION

A sympathectomized subject withstands blood pressures at shock levels induced by hemorrhage for longer periods than does one whose vasomotor control is intact. This principle is applied to minimize blood loss by reducing oozing in surgical procedures in which hemorrhage is anticipated. The denervation is accomplished either by using (1) a high spinal block, (2) a ganglionic blocking drug, (3) a sympatholytic drug or by (4) performing arteriotomy. When arteriotomy is performed, the blood is collected in a sterile receptacle containing an anticoagulant and retransfused. The vasomotor control remains and the advantages of sympathectomy are not obtained. The practical methods employ ganglionic blocking drugs such as hexamethonium or Arfonad. Thrombosis of the cerebral, coronary mesenteric and other vessels may occur. Cerebral damage from local tissue anoxia, anuria and reactionary hemorrhage are other sequelae and complications. The method is reserved for exceptional situations and is one which is not used routinely.

INTENTIONAL HYPOTHERMIA DURING OPERATION

Cooling the tissues reduces metabolic activity of cells. When a central nervous system depressant is administered, the heat regulating center is depressed and the body temperature tends to approach that of the external environment. Certain operations such as cardiac operations designed to relieve cyanosis due to congenital defects or vascular operations in which the circulation to an organ is interrupted for a period, are facilitated and made possible by the reduction in metabolic activity of the cells. The period of ischemia can be increased without causing undue harm. The patient is anesthetized with a volatile or nonvolatile drug and immersed in ice water or wrapped in special blankets through which ice water circulates. The body temperature is reduced to a temperature between 28 and 24° C. The procedure is not without hazard. Ventricular fibrillation readily occurs at low temperatures.

OPERATING ROOM DEATHS

Most fatalities in the operating room in which anesthesia is the primary cause are

due to (1) asphyxia or inadequate ventilation, (2) overdosage of the anesthetic drug, (3) a combination of overdosage and asphyxia, (4) untoward reactions to drugs or (5) sudden severe, neurogenic shock.

Often fatalities ascribed to anesthesia are found after careful postmortem examination to be due to causes not related to anesthesia. Emboli caused by clots, air or fat head the list. Sudden cardiac failure due to coronary artery disease or other cardiac ailments is the most common cause. Severe untreated or irreversible shock causes death during operation, but rarely is death sudden and unexpected. Cerebral vascular accidents are uncommon, but do occur. Anoxia, carbon dioxide excess, excitement due to inadequate preparation and the use of vasopressor drugs may precipitate cerebral hemorrhage. Death, however, occurs after operation and seldom is it sudden. Uncontrollable massive hemorrhage from technical errors may account for some operating room fatalities. Adrenal insufficiency is a rare but possible cause of sudden death during operation.

In the absence of postmortem examinations, operating room fatalities are often ascribed to obscure, highly speculative causes. The mythical status lymphaticus has in the past provided an explanation for technical anesthetic errors. This syndrome is no longer accepted as a clinical entity. Vagovagal reflex is another overemphasized and convenient alibi for technical errors of anesthesia. Controversy exists concerning this reflex. Its occurrence, however, is not denied, but how frequently it causes death is debatable. Available evidence indicates that vagovagal reflexes do not cause death except when complicated by anoxia. When oxygenation is adequate and no disturbance of acid-base balance exists, sudden death does not occur irrespective of the intensity of the vagal stimulation.

Fatalities due to asphyxia during general anesthesia result from (1) reduction of the oxygen tension of the inhaled mixture, (2) obstruction to respiration from relaxed tissues, secretions blood or vomitus or (3) inadequate tidal exchange from hypoventilation. Death is usually due to anoxia. Hypoventilation often results in respiratory acidosis which may be the causative factor rather than the suboxygenation. Deaths due to overdosage are actually asphyxial deaths because, under these circumstances, paralysis in the respiratory centers causes apnea.

Obstruction of the respiratory passages accounts for the majority of asphyxial

the stomach contents to gravitate into the nasopharynx.

4. Flammable anesthetics are not used in situations in which a fire hazard exists. Explosions may cause death of the patient by rupture of the trachea and alveoli and by other manifestations of a blast injury. Operating room personnel are not immune from injury.

5. Premedication should always be given if possible. Attempts to anesthetize patients without adequate premedication lead to unsatisfactory results.

6. The anesthetic should be administered by one trained in the principles of anesthesia. The tendency to consider anesthesia lightly is all too prevalent. A procedure of minor consequence from a surgical view point may become a major one when performed under general anesthesia. The practice of relegating the administration of anesthetics to interns and other personnel not familiar with the fundamentals of anesthesia should be condemned. General anesthesia should not be administered by persons not familiar with the basic principles of anesthesia and resuscitation. Generally physicians instructed in this aspect of medicine administer anesthetics. However the demand for personnel to administer anesthetics is so great that nurses are also taught the basic principles and administer anesthetics under supervision and direction of the surgeon or an anesthesiologist.

7. General anesthetics are not administered in situations in which no provision is made for recovery from its effects. A recovery room with a bed, suction, emesis basins and all the necessary resuscitative paraphernalia is highly desirable.

RESUSCITATION

Resuscitation is the restoration to life of the apparent dead. In no aspect of medicine do situations requiring resuscitation arise as frequently as they do in anesthesia. There are two phases to resuscitation: ventilatory which consists in starting artificial respiration for apnea, and cardiac, which is an attempt to reactivate a heart which has ceased to beat.

Ventilatory Resuscitation. Whenever ventilatory efforts cease, irrespective of the cause artificial respiration is indicated. The most practical and effective method of artificial respiration in operating rooms is the intermittent insufflation technique. The mask of the inhaler of the anesthesia apparatus is held firmly on the patient's face and the rubber breathing bag is compressed rhythmically sixteen to twenty times per minute. Inspiration is active, expiration is passive, owing to the elastic recoil of the lungs. The iron lung and other mechanical respirators are used only for protracted periods of artificial respiration. When an anesthesia apparatus is not available, manual methods may be used. The most practical for the operating room is Sylvester's method because it is performed with the patient in the supine position (Fig. 6). The patient's wrists are grasped by the operator standing at the head of the operating table. The thorax is compressed with the patient's elbows. The arms are then extended over the patient's head to inflate the thorax. The procedure is repeated fifteen to twenty times per minute. The arm lift back pressure method of Neilsen may be used for the prone position. Mouth-to-mouth breathing is an often forgotten effective method. The



Figure 6 The Sylvester method for performing artificial respiration. a, Inspiration is accomplished by extending the patient's arms over his head. b, Expiration is accomplished by compressing the elbows along the lower chest wall. This method is adaptable during surgery when no other immediate method of resuscitation is available. (Author's article in Ochsner and DeBakey: *Christopher's Minor Surgery* 7th ed.)

sympatholytic effect. Hypotension difficult to reverse which leads to serious consequences, may develop during anesthesia and operation.

The time and route of administration are important. The optimum effects are attained when the narcotic is administered subcutaneously one to one and one-half hours prior to the anticipated time of the induction of anesthesia. In urgent situations the optimum effects are obtained within five minutes if the drug is administered intravenously at a slow rate. The narcotics are combined with belladonna alkaloids—atropine, hyoscyamine or scopolamine—which are anticholinergic substances. They are used to minimize the secretion of mucus and saliva. Scopolamine and hyoscyamine are more effective than atropine. Scopolamine depresses the cerebral cortex and enhances the effects of morphine. The sedation is accompanied by amnesia and an air of indifference which facilitates operation and obviates psychic trauma. Atropine and hyoscyamine stimulate the cortex and antagonize the sedative action of morphine. The belladonna alkaloids are administered simultaneously even by the intravenous route. The average adult dose is $\frac{1}{4}$ grain (15 mg.) morphine with $\frac{1}{400}$ grain (0.6 mg.) of belladonna alkaloid. For patients beyond the fourth decade of life the morphine is reduced to $\frac{1}{6}$ grain (10 mg.). Patients in the sixth and seventh decades tolerate less— $\frac{1}{6}$ to $\frac{1}{8}$ grain (7.5 to 10 mg.). Those who are older require still less— $\frac{1}{8}$ to $\frac{1}{16}$ grain (5 to 7.5 mg.). The belladonna alkaloid is reduced correspondingly in a ratio of 1 part to 25 parts of morphine. Morphine is omitted if basal narcosis with an intravenous or a rectally administered drug is contemplated. In infants and children, ultra short acting barbiturates may be administered rectally for basal narcosis (1 gm. for each 50 lbs. of body weight) one-half hour prior to induction of anesthesia. Preanesthetic sedation is desirable but is usually omitted if the patient is to be ambulatory immediately after operation. Ambulation after the administration of narcotics and hypnotics or general anesthesia is undesirable. Ataxia, drowsiness, nausea and vomiting are common in ambulatory patients after narcotics are administered. If the patient is hospitalized, as he should be for most surgical procedures, morphine and scopolamine in the usual doses are employed. Barbiturates are recommended prior to regional anesthesia to

allay apprehension and to minimize or avoid convulsions due to local anesthetic drugs. Barbiturates completely antagonize or prevent stimulation by an overdose of a local anesthetic drug in basal narcotic doses only. Secobarbital or pentobarbital ($1\frac{1}{2}$ grains, 100 mg.) orally or intramuscularly one and one-half hours prior to operation allays but does not prevent excitement and convulsions. All efforts should be made to perform the operation under circumstances which permit the use of premedication. Many a successful regional anesthetic procedure has been classed as unsatisfactory because the patient was apprehensive.

PRECAUTIONS

There are certain generalizations which are applicable to all forms of anesthesia but more particularly to general anesthesia.

- 1 No general anesthetic, no matter how brief in duration is the anesthesia or how light the plane, should be administered in situations in which there are no provisions for immediately instituting artificial respiration. The apparatus employed for inhalation anesthesia is used to administer artificial respiration by the intermittent insufflation technique. This method is wholly adequate for resuscitation under circumstances encountered during surgical anesthesia. Unless an unimpeded airway is established, no method of artificial respiration is successful. Airways of the pharyngeal type are satisfactory for relieving supralaryngeal obstruction. Contrary to certain teachings, endotracheal intubation is not always necessary for resuscitation. Valuable time is often wasted in attempting to introduce an endotracheal catheter before artificial respiration is instituted. Intubation is indicated only when the usual measures for establishing an airway fail.

- 2 A suction apparatus for the exclusive use of the anesthetist should be in each operating room. A curved metal suction tip which permits cleansing secretions from the hypopharynx is essential. A catheter is unsatisfactory for this purpose because the patient emerging from anesthesia clamps his jaws and bites down upon it. Besides catheters are limp and are difficult to direct into the pharynx in order to accomplish thorough cleansing.

- 3 The operation should always be performed on a table which can be tilted to the head-down position in the event vomiting occurs. Aspiration is prevented by allowing

- Hale D.: Anesthesiology Philadelphia F. A. Davis Company 1931
- Hampton, J. L., and Little D. M.: Controlled Hypotension in Anesthesia. *Lancet* 1:1299 1951.
- Hirschfelder A. D., and Blatter R. N.: Local Anesthetics. *Physiol. Rev.* 12:190 1932.
- Kurtz, C. M., Bennett, J. H., and Shapiro H. H.: Electrocardiographic Studies during Surgical Anesthesia. *J.A.M.A.* 106:131 1936.
- Lahet, G.: Regional Anesthesia Philadelphia, W. B. Saunders Company 1927
- Leigh, M. D., and Belton, M. K.: Pediatric Anesthesia. New York, The Macmillan Company 1930.
- Lundy J. S.: Clinical Anesthesia. Philadelphia, W. B. Saunders Company 1912.
- Papper E. M.: Renal Function during General Anesthesia in Operation. *J.A.M.A.* 152:1686, 1933.
- Robbins, B. H.: Cyclopropane Anesthesia. Baltimore Williams & Wilkins Company 1940.
- Robbins, B. H., and Lundy J. A.: Curare and Curare-like Compounds. *Anesthesiology* 8:348, 1947
- Rupp R. E.: Modern Concepts of Refrigeration Anesthesia. *Anesth. & Analg.* 22:18 1943.
- Saklad, M.: Grading Patients for Surgical Procedures. *Anesthesiology* 2:281 1943.
- Seever, M. H., and Waters, R. M.: Pharmacology of Anesthetic Gases. *Physiol. Rev.* 18 447 1938
- Slocum, H. C., Hoeftich, E. A., and Allen, C. R.: Circulatory and Respiratory Distress from Extreme Position on the Operating Table. *Surg., Gynec. & Obst.* 85:1051 1947
- Smith, H. W., and others: The Effect of Spinal Anesthesia on the Circulation in Normal Unoperated Man with Reference to Autonomy of the Arterioles and Especially Those of the Renal Circulation. *J. Clin. Invest.* 18 319 1939
- Stephen, R.: Elements of Pediatric Anesthesia. Springfield, Ill., Charles C Thomas, Publisher 1934
- Virtue, R.: Hypothermia. Springfield, Ill., Charles C Thomas, Publisher 1933
- Waters, R. M., Orth O. S., and Gillespie, N. A.: Trichlorethylene Anesthesia and Cardiac Rhythm. *Anesthesiology* 4 1 1943.

operator inflates the lungs by placing his lips to those of the patient and pinching the nose. Too often resuscitative efforts fail because they are not instituted soon enough and because the airway is poor. Respiratory stimulants are rarely of any value for resuscitation.

Cardiac Resuscitation. The term "cardiac arrest" is used to designate unexpected cessation of effective cardiac activity occurring in the operating room or immediately after operation. The causes of cardiac arrest have been enumerated in the discussion of operating room fatalities. When cardiac arrest is suspected, the chest should be opened immediately and attempts made to resuscitate the heart. Cardiac arrest is due to asystole or ventricular fibrillation. It is imperative that massage be instituted as quickly as possible so that an effective head of blood pressure is maintained in the vascular system. Artificial respiration must be carried on simultaneously to provide proper oxygenation. Unless resuscitation is instituted immediately cerebral damage invariably results if the patient survives. It is useless to open the thorax and massage the heart unless effective artificial respiration is performed simultaneously.

The technique of cardiac resuscitation is simple. An incision is made between the fifth and sixth ribs on the left side from a point one inch lateral to the sternum to avoid the internal mammary vessels. The heart is grasped between both hands and compressed rhythmically at a rate as near normal as is possible so that an effective arterial pressure is maintained. If the myocardium is atonic and depressed, 0.25 mg. epinephrine (1:1000) or 5 cc. of 5 per cent calcium chloride is injected into the right atrium. Physical methods are necessary to reverse ventricular fibrillation; drugs are ineffective. The heart is shocked with the ordinary 110 volt-60 cycle alternating current. The shocks, $1\frac{1}{2}$ amperes for one-tenth second, are delivered by broad electrodes placed over the surface of the heart. Asystole develops after several shocks and massage is maintained until the beat resumes. Defibrillators specifically designed for the purpose are now available which should be a part of emergency operating room equipment.

READING REFERENCES

Adams, R. C.: *Intravenous Anesthesia*. New York, Paul B. Hoeber 1944.

- Adriani, J.: *The Chemistry of Anesthesia*. Springfield, Ill., Charles C Thomas, Publisher 1946.
- Adriani, J.: *The Techniques and Procedures of Anesthesia*, 2nd ed. Springfield, Ill., Charles C Thomas, Publisher 1956.
- Adriani, J.: *Local and Regional Anesthesia for Minor Surgery*. S. Clin. North America 31:1507 1951.
- Adriani, J.: *Deaths from Anesthesia*. M. Times 80: 329 1952.
- Adriani, J.: *The Pharmacology of Anesthetic Drugs*, 3rd ed. Springfield, Ill., Charles C Thomas, Publisher 1953, pp. 3-7.
- Adriani, J.: *Nerve Blocks*. Springfield, Ill., Charles C Thomas, Publisher 1954.
- Adriani, J.: *The Selection of Anesthesia: Its Pharmacological and Physiological Basis*. Springfield, Ill., Charles C Thomas, Publisher 1955.
- Adriani, J., and Roman, D. A.: *Saddle Block Anesthesia*. Am. J. Surg. 71:12, 1946.
- Alexander, F. D. A., and Cullen, S. C.: *Premedication*. Am. J. Surg. 34:428, 1936.
- A.S.A. *Fundamentals of Anesthesia*, 3rd ed. Philadelphia, W. B. Saunders Company 1954.
- Beecher, H. K.: *Anesthesia for Thoracic Surgery*. Springfield, Ill., Charles C Thomas, Publisher 1952.
- Beecher, H. K., and Adams, R.: *Ether Anesthesia in the Presence of Pulmonary Tuberculosis*. J.A.M.A. 118:1204, 1942.
- Bonke, J.: *Management of Pain*. Philadelphia, Lea & Febiger 1953.
- Brewer, N., Luckhardt, A. B., Least, W. N., and Bryant, D. S.: *Reflex of the Glottis by Stimulation of Visceral Afferent Nerves*. Anesth. & Analg. 13: 257 1934.
- Burnstein, C. L.: *Fundamental Considerations in Anesthesia*. New York, The Macmillan Company 1950.
- Clement, F.: *Nitrous Oxide Anesthesia*. Philadelphia, Lea & Febiger 1952.
- Collins, V. J.: *Principles and Practice of Anesthesiology*. Philadelphia, Lea & Febiger, 1952.
- Courville, C. G.: *Untoward Effects of Nitrous Oxide Anesthesia*. Mountain View, California, Pacific Press Publishing Association, 1939.
- Courville, C. B., and Batten, C. T.: *Mental Disturbances Following Nitrous Oxide Anesthesia*. Anesthesiology 1:261 1940.
- Cullen, S. *Anesthesia in General Practice*, 4th ed. Chicago, Year Book Publishers, 1956.
- Dripps, R. D.: *The Immediate Decrease in Blood Pressure Seen at the Conclusion of Cyclopropane Anesthesia*. Anesthesiology 8:15, 1947.
- Firothanes, a collection of papers. Canad. Anesth. Soc. J. 4:187-294, 1957.
- Folkes, F. F.: *Some Problems of Geriatric Anesthesia*. Anesthesiology 11:737 1950.
- Gillette, N. A.: *Signs of Anesthesia*. Anesth. & Analg. 22:275 1943.
- Gillette, N. A.: *Endotracheal Anesthesia*. Madison, University of Wisconsin Press, 1948.
- Graubard, D., and Peterson, M.: *Intravenous Procaine*. Springfield, Ill., Charles C Thomas, Publisher 1950.
- Griggs, T., Adriani, J., and Berson, W.: *Adds to Pediatric Anesthesia*. Anesth. & Analg. 32:340, 1953.
- Guedel, A. E.: *Inhalation Anesthesia*. New York, The Macmillan Company 1950.

factors which affect a burned patient. It is no longer just a local wound to be treated, it is a wound affecting the general condition of the patient. The wound and the systemic disturbance must be treated at the same time. Better understanding of the treatment of shock and altered nutrition, the control of infection and early coverage by skin grafting of the burned wound have been of great value in the improved management of these patients.

Burn wounds are caused by the contact of the skin with sufficient thermal energy to create pathologic changes within the skin and then, secondarily a response by the patient. These wounds may be caused by contact with flame, scalding water, steam, electricity and chemicals and the irritation of poison gases. Accidents from gasoline and kerosene explosions are a common cause of deep burns. One type of burn is commonly referred to as the "flash burn" and this is not actually a special type. Such a burn is caused by exposure to any high intensity heat for a very short interval and is confined to the exposed skin such as on the hands and face. It may easily occur in a gas explosion.

Burns are most common in individuals in the extremes of age and mortality rates are likewise higher in these groups. Males are more often burned than females, but the mortality is higher in the female. Burns have been a notorious cause of permanent deformity and loss of earning power and constitute a potent source of hospital bed occupancy following trauma. A study made of bed occupancy has shown that about one-third of burned patients require hospital care. The number of hospital days spent is unknown, but it has been estimated to approximate several hundred days in a hospital for each subject who has died as a result of burns. In 1958 there were 6405 deaths from fire and explosions in the United States. It is further estimated that the yearly incidence of seriously burned patients is roughly twenty times greater than the number of thermal deaths. Much can be done prophylactically by educating the public concerning the dangers both in the home and in the factory or the careless use of kerosene, gasoline or other flammable liquids or articles. Most of all the burning injuries of children could be avoided if education were practiced. Outlawing the sale of flammable clothing has been shown to be important in reducing the incidence of serious burns. Such clothing, however, is still

sold. In time of war the incidence of burns in combat zones and in the rear areas from gasoline explosion, flame throwers, incendiary bombs or simple accidents creates large numbers of long term casualties. Atomic bombings can of course be a source of a large number of seriously burned casualties.

Burned patients constitute a high percentage of those seen following catastrophes and it is necessary that hospital personnel be aware of the importance of being prepared for a sudden influx of large numbers of burned and injured patients. This would mean previous planning for their care and a stockpiling of the few essential items necessary for local wound care and the treatment of shock.

Depth of Burning Injuries. The usual classification of burns is based on the depth of the injury: first, second and third degree burns.

First degree burns are superficial injuries involving the epidermis and heal rapidly without scarring.

Second degree burns are also called *partial thickness burns*; they heal by epithelial regeneration if infection and other irritations can be avoided. The skin destruction involves varying depths of the corium as well as the epidermis. Two types of second degree burns are recognized: superficial partial thickness and deep dermal burns. A superficial partial thickness burn heals within ten to fifteen days. When recently healed, the surface lacks normal pigmentation. The color and architecture of the healed skin eventually approach normal skin after a few months. Deeper burning within the skin may leave sufficient viable epithelial appendages which permit slower but eventual epithelialization of the surface. The deeper second degree burns, called deep dermal burns, may heal within four weeks. The healed surface consists of thin, fragile skin called scar tissue epithelium. Future replacement of this skin with a thicker skin graft is frequently necessary to improve both function and appearance.

In full thickness or third degree burns the entire thickness of the skin is destroyed beyond repair and frequently the injury involves the underlying tissues. The depth of this injury may extend to and include the muscles, nerves and tendons. In electrical injuries involvement of the bone and joints is not uncommon. These deep burns cause death of all tissues which have received sufficient thermal energy. Eventually

THERMAL AND IRRADIATION INJURIES

Burns

By JOHN L. BELL, M D

Burns are thermal wounds which vary greatly in extent, depth and location. The responsibility of the doctor to the burned victim begins with the management of the acute injury. It should not end until the patient has been restored to an acceptable position in society. Any physician may be called upon and should be able to render adequate emergency care to burned patients, especially in the time of a catastrophe. In severe cases, however, the continuation of treatment often requires the attention of a surgeon who is familiar with the more intricate aspects of deep wounds resulting from thermal injury. In burns the objectives of care are to preserve life, to obtain healing of the wound as early as possible and to minimize loss of function and disfigurement.

Initially the magnitude of the burn injury can be anticipated by assessing the size and the depth of the burn. In extensive burns, immediate systemic alterations occur and should be managed before extended care is given to the local wound. Superficial

and deep burns should be differentiated as soon as possible because the latter often require well planned surgical procedures in order to achieve early closure of the wound with a minimal loss of function. Acute burns, superficial or deep provide highly susceptible avenues for local or invasive infections until complete healing has been attained.

Superficial burns heal spontaneously if infection and mechanical or chemical irritation of the wound are avoided. The topical application of drugs to burned surfaces has little effect upon the ability or rate for spontaneous healing of superficial burns. The skin is destroyed in deep burns and healing is obtained by skin grafting after removal of the necrotic tissue and the proper preparation of the recipient site.

With the renewed interest in the treatment of burns brought about by the recent wars and the atomic and thermonuclear bombs, the therapy of burns has shown great advancement. This progress is due to better understanding of the combination of

be accurately assessed immediately upon inspection following the injury. A fairly accurate estimation can be made with the help of the Berkow scale for estimating the extent of a burn (Fig. 1). A more rapid but a cruder estimation can be made by the use of the rule of nines (Fig. 2). The employment of this method, however frequently leads to dangerous overestimation of the percentage of body surface involved.

Clinical Course of Burning Injuries. When sufficient heat is applied to the body surface to create a burning injury, there is precipitated a chain of events which is now quite well known. When the extent of the burn is sufficient—beyond 15 to 20 per cent of the body surface—there is a general systemic response known as burn shock. Immediately after the burning injury there is sudden temporary pain which is often controlled when the involved region is protected from the cooler air.

In first degree burns skin redness is not persistently painful. This type of injury usually evokes little general response. Partial thickness burns are common after a scalding injury and, if these are sufficiently large, burn shock may occur. The wounds heal slowly and the healing rate depends upon the avoidance of infection and other irritants which could easily convert a superficial, incomplete thickness burn into a deep or whole thickness variety. When not infected or irritated, these burns usually heal. Healing occurs by the ingrowth of epithelium from the remaining elements, such as the sweat glands and hair follicles.

The third degree, or whole thickness burns occur most commonly when clothing catches fire and if the area is large enough—beyond 15 to 20 per cent of the body surface—will produce a shock state. The local burned wound appears charred frequently but may be white or range from brown to black in appearance. It is anesthetic to sensory stimuli. The full thickness burn is usually surrounded by a pink areola of less deeply burned regions which will epithelize and heal within ten to twelve days, leaving an open wound of varying size which is covered eventually with granulation tissue. The local wound can easily become infected after the first few days because of the presence of dead tissue at body temperature which encourages any type of infection. The eschar will begin to separate from the unburned tissue about the fifth to eighth day and if neglected will ultimately slowly slough away. This slough acts as a sequen-

trum and the host attempts to separate it from the underlying structure. The autolysis of the burned tissue accounts for a marked increase in wound discharge. Later if the wound is disregarded, local or invasive infection supervenes.

A burning injury will evoke a marked endocrine or stress reaction, which is almost maximal in degree. Initially the patient has great fear of pain and scarring and later if the burn becomes chronic the patient will lose morale and exhibit definite psychic changes. Extensive burns commonly produce this change and patients refuse to eat and complain bitterly of even minor discomforts. Weight loss and profound secondary anemia can become very marked as a result of the constant loss of protein from the wound. If infection has been added, the burn is thus deepened and protein and fluid loss is accentuated.

Burn wounds should be considered as potentially contaminated, open, soft tissue injuries, and as such demand careful local care at all stages to control infection until healing has occurred. It is the objective in all burn wounds to convert the open contaminated wound into a closed wound as early as is possible and practical. Any unnecessary delay in converting these open wounds into closed wounds deepens the injury and, when a sufficient extent of the body surface is involved, life itself may be jeopardized by septicemia.

Pathologic Changes of Burned Surfaces. The first degree burn is an injury of a superficial nature in which blebs do not form. In this type there is early redness and some edema but no necrosis.

The second degree, or partial thickness, burn denotes an injury in which there is superficial necrosis and, initially there is edema and bleb formation. The blebs are found within the layers of the skin and may be sufficiently large to rupture spontaneously. The burned surface is either pink or red and mottling can usually be noted. Exudation may be profuse. The wound extends into the dermis and there are viable remnants of epithelium, such as sweat glands and hair follicles, from which healing will occur without the necessity of grafting. The deep dermal burns are those in which there are only scattered skin appendages left for regeneration. It is impossible to diagnose a deep dermal burn in the acute phases. A healed burn of this type has a thin, fragile epithelial covering, which breaks down frequently after minute trauma.

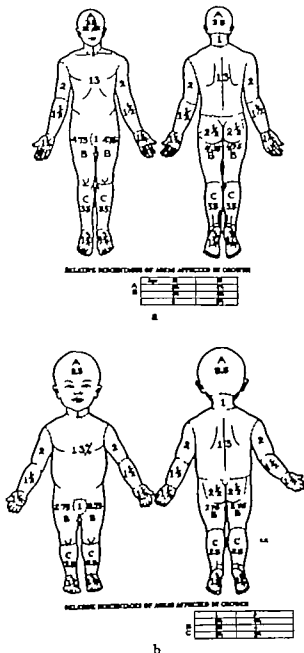


Figure 1 Bedkows plan for estimation of body surface burned. First degree burns are excluded in the estimation. a, Burn area estimation chart for adults. Below are given head, thigh and leg percent ages for patients from 10 to 15 years of age. b, Burn area estimation chart for infants 0 to 1 year of age. Below are given head, thigh and leg percentages for children from 1 to 5 years of age.

such a wound demarcates and the body attempts to slough off the damaged area as a foreign body creating a large open wound which must be closed by skin grafting.

Most burns are irregular in depth because of the protection of clothing or the anatomic variations in the thickness of the skin. The usual pattern is that of the greatest depth being in the center of the injured area and lessening of depth toward the periphery of

the wound. The depth of the burn at the time of initial inspection immediately after the injury is difficult to assess, except in those in which there is actual charring of the skin with anesthesia of the area, which indicates complete death, or a whole thickness burn.

Extent of Burns. The size of the burned wound is another indication of the severity of the injury. There is a definite rough correlation between the extent of the burn and the degree and severity of burn shock. However there is a wide variation in the individual response to the injury and often the extent of the burn can be misleading, for in one patient a 15 to 20 per cent body surface burn can be overwhelming, while in another patient, similar in size, it seems to evoke a minimal response.

A common error is that of overestimating the extent or the percentage of the body surface burned. The reason for this is that the exact extent of the burn wound cannot

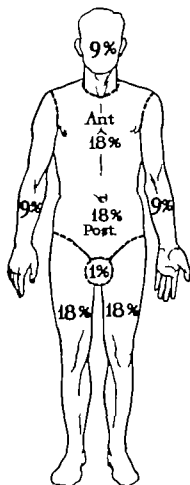


Figure 2 A simplified method of estimating body surface burned—"the rule of nines." This is a crude method and the surgeon must be constantly aware that he must not overestimate the body surface burned.

that there is a diminished response as measured by the steroid excretion and the eosinophil count.

The hematologic changes which occur after a burn are of two varieties. First the red cells are often trapped and held within vessels which are permanently damaged by the heat. Moreover the red cells are very susceptible to heat and this increases their fragility. This marked fragility produces a great increase of cell debris which can produce hemoglobinemia, which when sufficiently severe can affect the function of the liver and the kidney. The continuation of hemoglobinemia can produce delayed jaundice and hepatitis. When these occur the prognosis is greatly altered.

The second response of the blood is hemocoagulation due to sudden and continued withdrawal of the fluid portion of the blood into the burned region. The blood is more viscid than normal and a rising hematocrit reading is common in the immediate postinjury course. With the increase in the viscosity of the blood, the blood volume is actually lowered and the body attempts to restore and maintain the blood volume by withdrawing from the available fluid reserves, thus creating dehydration of the unburned regions. In profound shock, constriction of the peripheral blood vessels is common. Occasionally these markedly constricted vessels are filled with black syrup-like blood.

There is an immediate leukocytosis which is both true and relative owing to the concentration of the blood. This leukocytic response subsides unless infection supervenes. The blood changes are only a matter of degree and will not be apparent in a patient who does not exhibit some form of burn shock.

Changes in the urine depend upon the maintenance of the blood supply to the kidney. If this blood supply is reduced as the result of burn shock, the volume of urine is reduced and the specific gravity increases. Albuminuria is common. Later nitrogen excretion is greatly increased, which correlates well with the stress response of these patients. In patients with serious burns in whom there is a marked breakdown of red cells hemoglobinuria may occur. Urinary changes will continue until there is correction and improvement of the circulating volume of blood. After three to four days diuresis will be noted and this is the result of the edema fluid being reabsorbed.

The blood chemical changes following a

burn are due in part to the dehydration of the blood and secondly to the alarm reaction. The sodium is conserved following a thermal injury and the blood level is lowered. Potassium may be lost particularly when there is diarrhea or vomiting. Transient hyperglycemia occurs after the burn. There is a temporary rise in plasma proteins and later with continued withdrawal of the fluid portions which are high in proteins there is produced a hypoproteinemia. The circulating proteins are drawn into the burned tissues and this state remains until there is a reversal of the edema fluid from the burned regions usually in three to four days. A dilution of the circulating proteins occurs after the diuresis. The protein loss may continue and be accentuated if infection has been added. Reversal of the albumin globulin ratio occurs and such severe lowering of the proteins may produce a hypoproteinemic edema.

The studies on vitamins have shown a marked reduction in the ascorbic acid levels immediately following a burn. This becomes more marked if the burn has been allowed to become chronic.

The pathologic changes seen following a burn depend upon the extent and the depth of the burn, and particularly upon the patient's individual response to the injury. Not all burned patients will exhibit all of the changes mentioned, not even those with extensive burns. The injuries do not follow an exact pattern. In some patients the injury is an overwhelming assault and causes marked tissue damage and chemical changes. Protein loss may be rapid and marked weight loss may occur. The body fat is consumed first and then the protein mass is used to provide sufficient calories to maintain the increase in the metabolic rate of such patients. There is a catabolic response in each patient which creates a negative nitrogen balance. This can be limited when there is proper protein replacement by blood, diet and supplementary feedings. When protein loss is allowed to continue, the nitrogen reserves of the body can be so depleted that healing can never be accomplished and it is in these patients that infection is common and all attempts at skin grafting fail.

Infection added to the large open wounds creates havoc and septicemia is a common cause of death. Pyogenic infections are greatly enhanced because of the presence of dead tissue, body moisture and body heat. When malnutrition and secondary anemia

The pathologic changes which occur within the skin and subcutaneous tissue or the deeper structures, depend naturally upon the intensity of the heat and the length of time that it has been applied. Full thickness burns may immediately show heat coagulation and necrosis. The underlying subcutaneous tissues may be dry and hard and surrounded by marked edema. Subcutaneous vessels are filled with thrombi. When involvement of the muscle has occurred, the tissue immediately becomes very pale and friable. In electrical burns the bone may be involved but the demarcation of the dead bone is very slow and takes many weeks.

Systemic Alterations Following Burns. The burning injury evokes a response which is characterized by rapid pouring out of fluid into and around the wound. It is as though a fluid barrier were thrown about the site to wall off the injury. This fluid comes from the local vessels, lymphatics and capillaries and also from the fluid within the cells which have burst as the result of the heat applied.

The fluid which escapes into the burned area is carried away by lymphatic drainage from the local site, but soon the amount of fluid exceeds the ability of the lymph channels to withdraw it and the accumulation will pile up in the interstitial spaces, thus producing edema, not only of the wound but also of the area surrounding the wound. Large amounts of fluid can be hidden when infiltrated deep into the soft tissues and about the burned organs. This can be misleading if one does not realize that this amount of fluid can represent a high percentage of the total circulating fluid volume of the body. The fluid escaping from the circulation into the wound is not identical, but is similar to plasma. It has a lower concentration of protein than has plasma but has the same concentration of electrolytes. In some instances, the swelling and the edema may become so intense as to jeopardize the vascular supply of the part.

The pathologic changes in organs which are not directly involved by heat could be assumed to result from reduction of the circulating blood volume, first creating immediate dehydration of these organs and later if the blood flow continues to be lowered, depriving these organs of their blood and oxygen, and hence interfering with their function. This is apparent in the kidneys, brain and liver where irreversible changes may occur.

The gastrointestinal tract is not often directly affected by heat, but there may be an indirect response varying from anorexia to vomiting and paralytic ileus is a common complication in patients with severe shock. One of the rare complications of the severe burn is a Curling's ulcer an ulceration of the upper gastrointestinal tract, and in some cases the only indication is hematemesis or blood in the stools. In others there may be an actual perforation of the ulcer causing peritonitis and death. The cause of Curling's ulcer is as yet unknown. It has been ascribed to the general stress response in which gastroduodenal perforation is not uncommon.

Pathologic changes within the respiratory tract depend upon the actual inspiration of either hot fumes or poison gases seldom does flame itself penetrate beyond the upper respiratory region. The upper tract response is edema, which may be so marked as to produce swelling of the arytenoid tissues which will actually obliterate and obstruct the larynx. Lower respiratory tract injury produces damage to the bronchial and bronchiolar epithelium. Spasm and edema and, eventually desquamation may cause respiratory obstruction and death. In lower tract injuries there is a great outpouring of frothy fluid which signifies marked involvement.

The cardiovascular system seldom exhibits altered changes other than the usual responses which would be elicited in shock from any cause. There is, however a great tendency for thrombophlebitis to occur because of the prolonged period of thickening of the blood, due to hemoconcentration. Thrombotic phenomena are not rare.

With reduction in the circulating blood volume there is a reduced cardiac output in burn shock and, late in the course, the blood pressure will fall, but this has no neurogenic or endocrine etiologic significance. It is purely due to lack of sufficient blood volume to maintain the blood pressure at a working level.

Burning injuries evoke a general endocrine response. The alarm reaction in burns is maximal. Initially there is a rapid rise of the ketosteroid excretion and a lowering of the number of eosinophils. There is a loss of nitrogen through the kidneys which accounts for the negative nitrogen balance which appears in a burn patient after burn shock. This response to stress will continue until the wound is healed, while complications, such as infection, may prolong the response. In fatal cases it may be seen early

suggested are only a guide to start the therapy

It is apparent that no formula takes into account the variation in the individual's response to the different types depths and extent of burns. No formula takes into account the age of the patient, the previous health or hydration or the individual's reaction to trauma. Clinically it is seen that children with extensive scalding have shown accentuated shock response for there is a very intense persistent fluid loss into and from the wound. This is more apparent than in patients with a burn of similar extent due to a flame type of injury in which the skin is hard and charred, prohibiting the oozing of fluids from the wound. However the damage to the red cell mass associated with a flame burn is greater than that due to scalding. Anemia always occurs earlier following a flame burn. Actually there is true anemia which is not apparent until late, when diuresis has occurred and the hemoconcentration has been alleviated.

A formula may be useful as a guide to initiating treatment of burn shock. The formula propounded by Evans, in 1952, is based upon the percentage of body surface burned times weight in kilograms. This formula permits an estimation of the amount of fluid which is to be provided in plasma, plasma expanders or blood needed during the shock state. Evans believed that an equal amount of isotonic saline solution should be administered during the shock phase and that some fluid should be given for renal excretion and the insensible loss.

For example, the fluid requirements in the first twenty four hours for a man weighing 70 kg. and suffering from a 30 per cent burn would be as follows:

Plasma, plasma substitute or whole blood	$70 \times 30 = 2100$ cc.
Saline solution—0.9 per cent	$70 \times 30 = 2100$ cc.
Dextrose in water—5 per cent	2000 cc.
Total in the first 24 hours	6200 cc.

Evans recommended that the fluid requirements should be given in the following manner. One-half of the total estimated fluid for the first twenty-four hours should be given in the first eight hours after burning, and one-quarter in each of the subsequent eight hour periods of the first day. During the second twenty four hours the colloid and electrolyte requirements are roughly one-half of the previous amount recommended for the first twenty four hours. Evans wisely recommended that, after forty

eight hours fluids should be supplied mainly by the oral route. Fluid requirements for a patient with burns involving more than 50 per cent of the body surface should be estimated as 50 per cent surface burns and never for more than this, for no patient should receive more than 10 000 cc. of fluid in the first twenty four hours. A modification of the Evans formula is advocated by the Brooke Army Medical Center. The Brooke formula differs from the Evans formula in that an electrolyte-colloid ratio of 3:1 is proposed.

There are but few chemical or laboratory tests which will aid in judging the efficacy of burn shock therapy. However constant observation of the general appearance of the patient, the pulse rate the color and the degrees of restlessness and thirst will provide indications of progress. The most important and best index for judging the adequacy of burn shock therapy is the amount of hourly urinary excretion (Fig 3).

In any patient with clinical signs of shock, or in any in whom the extent of the burn suggests the possibility of burn shock, an indwelling catheter is placed in the bladder so that the hourly urinary flow may be accurately recorded. The initial residual urine in the bladder is examined in the usual routine manner. The objective of all shock therapy is to give sufficient fluid to obtain an average urinary flow of 25 to 50 cc. per hour for the average adult.

Most burned patients can tolerate limited amounts of oral fluids if administered at a definite rate that does not result in overloading the gastrointestinal system. Although the burn shock patient is very thirsty this condition must be combated by giving fluids intravenously because only limited amounts of oral fluid can be tolerated without vomiting. Vomiting is to be avoided because of the additional loss of electrolytes which will only further endanger the patient. Clinically it is found that an oral intake for an average adult of only 200 cc. per hour is adequate and that this reduces the total amount of fluids needed by vein. This amount of fluid given by mouth is well tolerated if it is a chilled solution consisting of salt and bicarbonate of soda. The solution contains 1 teaspoonful of salt and $\frac{1}{4}$ teaspoonful of bicarbonate of soda to a quart of water.

The colloid solutions are given in the form of either whole blood, virus-free plasma or one of the plasma expanders, and no rigid rule can be given as to how much of each

are allowed to progress many strains of virulent pyogenic bacteria can readily break through the wound barrier and gain access to the circulation.

Burn Shock and Its Treatment. The systemic response to burning injuries is called *burn shock*. This usually follows a burn which is greater than 15 to 20 per cent of the body surface, but it should be anticipated in any patient with burns which involve more surface area than the "flash burn" areas, that is the exposed hands and head.

There are many as yet unexplained reactions in burn shock and too often these are reputedly said to be due to "toxins." Burn shock is a combination of the responses of the local bodily defenses against a burning injury with resulting effect on the chemical composition of the blood plus the interplay of the endocrine glands. Burn shock results in reduction of the circulating blood volume, both as to the quality and quantity of the blood. The blood volume is reduced because of the continued withdrawal of serum and plasma like substances into the burned area, producing local edema as if the body were attempting to isolate the injury or to wall it off with a protective fluid barrier. This continuing withdrawal of fluid portions of the circulating blood produces dehydration of the unburned portion of the body and this continuing process increases the viscosity of the blood and produces a hemoconcentration. The reduction of circulating blood volume is reflected in the amount of blood and oxygen supplied to the vital organs such as the brain, the heart and the kidneys. Further a thermal injury will trap and destroy a certain amount of the red cell mass. Therefore, the fluid, plasma and red cell volumes are reduced as the result of burn shock.

Burn shock is a steadily progressive constantly changing phenomenon which continues for approximately forty-eight hours and will during this time demand constant attention. Edema formation is marked and occurs at a rapid rate during the first few hours. Clinical evidence indicates that burn shock reaches its maximum within four to twelve hours following injury.

The clinical picture of burn shock clearly manifests itself when the patient is carefully examined. In a patient who has 20 per cent or more of the body surface burned, there will be obvious signs of burn shock. The patient is moderately restless and apprehen-

sive and there will be pallor or cyanosis of the extremities and the mucous membranes. The extremities vary in temperature from cool to cold and sweat in variable degrees is observed. The patient rarely complains of pain but often will complain bitterly of the cold and ask to be covered by a blanket. Most patients are apprehensive and, if the shock is untreated, they become rather apathetic and this apathy may continue to an actual comatose state and death. The pulse rate is generally rapid and becomes more so as the shock progresses. This is far more notable, as are all other signs, in children and in elderly patients. The respiratory rate is slightly increased in burn shock and respirations are usually shallow. Thirst, a common complaint, is one of the outstanding clinical signs, for it is far more pronounced in burn shock than in shock produced by other trauma. Occasionally the thirst is even accompanied by vomiting. The severely burned patient will exhibit a collapse of the peripheral veins and it is difficult to obtain blood samples from the usual available veins without performing a cut-down. It is good practice to perform a cut-down and insert a good sized cannula, which will be the lifeline for later therapy. Occasionally in far advanced shock the blood within the collapsed peripheral veins is black and syrup-like in consistency. The blood pressure is not an accurate guide as to the presence or probability of burn shock.

The restlessness associated with burn shock is due to anoxia of the brain and is too often interpreted by the doctor as being due to pain. The use of morphine, given subcutaneously will not aid this situation, but rather its administration may obscure the usual findings of burn shock. It is imperative that attention be directed to the restoration of the blood volume to a working level. There is no drug which will aid or prevent burn shock.

The amount of fluid which must be provided to bring back the blood volume to normal, and to maintain it during the interval of burn shock for each patient, must be sufficient, first, to restore blood volume and the red cell mass to normal; second, to maintain blood volume at a normal range during the continued withdrawal of fluid, that is the first forty to forty-eight hours; and, third, to allow for renal deviation and insensible loss. It cannot be stressed too much that the amount of fluid necessary will vary widely from patient to patient and the formulas

or plasma substitute has been given, it has been the practice to administer whole blood at the ratio of two parts of virus free plasma, or substitute, to one of whole blood. In extensive full thickness burns, the damaged red cell mass may be partially restored by the early use of whole blood transfusions.

Whenever vomiting has occurred, additional electrolytes must be administered. If the urinary flow exceeds 50 cc. an hour the amount of fluid given intravenously should be reduced for it is obvious that too much fluid is being administered. If the urinary output is reduced to 15 cc. or less per hour inadequate amounts of fluid are being provided. The amount of fluid given orally is constant, the only variable factor in the treatment of burn shock is the rate of the flow of the solutions given intravenously which should be adjusted to the patient's general condition and the rate of urinary flow. Obviously the amount of fluid given and the amount of urinary output are altered with the extremes of age. In elderly patients, and in any patient with a pulmonary burn, it is best if the urinary output does not exceed 25 cc. per hour for there is always danger of overloading the cardiovascular and pulmonary systems.

At times the patient will benefit by the use of an oxygen tent, but this is only empirical for the oxygen-carrying capacity of the blood in burn shock is not greatly altered, unless there has been marked pulmonary injury from noxious gases or actual heat.

After burn shock has been controlled, there is beginning resorption of edema fluid from around the wound and large amounts of electrolyte solutions given at this time may overload the cardiovascular system and cause pulmonary edema. Diuresis occurs after the forty-eighth hour and continues for another one to two days. Careful observation of the urinary output and the patient's clinical course is necessary during this time.

Nutrition. After the patient has entered the phase of diuresis, a period of negative nitrogen balance with marked excretion of nitrogen bearing colloid is apparent. Clinically a series of events which are very evident will be seen. Progressive secondary anemia and rapid weight loss with marked reduction in the protein level of the blood and a reversal of the albumin globulin ratio occur. The surgeon should anticipate these events and combat them in a logical manner.

In order to combat weight loss and anemia, the patient should be provided with an adequate diet, whole blood and vitamins

so that this deterioration does not continue and that his general well being will not be so lowered that a regrettable infection develops in which the wound is subjected to the ever present danger of contamination.

The nutritional problem of a burned patient after recovering from burn shock and during the phase of healing can be very serious if there is any delay in complete closure of the wound. If the timetable of management of the patient can be kept so that the shock is efficiently treated and the patient is maintained at a good healing level, then the burn slough can be rapidly removed and the wound closed by the use of skin grafts within three weeks from the time of injury. After closure of the wound, nutritional problems are seldom troublesome for loss of protein from the wound ceases. When the wound is closed early there is likewise less danger of infection. Any delay in wound closure or failure to maintain the patient's nutritional level will be manifested by the sluggish appearance of the granulating wound and the poor growth of epithelium from the wound's edges. Actually the appearance of the burn wound is a reflection of the patient's general condition.

One should attempt to minimize the altered balance of metabolism by providing the patient with a diet which will supply the elements needed to compensate for the marked protein loss and meet the demands of tissue repair, red cell regeneration and metabolism. The dietary requirements are determined largely by the extent and depth of the burn. The diet must be palatable, yet properly balanced, and must be high in protein content.

It is recommended that there be a daily intake of from 2 to 3 gm. of protein per kg. of body weight, and from 3000 to 6000 calories per day as a basic minimum. Fat in the diet is of value because it will provide sufficient calories while sparing the ingested proteins which are so necessary for hemoglobin formation and wound healing. In the occasional patient who cannot tolerate this diet, as a supplementary measure it is wise to use intragastric tube feedings of high protein liquid mixtures to provide from 2000 to 3000 calories a day. The various high protein mixtures include some form of whole milk, powdered milk or protein concentrates. Mixtures high in fat content sometimes produce diarrhea and when this occurs adjustments must be made.

Marked vitamin deficiency particularly of vitamin C, accompanies severe burns. Daily

wound, namely the conversion of the open contaminated wound into a clean closed wound as rapidly as is practical for the patient. In burns, skin grafting is often demanded in closure of the wound.

First aid for a burn can be very simple in civilian practice for all that is required is to protect the wound carefully and to transfer the patient immediately to a hospital where facilities are available for complete care. The application of home remedies such as ointment, butter and lard, too often adds further delay and always adds unnecessary contamination. Simple protection by the use of the cleanest available material to exclude the air is the most practical type of first aid.

Immediately upon admission to the hospital, the patient should be quickly assessed as to the type and, roughly, the extent of the burn. Many minor burns do not demand hospitalization nor will the patients exhibit shock, but, when the burned area is larger than the "flash burn" regions, in all probability hospitalization will be required and burn shock may be anticipated.

The initial care of the burn is performed without the use of anesthesia. For those with extensive burns, anesthesia is contraindicated because of its deleterious effect on the accompanying burn shock.

Narcotic drugs are not routinely administered to the acutely burned patient. If pain is a factor a small dose of morphine may be given intravenously. In patients with burn shock, the subcutaneous injection of morphine is dangerous and useless. In these patients, the drug cannot be absorbed until the circulatory deficit has been corrected. If repeated doses have been given, morphine poisoning can occur with the return of circulatory efficiency. Restlessness is a symptom of severe burn shock and is not an indication for a narcotic. Pain can be greatly diminished if the local care is undertaken in a room which is moderately warm and where draughts of air can be avoided.

Under aseptic conditions, the burned site and the area well beyond it in all directions are gently cleansed with large pledgets of sterile cotton that have been dipped in a mixture of bland soap and warm sterile water. The wound is carefully flushed and all obvious blebs and loose bits of skin are removed. The procedure can be done carefully and yet rapidly. It is remarkably painless if gentleness is practiced and sterile water at body temperature is used for washing and flushing.

A single layer only of sterile, fine-mesh

gauze is placed over the wound and the entire area is then covered with a previously prepared dressing. The fine-mesh gauze may be impregnated with a light coating of petrolatum prior to its sterilization. This is held in place by some form of bandage which produces a uniform compression dressing. The compression dressing must not be tight and must have a definite amount of resiliency. If the burn involves an extremity, immobilization is secured by applying a splint. When the wound is covered and air is excluded there is almost complete absence of pain.

Every hospital or first-aid station should be equipped with previously prepared sterile dressings always in readiness for burned patients or extensive open injuries. These dressings are made with 2-4-ply Cellucotton with fine-mesh gauze attached to one side. They are made in two sizes, one suitable for small burns or burns of the hand and forearm, the other sufficiently large for either the posterior or anterior trunk, or to encase the entire lower extremity of an adult. These stored sterile dressings can be taken out quickly and applied rapidly. They are useful not only for emergency local care of the wound but also in later care if skin grafting is required. The dressings are of sufficient thickness to allow uniform compression to the open wound.

The petrolatum impregnated gauze is used only to permit ease of removal later. It is merely a mechanical agent and has no healing power. At the time of removal, the gauze should be gently lifted away to minimize tearing of the new epithelial layers.

The dressings are usually not disturbed for from five to eight days unless there is indication that they should be restored.

The initially applied burn dressing may be useful in most areas except the face and the perineum. These regions are usually left exposed.

One of the rediscovered methods of local care is that which is termed "exposure." This method relies on exposing the burned wound to the atmosphere and its value lies in drying of the wound which reduces the possibility of infection. It eliminates the necessity of dressings, but, in order to be successful excellent nursing care and a thorough understanding and ability to cooperate on the part of the patient are required. The dry crusts are allowed to remain in place until epithelialization has occurred, permitting the crusts to be lifted away.

Exposure of a burned area is initially pain

vitamin supplements should be given in the following amounts: vitamin C 1000 to 1500 mg, thiamine, 50 mg, riboflavin, 50 mg., and nicotinamide, 500 mg.

During the phase following burn shock and until the wound has healed, whole blood transfusions are often required. It is essential to maintain the hemoglobin and the total blood protein at a high level to combat infection and to facilitate normal epithelization. As a good working basis, the total proteins should be kept above 6 gm per 100 cc. but it must be cautioned that any estimation of the circulating blood protein is no criterion of the body stores of protein, which can be greatly depleted. Clinically it is of value to keep regular recordings of the patient's weight for this is a remarkably accurate guide to his progress. If this is not practical, there should be frequent checks by the laboratory of the hemoglobin, red cell and total protein values. If the blood levels and the patient's weight can be maintained, healing is greatly facilitated.

Infection. Infection can produce serious complications. Every burned wound is contaminated and when there is a break in the integrity of the epithelium infection may become invasive and be disastrous.

All of the factors which are necessary for bacterial growth are present in a burned wound. There is contamination, dead tissue, body moisture and body heat. When further bacterial contamination is added to the burned wound, the remaining useful epithelial structures which are necessary for healing of an incomplete thickness burn can be readily destroyed. Further the presence of infection can create a more profound wound defect. Infection elicits a greater response from the wound in that a much more rapid outpouring of protein and fluid will occur thereby compounding the problem of fluid loss. When a burn is first seen it is contaminated and it is necessary to prevent this wound, at all costs, from becoming infected. Therefore, careful aseptic care must be practiced throughout, from the time of the initial local care until the wound is healed. Pyogenic bacteria are the most common invaders. Frequently there is a mixed variety of bacteria present in these wounds. Septicemia is a frequent cause of death in patients with extensive burns.

One of the most potent sources of cross-infection is that of dressings done carelessly without due precaution against contamination. When large wounds are exposed to the air in a ward or dressing room there is

the ever present danger of bacteria being carried to the wound from the dust and draughts of air. It is imperative to take all precautions to avoid adding infection from this source.

Tetanus is not an uncommon complication in burns. Tetanus immunization must be given to all burned patients. If toxoid has been previously administered, a booster dose is all that is required. If doubt exists as to previous immunization with toxoid then tetanus antitoxin should be given.

The systemic use of antibiotics in burned patients is a much debated problem. Too frequently the antibiotics are administered routinely when they are not necessary. Unless there has been poor first aid, some delay in the initial care, or if the burn presents a complicating factor such as lung involvement or burns of the perineum, antibiotics should not be routinely administered. The use of proper antibiotics should be reserved either for definite complications or for short intervals as when burn eschar is removed or skin grafting is performed. Resistant infections due to *Staphylococcus aureus*, coagulase positive microorganisms, are being reported frequently and can reach epidemic proportions. This type of infection is mainly due to a breakdown of aseptic technique and a blind reliance upon antibiotics to prevent wound infection. Obviously a chronically infected burn demands antibiotic therapy. However this should not be instituted until a culture has been made and the sensitivity of the bacteria to the various antibiotics has been demonstrated.

The local application of antibiotics or antiseptics to an open wound has not proved to be the solution for the problem of infection. Too frequently one is prone to rely on a drug to clean the wound in preparation for surgery. This is not wise, for it is well known that the routine local use of a drug greatly increases the resistance of the bacteria and the sensitivity to it.

Initial Treatment of the Burned Wound. The objective of the care to be given to the burned patient is the immediate cleansing and protection of the wound and the repair of the systemic alteration resulting from the burn shock state. Initially a burn is very difficult to assess accurately as to depth or extent. Therefore, the wound must be protected and attention directed to care of the burn shock. After a few days, an accurate estimation of the extent and depth of the burn can be determined. The problem in burns is identical to that of any traumatic

wound, namely the conversion of the open contaminated wound into a clean closed wound as rapidly as is practical for the patient. In burns skin grafting is often demanded in closure of the wound.

First aid for a burn can be very simple in civilian practice, for all that is required is to protect the wound carefully and to transfer the patient immediately to a hospital where facilities are available for complete care. The application of home remedies, such as ointment, butter and lard, too often adds further delay and always adds unnecessary contamination. Simple protection by the use of the cleanest available material to exclude the air is the most practical type of first aid.

Immediately upon admission to the hospital, the patient should be quickly assessed as to the type and, roughly the extent of the burn. Many minor burns do not demand hospitalization nor will the patients exhibit shock, but, when the burned area is larger than the "flash burn" regions in all probability hospitalization will be required and burn shock may be anticipated.

The initial care of the burn is performed without the use of anesthesia. For those with extensive burns anesthesia is contraindicated because of its deleterious effect on the accompanying burn shock.

Narcotic drugs are not routinely administered to the acutely burned patient. If pain is a factor a small dose of morphine may be given intravenously. In patients with burn shock, the subcutaneous injection of morphine is dangerous and useless. In these patients, the drug cannot be absorbed until the circulatory deficit has been corrected. If repeated doses have been given, morphine poisoning can occur with the return of circulatory efficiency. Restlessness is a symptom of severe burn shock and is not an indication for a narcotic. Pain can be greatly diminished if the local care is undertaken in a room which is moderately warm and where draughts of air can be avoided.

Under aseptic conditions, the burned site and the area well beyond it in all directions are gently cleansed with large pledgets of sterile cotton that have been dipped in a mixture of bland soap and warm sterile water. The wound is carefully flushed and all obvious blebs and loose bits of skin are removed. The procedure can be done carefully and yet rapidly. It is remarkably painless if gentleness is practiced and sterile water at body temperature is used for washing and flushing.

A single layer only of sterile, fine-mesh

gauze is placed over the wound and the entire area is then covered with a previously prepared dressing. The fine-mesh gauze may be impregnated with a light coating of petrolatum prior to its sterilization. This is held in place by some form of bandage which produces a uniform compression dressing. The compression dressing must not be tight and must have a definite amount of resiliency. If the burn involves an extremity immobilization is secured by applying a splint. When the wound is covered and air is excluded, there is almost complete absence of pain.

Every hospital or first aid station should be equipped with previously prepared sterile dressings always in readiness for burned patients or extensive open injuries. These dressings are made with 24-ply Cellucotton with fine-mesh gauze attached to one side. They are made in two sizes: one suitable for small burns or burns of the hand and forearm, the other sufficiently large for either the posterior or anterior trunk, or to encase the entire lower extremity of an adult. These stored sterile dressings can be taken out quickly and applied rapidly. They are useful not only for emergency local care of the wound but also in later care if skin grafting is required. The dressings are of sufficient thickness to allow uniform compression to the open wound.

The petrolatum-impregnated gauze is used only to permit ease of removal later; it is merely a mechanical agent and has no healing power. At the time of removal the gauze should be gently lifted away to minimize tearing of the new epithelial layers.

The dressings are usually not disturbed for from five to eight days unless there is indication that they should be restored.

The initially applied burn dressing may be useful in most areas except the face and the perineum. These regions are usually left exposed.

One of the rediscovered methods of local care is that which is termed "exposure." This method relies on exposing the burned wound to the atmosphere and its value lies in drying of the wound which reduces the possibility of infection. It eliminates the necessity of dressings but, in order to be successful, excellent nursing care and a thorough understanding and ability to cooperate on the part of the patient are required. The dry crusts are allowed to remain in place until epithelialization has occurred, permitting the crusts to be lifted away.

Exposure of a burned area is initially pain

vitamin supplements should be given in the following amounts: vitamin C 1000 to 1500 mg., thiamine, 50 mg., riboflavin 50 mg., and nicotinamide 500 mg.

During the phase following burn shock and until the wound has healed, whole blood transfusions are often required. It is essential to maintain the hemoglobin and the total blood protein at a high level to combat infection and to facilitate normal epithelialization. As a good working basis the total proteins should be kept above 6 gm. per 100 cc., but it must be cautioned that any estimation of the circulating blood protein is no criterion of the body stores of protein which can be greatly depleted. Clinically it is of value to keep regular recordings of the patient's weight for this is a remarkably accurate guide to his progress. If this is not practical, there should be frequent checks by the laboratory of the hemoglobin red cell and total protein values. If the blood levels and the patient's weight can be maintained, healing is greatly facilitated.

Infection. Infection can produce serious complications. Every burned wound is contaminated and when there is a break in the integrity of the epithelium infection may become invasive and be disastrous.

All of the factors which are necessary for bacterial growth are present in a burned wound. There is contamination, dead tissue, body moisture and body heat. When further bacterial contamination is added to the burned wound the remaining useful epithelial structures which are necessary for healing of an incomplete thickness burn can be readily destroyed. Further the presence of infection can create a more profound wound defect. Infection elicits a greater response from the wound in that a much more rapid outpouring of protein and fluid will occur thereby compounding the problem of fluid loss. When a burn is first seen it is contaminated and it is necessary to prevent this wound, at all costs, from becoming infected. Therefore careful aseptic care must be practiced throughout, from the time of the initial local care until the wound is healed. Pyogenic bacteria are the most common invaders. Frequently there is a mixed variety of bacteria present in these wounds. Septicemia is a frequent cause of death in patients with extensive burns.

One of the most potent sources of cross-infection is that of dressings done carelessly without due precaution against contamination. When large wounds are exposed to the air in a ward or dressing room, there is

the ever present danger of bacteria being carried to the wound from the dust and draughts of air. It is imperative to take all precautions to avoid adding infection from this source.

Tetanus is not an uncommon complication in burns. Tetanus immunization must be given to all burned patients. If toxoid has been previously administered, a booster dose is all that is required. If doubt exists as to previous immunization with toxoid then tetanus antitoxin should be given.

The systemic use of antibiotics in burned patients is a much debated problem. Too frequently the antibiotics are administered routinely when they are not necessary. Unless there has been poor first aid, some delay in the initial care or if the burn presents a complicating factor such as lung involvement or burns of the perineum, antibiotics should not be routinely administered. The use of proper antibiotics should be reserved either for definite complications or for short intervals as when burn eschar is removed or skin grafting is performed. Resistant infections due to *Staphylococcus aureus* coagulase positive microorganisms are being reported frequently and can reach epidemic proportions. This type of infection is mainly due to a breakdown of aseptic technique and a blind reliance upon antibiotics to prevent wound infection. Obviously a chronically infected burn demands antibiotic therapy. However this should not be instituted until a culture has been made and the sensitivity of the bacteria to the various antibiotics has been demonstrated.

The local application of antibiotics or antiseptics to an open wound has not proved to be the solution for the problem of infection. Too frequently one is prone to rely on a drug to clean the wound in preparation for surgery. This is not wise for it is well known that the routine local use of a drug greatly increases the resistance of the bacteria and the sensitivity to it.

Initial Treatment of the Burned Wound. The objective of the care to be given to the burned patient is the immediate cleansing and protection of the wound and the repair of the systemic alteration resulting from the burn shock state. Initially a burn is very difficult to assess accurately as to depth or extent. Therefore, the wound must be protected and attention directed to care of the burn shock. After a few days an accurate estimation of the extent and depth of the burn can be determined. The problem in burns is identical to that of any traumatic

wound, namely the conversion of the open contaminated wound into a clean closed wound as rapidly as is practical for the patient. In burns, skin grafting is often demanded in closure of the wound.

First aid for a burn can be very simple in civilian practice, for all that is required is to protect the wound carefully and to transfer the patient immediately to a hospital where facilities are available for complete care. The application of home remedies, such as ointment, butter and lard too often adds further delay and always adds unnecessary contamination. Simple protection by the use of the cleanest available material to exclude the air is the most practical type of first aid.

Immediately upon admission to the hospital the patient should be quickly assessed as to the type and, roughly, the extent of the burn. Many minor burns do not demand hospitalization nor will the patients exhibit shock, but, when the burned area is larger than the "flash burn" regions, in all probability hospitalization will be required and burn shock may be anticipated.

The initial care of the burn is performed without the use of anesthesia. For those with extensive burns anesthesia is contraindicated because of its deleterious effect on the accompanying burn shock.

Narcotic drugs are not routinely administered to the acutely burned patient. If pain is a factor a small dose of morphine may be given intravenously. In patients with burn shock, the subcutaneous injection of morphine is dangerous and useless. In these patients the drug cannot be absorbed until the circulatory deficit has been corrected. If repeated doses have been given morphine poisoning can occur with the return of circulatory efficiency. Restlessness is a symptom of severe burn shock and is not an indication for a narcotic. Pain can be greatly diminished if the local care is undertaken in a room which is moderately warm and where draughts of air can be avoided.

Under aseptic conditions the burned site and the area well beyond it in all directions are gently cleansed with large pledgets of sterile cotton that have been dipped in a mixture of bland soap and warm sterile water. The wound is carefully flushed and all obvious blebs and loose bits of skin are removed. The procedure can be done carefully and yet rapidly. It is remarkably painless if gentleness is practiced and sterile water at body temperature is used for washing and flushing.

A single layer only of sterile fine-mesh

gauze is placed over the wound and the entire area is then covered with a previously prepared dressing. The fine-mesh gauze may be impregnated with a light coating of petrolatum prior to its sterilization. This is held in place by some form of bandage which produces a uniform compression dressing. The compression dressing must not be tight and must have a definite amount of resiliency. If the burn involves an extremity immobilization is secured by applying a splint. When the wound is covered and air is excluded there is almost complete absence of pain.

Every hospital or first aid station should be equipped with previously prepared sterile dressings always in readiness for burned patients or extensive open injuries. These dressings are made with 24 ply Cellucotton with fine-mesh gauze attached to one side. They are made in two sizes, one suitable for small burns or burns of the hand and forearm, the other sufficiently large for either the posterior or anterior trunk or to encase the entire lower extremity of an adult. These stored sterile dressings can be taken out quickly and applied rapidly. They are useful not only for emergency local care of the wound but also in later care if skin grafting is required. The dressings are of sufficient thickness to allow uniform compression to the open wound.

The petrolatum impregnated gauze is used only to permit ease of removal later. It is merely a mechanical agent and has no healing power. At the time of removal the gauze should be gently lifted away to minimize tearing of the new epithelial layers.

The dressings are usually not disturbed for from five to eight days unless there is indication that they should be restored.

The initially applied burn dressing may be useful in most areas except the face and the perineum. These regions are usually left exposed.

One of the rediscovered methods of local care is that which is termed "exposure." This method relies on exposing the burned wound to the atmosphere and its value lies in drying of the wound which reduces the possibility of infection. It eliminates the necessity of dressings but, in order to be successful, excellent nursing care and a thorough understanding and ability to cooperate on the part of the patient are required. The dry crusts are allowed to remain in place until epithelialization has occurred, permitting the crusts to be lifted away.

Exposure of a burned area is initially pain

ful and drugs are therefore, necessary to control this pain. Thus use of drugs may at times confuse the clinical picture of burn shock. Without excellent nursing care the exposure method is impractical for burned children and is difficult in all patients with an encircling burn of an extremity or of the trunk. However in encircling burns it can be used if the patient is turned frequently during the first few days to aid in the drying process. The method cannot be used for burns complicated by open injuries. Finally, it is of value only during the early period of treatment, for after five to eight days, when a whole thickness burn is evident, removal of the slough and a compression dressing over this burned region are demanded. Exposure has no place in the treatment of raw open wounds and, therefore, when such a wound is apparent this method must be abandoned. Briefly then, unless the burn is superficial, the exposure method is of value only during the first few days or the early phases of a burn.

Later Care of the Burned Wound. After five to eight days, it is entirely logical to inspect the burned wound. It is seldom necessary to use an anesthetic for the redressing procedure. Since at this time nothing will be gained by complete removal of the entire dressing, only the outer prepared dressing is removed and the initial fine mesh gauze is not disturbed. It is actually harmful to remove the gauze initially placed next to the wound, for this will only add trauma to the surface and may tear away the healing epithelium. It is possible to see the wound through the single layer of fine-mesh gauze and to determine the true extent and depth of the burn after five to eight days. At this time the burned wound is well demarcated, both as to extent and depth, and it is obvious if it is of third degree. The burn of incomplete thickness or second degree is dry and epithelization is beginning from the hair follicles and sweat glands. The whole thickness or third degree burn will show alterations of color and a depressed eschar which is anesthetic, beneath the surrounding skin level. The edges of the whole thickness eschar or slough may be moist. The state of the wound can be determined by careful inspection.

If the burn is superficial or partial thickness in character or if there are scattered small whole thickness burn areas, it is then wise and proper to simply re-apply a fresh sterile compression dressing over the undisturbed

initially placed single-gauze layer and to continue this protection of the wound until complete healing has occurred. If the character of the wound is estimated correctly, healing is accomplished within ten to fifteen days. However when there is whole thickness loss of skin the surgeon must decide in what manner to remove this slough and how best to convert the resulting open wound into a closed wound. The slough of a third degree burn may involve skin, subcutaneous tissue, muscle, fascia, tendon and bone but this in no way alters the principle or the necessity for removal of the dead mass. A whole thickness burn evokes a foreign body type of reaction and the host tries to sequestrate the dead tissue. Infection is not common five to eight days after injury in a well treated burn. The secretion emanating from the edges of the slough simply represents liquefaction of the debris.

Removal of the whole thickness burn slough should be accomplished early and may be performed in one of various methods.

Under the most scrupulous aseptic technique the wound is redressed and the dead material is cut away daily. This method is time consuming, the patient experiences considerable pain and daily dressings are required for maximal efficiency. There is an ever present danger of adding infection or establishing a cross infection by this technique. The method is satisfactory, however, in patients with burns of the face where the blood supply is very rich and sequestration occurs rapidly. It is possible to start dressings and removal of the slough of the face in five to eight days following the injury so as to have the open wound in readiness for grafting by the tenth to fourteenth day.

Chemicals such as pyruvic acid and acetic acid, have been used for removal of the burn slough. There are also various enzymatic drugs which can digest the dead tissues. These drugs and associated remedies are effective only in superficial deep burns. However when the burn slough involves fascia or deeper tissues these agents are not efficient and frequently do not penetrate to the undersurface of the eschar to aid in digesting the collagen fibers. The principal advantage of these agents is that they can be applied easily. However they are not always effective in accomplishing the desired result and often there is pain associated in their administration.

Surgical excision is an ideal method for removing the larger sloughs of tissue. It should never be used on the face or the neck where there is adequate blood supply which normally produces rapid sequestration. After five to eight days, when it has been proved that whole thickness burn is present and the patient's general condition is at a satisfactory level, in the operating room, with the patient under general anesthesia the areas surrounding the wound are gently cleansed and with sharp dissection the necrotic tissue is removed. This is not a "block dissection" of the mass. It is properly a surgical excision and only the obvious dead tissue is removed. The dissection follows not only the irregular contour of the burn edges but also the irregular depth of the wound. When the dissection is performed between five and eight days after injury the surgeon will follow a line of edema in which there is minimal bleeding. If the dissection is too deep bleeding will occur which signifies unnecessary removal of healthy tissue. All tissue must be saved when there is reasonable doubt of its involvement. Recently the removal of eschars has been facilitated by using the electric dermatome to remove the necrotic skin. This has to be done prior to the stage of extensive autolysis of the burned tissue. When the resulting open wound is extensive, then it is wisest to cover the wound with a large previously prepared sterile dressing and obtain compression over the wound. No ointment or drug is applied. The patient is returned to his bed and forty-eight hours later after the blood levels have been restored, he is returned to the operating room where skin grafting or skin dressing is performed.

When the wound is inspected forty-eight hours after surgical excision, there may be small areas which show that the entire burn slough has not been removed. This is not to be considered a fault, but rather that conservatism was used at the time of excision. It is entirely possible to remove these small areas of deeper slough at this time and to proceed with closure of the wound by skin grafting. In localized burns of the hand or of the foot, it is entirely possible to control adequately the oozing and hemorrhage and immediately to skin graft the wound and perform closure at the time of excision.

Regardless of the method used to dissect the whole thickness eschar it is imperative that the necrotic mass be removed before the wound becomes severely infected and

before the continued and increasing wound discharge depletes the patient's protein reserves to a dangerously low level. Procrastination in waiting for spontaneous separation of the slough or reliance on some particular drug to accomplish this jeopardizes the progress of the patient. Too often extensive burns appear to be in excellent condition in ten days in fair condition in twenty days and in critical condition in thirty days. This clinical picture is the result of the continuing progress of deterioration due to the loss of protein, weight loss and secondary anemia. Every effort should be directed to the conversion of the burned wounds into closed wounds between the second and third week, when at all practical for it is at this time that the patient has the ability to heal and to respond to such therapy.

Autogenous skin is essential for the permanent coverage of large full thickness burns. However in extensively burned patients homografting of skin may be a life-saving measure. Fresh cadaver homografts can be used to cover temporarily a large raw surface, particularly when the wound is clean and ready to receive skin grafts and when the available donor areas will not yield sufficient autografts to close more than a fraction of the open wound. The homografts will slough in about three weeks but halt the profuse loss of protein and blood from the large open surface during that time. Meanwhile, if the patient's donor areas heal without infection, these areas may be re-used to obtain more autografts. It is not uncommon to use the same donor area three or four times to obtain successive crops of skin during the course of treatment of the patient with extensive full thickness burns. Donor sites are comparable to partial thickness burns. If infection and irritation are avoided and thin grafts are taken, the donor site heals within two weeks.

In extensively burned patients certain regions should take precedence in the application of available autografts. The full thickness burns over joints, hands, feet, ears and the face should be covered with skin as soon as possible. In large burns on the trunk, thighs, arms and legs the skin grafts are often applied in postage stamp size. The grafts proliferate and frequently coalesce. For smaller burns in which donor areas are plentiful, the wound should be closed by applying wide strips of skin. The skin grafts are placed in a direction paralleling the natural skin creases particularly over joints.

If infection has occurred, the granulations become pale and exuberant. The patient's general condition is poor and is reflected in the appearance of the wound. To prepare such a wound for grafting demands aseptic care with moist daily dressings to encourage drainage of the wound. Cultures of these wounds should be taken to determine the exact type of the bacteria and the proper antibiotic to be administered. Before grafting, it is necessary to restore the patient's blood to a high normal level.

The discussion of chronic burns is an admission of failure to heal burns during their normal progress. No well treated burn should become chronic. It is simply delay and procrastination in allowing the patient's general condition to deteriorate and the wounds to become infected. Too frequently reliance upon the effectiveness of a locally applied drug and failure to have a program of care are the causes for burns coming into a chronic state.

Special Treatment. The treatment of burns in infants and elderly patients requires special care. The infant shows a marked shock state which can vary considerably from hour to hour. This is due to the smaller blood volume, which with even minimal dehydration can produce astounding symptoms. The principles of care for shock are identical to those for adults but the volume of fluid administration must be more carefully observed and the surgeon must be content with a urinary output in proportion to the patient's size. Skin grafting on infants and young children is not complicated except for the problem of the thinner donor skin. Fortunately children tolerate these operative procedures remarkably well. In the aged, the problem is one of being constantly aware of the potential of overloading the cardiovascular system during the shock state. At this time it is wise to keep these patients on the dry side. Constant careful observation of the urinary output and checking of the lung fields is mandatory. One should be content with a smaller but steady urinary output than might be desired in a younger healthier adult. In the elderly patient, 20 to 30 cc. of urine per hour should be considered satisfactory. Elderly burned patients often have organic disease renal, cardiac or hepatic, which must be treated concomitantly. Finally an elderly patient's nutrition may not be at a normal level and these patients need correction of any nutritional problems before surgery is attempted. Elderly persons

do not have reserves of fat and protein to draw upon for healing.

Burns of the face are common but fortunately are seldom as deep as those seen elsewhere on the body. At the time of injury the patient at all costs automatically attempts to protect the face. The face has an excellent blood supply and will rapidly sequesterate a whole thickness burn. Separation of the whole thickness slough of a facial burn starts about the fifth to eighth day following the injury and is accomplished by daily cutting away only the obvious slough followed by the application of sterile dressings. It is possible to prepare these wounds for closure by the tenth to fourteenth day following the injury as grafts take very readily on such an area.

Flame burns of the face should always be a warning that one should anticipate involvement of the upper respiratory tract. Inhalation of noxious fumes, hot air and gases can and does occur producing marked swelling and edema of the entire respiratory tract. Whenever severe respiratory damage is suspected, a tracheotomy is necessary to save life. The clinical symptoms of impending respiratory obstruction may be sparse. There is often moderate edema of the tongue but seldom is the voice greatly altered. The patient may frequently have a continuous dry hacking cough. Unfortunately respiratory obstruction is not revealed immediately and it is not until some time has elapsed, usually six to eight hours that it becomes apparent. Cyanosis, air hunger and rapid respirations are the signs of established respiratory difficulty. When these are present there should be no further procrastination an immediate tracheotomy is indicated. Serious burns of the face usually involve the neck and the physician is confronted with a tense swollen neck through which he must very quickly reach the trachea to perform the tracheotomy.

Burns of the hand are a common problem and the principles of care are the same as for burns elsewhere in the body. Initially the burn of the hand should be carefully cleansed. Rings should be removed, the fingers dressed individually and the entire hand placed on a splint to maintain the position of function to enable the surgeon to apply uniform compression to the entire involved part. The hand is inspected within five to eight days if there is frank whole thickness involvement of the skin or a third degree burn, it is advantageous to remove this whole thickness slough while the pa-

tient is under general anesthesia and with the use of a bloodless field obtained by means of the blood pressure cuff. Skin grafting can be done at this time if the wound is surgically clean and hemostasis is complete. If the burn is of such depth that the fingers are gangrenous they should be removed at the level of demarcation. The level of demarcation is not transverse to the long axis of the finger but instead is conical in shape: the deeper structures are preserved distally. Burns on children's hands occur more frequently on the palmar aspect while burns of adult hands are more common on the dorsum. However regardless of the anatomic site, rapid removal of whole thickness slough and coverage by skin dressing will often prevent small joints and tendons from becoming necrotic. This maneuver is done to salvage tendons and joints, later more satisfactory skin replacement may be accomplished after healing is complete.

Burns of the perineum present a difficult problem of management, particularly in young children and infants. These burns fortunately are most often of the incomplete thickness type, and are often the result of sitting in a bucket of boiling water. The use of a catheter may avoid contamination from urine during the first few days. After each bowel movement, the exposed area is carefully cleansed with soap and water. It is fortunate that most burns at this site heal readily and grafting is rarely necessary. There is a definite resistance of the skin in this area to fecal contamination, but when grafting is required the bowels are thoroughly evacuated.

If the patient is placed in the prone position the grafted area may be exposed. The exposed skin grafts usually take very well if careful attention is given to the removal of exudates with moistened cotton applicators.

READING REFERENCES

- Allen, H. S.: Local Treatment of the Whole Thickness Burn Surface. *S. Clin. North America* 18:123, 1918.
- Allen, H. S.: Management of Whole Thickness Burns with Involvement of Bones and Tendons. *Quart. Bull. Northwestern Univ. M. School* 22:115, 1948.
- Artz, C. P., and Reiss, E.: *The Treatment of Burns*. Philadelphia, W. B. Saunders Co., 1957.
- Artz, C. P., and Saroff H. S.: Modern Concepts in the Treatment of Burns. *J.A.M.A.* 159:111, 1955.
- Berkow, S. G.: Method of Estimating the Extensiveness of Lesions, Burns and Scalds—Based on Surface Area Proportions. *Arch. Surg.* 8:133, 1904.
- Brown, J. B., Fryer M. P., and Zaydon T. J.: A Skin Bank for Post Mortem Homografts. *Surg. Gynec. & Obst.* 101:101, 1955.
- Evans, E. L., and others: Fluid and Electrolyte Requirements in Severe Burns. *Ann. Surg.* 135:604, 1952.
- Mason, M. L., and Bell, J. L.: The Management of Burns. *M. Clin. North America* 40:1503, 1956.
- Neecker I. A., Jr., and Snyder W. H., Jr.: Dermatomic Debridement and Early Grafting of Extensive Third Degree Burns in Children. *Surg. Gynec. & Obst.* 103:527, 1956.
- Moyer, C. A.: Sociological Aspects of Trauma with Particular Reference to Thermal Injury. *Am. J. Surg.* 87:421, 1954.
- Symposium on Burns. Academy of Sciences, National Research Council, Washington, D.C., 1951.
- Wallace, A. B.: A Present (1957) Outlook on Burns. *J. Plast. & Reconst. Surg.* 21:243, 1958.
- Womack, N. A., comp. and ed.: *On Burns*. Springfield, Ill., Charles C Thomas, Publisher, 1953.

Local Cold Injury

By ROBERT B LEWIS M D

ROBERT B LEWIS an Illinoisan by birth was educated at the State University of Iowa. He trained in pathology and after joining the Regular Army Medical Corps, he has devoted his interest to this field. His investigative interests led him into the problems of aviation medicine and, in particular the damage produced by cold injury. Colonel Lewis is Deputy Chief of Pathology Branch, Allied Sciences Division, U S Army Biological Warfare Laboratories, Fort Detrick, Frederick, Maryland.

Predisposing Factors. The temperature of the body represents a balance between metabolic heat production and heat loss especially from the skin. Normally the central body temperature (core) representing that of the deep internal organs is constant at 37.5 C. The temperature of the skeletal muscles and skin (shell) may be one or two degrees cooler than that of the core particularly in the more peripheral portions of the extremities where the relatively large skin area invites heat loss in excess of the amount conveyed by the blood from the core and that resulting from local tissue metabolism. This difference in temperature between the central and peripheral parts of the body becomes progressively greater during continued exposure to cold and accounts largely for the observation that the fingers, toes, ears and nose are most frequently affected in cold injuries.

It is quite apparent that local cold injury will be most likely to occur under conditions which favor heat loss from the skin surface or inhibit heat supply from within. Conditions which decrease the internal heat supply include those which mechanically obstruct blood flow to the extremities such as constrictive clothing or other pressure-producing objects. Tight or stiff clothing is especially harmful if individuals must remain in cramped quarters with arms and legs flexed. Pressure in the popliteal spaces from the rungs of deck chairs was found to enhance cold injury among bomb shelter occupants during World War II in England.

General hypothermia during cold exposure increases the chances of local cold

injury by decreasing the amount of heat transferred to the extremities by the blood stream. Shock, accompanying injury operates in the same manner.

It has been stated that fatigue and malnutrition predispose to cold injury but the importance of these factors remains to be determined. It is conceivable that malnutrition, sufficiently severe to decrease caloric production appreciably might enhance the development of cold injury.

The most important predisposing factors have to do with increasing tissue heat loss. Wind blast was a prominent feature leading to the development of cold injuries in high altitude bomber crew members during the early part of World War II. On the ground, lesser cold breezes favor the development of cold injury. In either case the movement of cold air removes the warm layer of insulating air from around the exposed parts. When water or metal replaces air as the medium surrounding cold-exposed parts, cold injury is more apt to occur than when air is the medium since water or metal conducts heat better than does air. For this reason persons with wet feet develop injury at considerably higher temperatures than those exposed in cold dry air. Contact of the skin with cold metal can produce almost instantaneous freezing of tissue.

Environmental protection, including shelter and the quality and quantity of clothing, has a profound effect upon the development of cold injuries. Wet clothing is especially dangerous.

There is considerable variation in individual susceptibility to cold which can be

altered by acclimatization and training and is related to alterations in peripheral blood flow.

For reasons which are not clear tissues which have been injured by cold are there after particularly sensitive to low temperatures.

Some surveys in the military service suggest that Negroes are more susceptible to cold injury than Caucasians. However this impression may be erroneous because of differences in factors such as education, training and motivation rendering the same groups nonhomogeneous.

It has not been possible to correlate the presence or levels of blood cold agglutinin titers with the incidence or extent of cold injury.

Classification. It is customary to name cold injuries according to the physical conditions prevailing when the injuries are incurred. Thus, the term "trench foot" is applied to cold injuries occurring in personnel occupying trenches in cold wet climates and similar injuries developing in shipwrecked mariners, whose extremities have dangled in cold or cool water for hours or days are called "immersion leg, foot or hand." Also, "shelter foot" has been applied to cold injuries occurring in bomb shelters. The term "frostbite" has been reserved for cold injuries resulting from the actual freezing of tissues. It must not be construed that these terms represent different degrees of cold injury or even different pathologic processes.

It is desirable and feasible to classify cold injuries on the basis of severity regardless of the manner in which they are incurred, in much the same way as are burns. Such a classification includes four degrees of injury which are in the order of increasing severity as follows:

1. Numbness, swelling, and erythema, without vesiculation. Superficial desquamation in some cases.

2. Partial skin thickness vesiculation.

3. Involvement of entire thickness of skin and varying depths of subcutaneous tissue.

4. Involvement resulting in loss of the part.

Pathology. The pathologic changes resulting from freezing injuries (frostbite) and nonfreezing injuries (trench foot or immersion foot) are identical, although generally a greater volume of tissue is involved in the latter.

The abnormalities produced in the local blood vascular system and other exposed tissues are the same as observed in burn

injuries except that incineration of tissue may occur in the latter.

The fact that freezing and nonfreezing injuries are the same pathologically and that both may or may not result in gangrene brings out the point that it is not the attained tissue temperature that will determine the final injury but rather the product of temperature and exposure time. Of course the surrounding conducting medium, whether it be air, water or metal, will affect this product appreciably.

It has been shown experimentally by covering exposed parts with rubber boots during immersion that water merely acts as a thermal conductor in the production of immersion type injuries. Maceration of the skin, commonly observed in this type injury should not be considered as being due to cold but as something caused by the local effects of moisture.

The various fixed tissues do not show the same degree of susceptibility to cold injury. Nerves and striated muscle are highly susceptible; skin, fascia and connective tissue are quite resistant but not as resistant as compact bone or tendon. Blood vessels are highly susceptible to injury resulting in leakage of plasma into the surrounding tissue but it is important to know that they are relatively resistant to cold induced necrosis. This difference in tissue susceptibility has resulted in the observation in both human beings and experimental animals of muscle gangrene without necrosis of the overlying skin following cold exposure thus producing a closed lesion.

The great majority of cold injuries are of the nonfreezing type and are generally associated with wetness with the ambient temperatures several degrees above freezing. Immersion of extremities in the South Atlantic Ocean at temperatures considerably above freezing (60 to 70° F) for days or even weeks produced serious cold injuries, often with gangrene, during World War II. Actual solidification of tissue from freezing has been observed relatively rarely. Probably in most persons with so-called frostbite incurred during exposure to dry air at below freezing temperatures, actual freezing of the extremities has not occurred. Under such conditions of exposure, the body can defend against the cold quite effectively in spite of steep temperature gradients existing between the body and the surrounding atmosphere because air is a rather good thermal insulator. Prolonged exposure may thus be endured without resulting in tissue

solidification although sufficiently severe chilling of the parts does occur to produce damage.

Supercooling of tissues has been observed in both skin and muscle. By this process tissues are cooled considerably below their freezing temperatures (-2.2 to -2.5 C) without ice formation. Its significance is not known.

Intracellular or extracellular ice crystal formation apparently is not an important factor in the production of cold injuries. The freezing of tissues does not necessarily result in gangrene and cell death can occur from exposures to temperatures above freezing.

Blood vascular system. It has been determined that an ambient air temperature of 16° C (61° F) usually results in loss of skin heat, especially in those areas where the ratio of surface area to tissue mass is high, namely in fingers, toes ears and nose. The heat loss is controlled by changes in the cutaneous blood vessels as follows: a direct effect of the cold resulting in constriction of the vessels; a temporary generalized vasoconstriction initiated through central nervous system action and a persistent constriction of the vessels resulting from cooled venous blood acting on the central vasomotor centers. Arteries, arterioles, capillaries, venules and veins participate in these reactions. The purpose of these vascular responses is to conserve body heat, but by so doing the peripheral parts are made more liable to cold injury as the exposure conditions become more severe since obstruction of blood flow to a part enhances tissue cooling. This apparent paradox is a process of sacrificing a part for the good of the whole.

It has been observed that, following the drop in skin temperature of a part during cold exposure there may be periodic elevations of temperature of as much as 16° C caused by periods of vasodilatation. This mechanism, called "hunting," is an attempt to keep the peripheral tissues warm while at the same time trying to maintain the core temperature.

After thawing of frozen parts or warming of tissue after prolonged severe chilling, blood flow begins in the capillaries which become dilated. Stasis begins immediately and becomes complete in some ten minutes. After stasis has rendered the capillaries non-functional, blood flow occurs in arteriovenous anastomoses and in arteriolar venular capillaries. As stasis develops, fluid is

lost through the injured capillary walls and the stranded red blood cells become packed together in masses to form what has been called sludges. The edema, which is mostly subcutaneous, becomes maximum about six hours after warming and begins to subside after approximately twenty four hours. Before resorption is complete organization of the edema fluid tends to occur forming adhesions between the skin and the underlying muscle fascia.

The blood flow increases after warming of cold injured parts as determined by direct blood flow studies or indirectly by tissue temperature measurements, although such determinations do not inform us of the course the blood takes through the tissues.

The question of the role played by these vascular reactions in the production of cold injuries including gangrene cannot be answered with certainty. These same changes including the initial vasoconstriction, occur in burn injuries so it would seem that the same significance should be attached to the local blood vascular abnormalities in both types of thermal injury. They probably are of minor importance compared to the direct effects of cold on tissue cells, but certainly vasoconstriction during exposure may secondarily enhance the injury by restricting the heat supply to the parts.

It is significant that living tissue can be killed by cold exposure without benefit of a vascular supply as has been accomplished with tissue cultures.

The similarity of the vascular abnormalities as well as the identical histologic tissue changes and even the same clinical manifestations as are observed in both cold and heat injuries suggests that both have the same pathogenesis and are true thermal injuries.

After the acute stages have passed, arteries and arterioles may develop fibroblastic proliferation of the intima with narrowing of the lumen. Fibrous replacement of degenerated smooth muscle in vascular walls with subsequent contracture probably also occurs to decrease further the diameter of vessel openings.

In some cases, the chronic stage of cold injury is characterized by increased warmth of the affected parts even adjacent to gangrenous areas. In others, there are lowered tissue temperatures which have been assumed, but not proved, to be due to overactivity of the sympathetic nerves with persistent vasoconstriction. Further investigations will be necessary to determine

whether the diminished blood flow in these cases is functional or due to organic changes in blood vessel walls.

Thrombosis occurs after twenty four to thirty six hours provided the blood vessels are sufficiently injured to produce acute inflammation with or without necrosis of the walls. This thrombosis is a secondary phenomenon which occurs in other types of physical injury rather than being the cause of gangrene.

Skin Intracellular vacuoles can be observed in the cold injured epidermis microscopically and are evidence of edema. Necrotic areas of epidermis have been observed as early as two hours after freezing of skin in experimental animals. The gangrene begins in the superficial epithelial layers and may be confined here to produce superficial desquamation with survival of the deeper layers which in the end often become atrophic. The atrophy may either be accompanied by hyperkeratosis or a reduced number of epithelial cell layers. The papillae tend to become flattened. As the intensity of exposure increases, the resulting gangrene becomes deeper until all layers of the skin and subcutaneous tissue become involved. The gangrene is dry unless secondary infection occurs.

Nerve tissue Relatively mild cold exposures result in immediate functional disturbances of nerves characterized by sensory loss and muscle weakness or paralysis. This latter may be due to a combination of nerve and muscle injury. More severe exposures produce edema of nerve fibers granular degeneration of myelin sheaths and swelling or degeneration of axil cylinders.

The rapidity with which muscular paresis or paralysis occurs immediately following exposure in experimental animals can only be explained on the basis of direct cold effects on tissue cells. To be caused secondarily by vascular events such as constriction, dilatation with edema, stasis with sludging of red cells or thrombosis would require a lapse of some time.

Muscle Muscle shows several pathologic lesions, after exposure to either freezing or nonfreezing cold, which represent different degrees of injury. The least severe lesion is simple atrophy in which the fibers are reduced in size but maintain their form. Muscle atrophy has been a frequent and persistent sequela of World War II cold injuries.

A little more intense exposure results in slow muscle cell death within a few days.

The cells degenerate beyond simple atrophy so that they eventually waste away and die and are replaced with scar tissue. Often such dying fiber fragments show hypernucleation which apparently represents a futile attempt at cell reproduction. This type of slow cell death has been called "degenerative atrophy" in the older literature to distinguish it from simple atrophy but "slow necrosis" would seem a preferable term.

The most severe muscle change is coagulation necrosis and represents acute cell death. The fibers are swollen, homogeneous and, as a rule show loss of cross striations. In some instances the cross-striations are more prominent than is normal. This lesion can be observed as early as fifteen or thirty minutes after severe local cold exposure in experimental animals.

Observation of acute necrosis in so short a time does not support the vascular theory of cold induced gangrene since complete tourniquet induced ischemia must act for about three hours to produce like changes. Cold must produce metabolic cellular changes in muscle cells by direct action which in relatively mild exposures result in disturbed metabolism leading to atrophy and in more severe exposures lead to cell death either acutely (coagulation necrosis) or more slowly (slow necrosis) (Fig. 4).

These three lesions may be present in the same muscle when conditions of exposure are right and are layered from the cutaneous surface in the order of decreasing severity. Thus, coagulation necrosis is present at the surface with a layer of slow necrosis just beneath and still deeper is to be found simple atrophy. This arrangement is not surprising since the temperatures in an ex-



Figure 4 Tibialis anticus muscle from rabbit sacrificed one week after exposure of a hind leg to an alcohol bath at -38°C . for two minutes, forty seconds. Three degrees of injury are present which are layered from the cutaneous surface in the order of decreasing severity as follows (1) coagulation necrosis, (2) slow necrosis and (3) atrophy-hematuria, eosin, $\times 100$.

solidification although sufficiently severe chilling of the parts does occur to produce damage.

Supercooling of tissues has been observed in both skin and muscle. By this process tissues are cooled considerably below their freezing temperatures (-2.2 to -2.5°C) without ice formation. Its significance is not known.

Intracellular or extracellular ice crystal formation apparently is not an important factor in the production of cold injuries. The freezing of tissues does not necessarily result in gangrene and cell death can occur from exposures to temperatures above freezing.

Blood vascular system. It has been determined that an ambient air temperature of 16°C (61°F) usually results in loss of skin heat, especially in those areas where the ratio of surface area to tissue mass is high, namely in fingers, toes, ears and nose. The heat loss is controlled by changes in the cutaneous blood vessels as follows: a direct effect of the cold resulting in constriction of the vessels; a temporary generalized vasoconstriction initiated through central nervous system action and a persistent constriction of the vessels resulting from cooled venous blood acting on the central vasomotor centers. Arteries, arterioles, capillaries, venules and veins participate in these reactions. The purpose of these vascular responses is to conserve body heat, but by so doing the peripheral parts are made more liable to cold injury as the exposure conditions become more severe since obstruction of blood flow to a part enhances tissue cooling. This apparent paradox is a process of sacrificing a part for the good of the whole.

It has been observed that following the drop in skin temperature of a part during cold exposure, there may be periodic elevations of temperature of as much as 16°C caused by periods of vasodilatation. This mechanism, called "hunting," is an attempt to keep the peripheral tissues warm while at the same time trying to maintain the core temperature.

After thawing of frozen parts or warming of tissue after prolonged severe chilling, blood flow begins in the capillaries which become dilated. Stasis begins immediately and becomes complete in some ten minutes. After stasis has rendered the capillaries non-functional, blood flow occurs in arteriovenous anastomoses and in arteriolar-venular capillaries. As stasis develops, fluid is

lost through the injured capillary walls and the stranded red blood cells become packed together in masses to form what has been called sludges. The edema, which is mostly subcutaneous, becomes maximum about six hours after warming and begins to subside after approximately twenty-four hours. Before resorption is complete, organization of the edema fluid tends to occur forming adhesions between the skin and the underlying muscle fascia.

The blood flow increases after warming of cold injured parts as determined by direct blood flow studies or indirectly by tissue temperature measurements although such determinations do not inform us of the course the blood takes through the tissues.

The question of the role played by these vascular reactions in the production of cold injuries including gangrene, cannot be answered with certainty. These same changes, including the initial vasoconstriction, occur in burn injuries so it would seem that the same significance should be attached to the local blood vascular abnormalities in both types of thermal injury. They probably are of minor importance compared to the direct effects of cold on tissue cells but certainly vasoconstriction during exposure may secondarily enhance the injury by restricting the heat supply to the parts.

It is significant that living tissue can be killed by cold exposure without benefit of a vascular supply as has been accomplished with tissue cultures.

The similarity of the vascular abnormalities, as well as the identical histologic tissue changes and even the same clinical manifestations as are observed in both cold and heat injuries, suggests that both have the same pathogenesis and are true thermal injuries.

After the acute stages have passed, arteries and arterioles may develop fibroblastic proliferation of the intima with narrowing of the lumen. Fibrous replacement of degenerated smooth muscle in vascular walls with subsequent contracture probably also occurs to decrease further the diameter of vessel openings.

In some cases, the chronic stage of cold injury is characterized by increased warmth of the affected parts even adjacent to gangrenous areas. In others there are lowered tissue temperatures which have been assumed, but not proved, to be due to overactivity of the sympathetic nerves with persistent vasoconstriction. Further investigations will be necessary to determine

eventually become mummified. The final line of demarcation between living and dead tissue generally does not become sharply delineated for about a week to ten days.

In somewhat less severe injuries desquamation of epidermal casts of involved fingers or toes, including the nails, may occur or even shedding of practically the entire soles of the feet.

When large volumes of tissue have been involved, as in immersion type injuries, muscular paresis or even paralysis may ensue depending on the severity of exposure.

Some patients develop anhidrosis of the cold injured areas whereas others show hyperhidrosis with or without concomitant maceration of the skin.

The persistence of signs and symptoms and their severity depend on the degree of injury. Patients with mild cases recover completely whereas those having severe ones tend to develop disabling sequelae. Some of these latter patients show hyperhidrosis, low skin temperatures (accentuated by the evaporation of sweat) and cyanosis and others present manifestations of peripheral nerve involvement but the majority have a mixture of these two types.

Contrary to popular opinion a considerable number of patients with late cases show normal plethysmographic recordings and as warm or warmer skin temperatures in the more severely injured areas.

Those with the nerve type show tenderness over the metatarsal-phalangeal portion of the foot with aching or sharp shooting pains which radiate toward the ankle. Pain on weight bearing areas of lower extremities makes walking extremely difficult. Many have areas of hypesthesia to pin prick and cotton, whereas others show hyperesthesia.

Persistent ulcers on the feet develop at times and heal with difficulty.

Hyperextension of the toes, especially of the great toe occurs in some cases in the chronic stage and represents contracture of fibrosed muscle.

Treatment. Therapy should be divided into that of the cold injuries per se and of the late sequelae. The former can be further divided into initial, or first-aid, and hospital treatment.

Rest of the parts is essential. Involved upper extremities should be placed in slings and persons with lower extremity involvement should be transported by litter. Walking on cold-injured feet only serves to aggravate the damage. Massage or rubbing of the cold-injured parts should be avoided.

The local application of snow or other means of cooling has no place in the treatment.

At the earliest practicable time efforts should be directed toward the return of the local tissue temperatures to 37.5° C. without overheating the tissue. This should be done even though the parts are thawed but still cool. Probably the most efficacious and safest way to accomplish this is by immersion of the cold injured part in warm water. The temperature of the water should not exceed 12° C. (104° F.) and the time of immersion should only be sufficiently long to return the tissue temperature to normal. Anesthesia may be required to control pain during the procedure.

After the tissue has been brought to normal temperature the injured parts should receive the same supportive care as they would following any other physical injury. Complete bed rest is important until the degree of damage can be ascertained and it should be continued if blebs or gangrene develop.

Strict asepsis must be adhered to in the handling and dressing of cold injuries. As a prophylaxis against secondary infection, penicillin (a minimum of 300,000 units daily) should be administered to all second, third or fourth degree injuries. When secondary infection becomes apparent, isolation of the offending organisms should be accomplished and sensitivity tests performed to determine the desirable antibiotics or sulfa drugs for use in each case. Infection with *Pseudomonas aeruginosa* is not uncommon and is very resistant to ordinary treatment but has been found to respond favorably to Sulfamylon or polymyxin therapy. Sulfamylon applied topically as an ointment at 5 per cent strength has proved effective.

Blebs should be left intact or if they break spontaneously careful superficial debridement is indicated.

Prophylactic tetanus antitoxin or a booster toxoid injection for those previously immunized, is indicated for patients with broken skin.

Slight elevation of the extremities assists the disappearance of edema, but too great elevation results in disturbed circulation and is to be avoided.

Provided the skin is intact without vesicles dressings are not necessary. In the more severe injuries, loose dry gauze dressings may be applied and a cradle to prevent contact with bed covers is advantageous. A room temperature of 70° to 78° F. is satis-

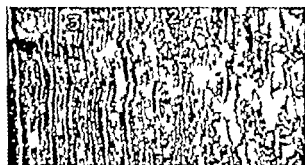


Figure 5 Tibialis anterior muscle from rabbit sacrificed one week after exposure of a hind leg to warm water at $+32^{\circ}\text{C}$. for six minutes. The same lesions, occupying the same relative positions, are present as in Figure 4; hematoxylin and eosin, $\times 87$

limb during cold exposure decrease with depth following an exponential curve. This same picture can be produced by controlled exposure to heat (Fig. 5)

Acute inflammatory exudate appears in the areas of muscular necrosis four to six hours after exposure. Fibroblastic replacement of dead muscle begins in about twenty-four hours and is complete in a week. Within thirty-six to forty-eight hours, myriads of macrophages invade the gangrenous area to remove the dead muscle cells and the acute inflammatory exudate recedes.

Bone cartilage and tendon. The marrow of bone is quite sensitive to cold. The cells disappear and a serous-gelatinous mass remains which eventually becomes replaced with connective tissue. Changes in the cortex vary from atrophy to actual necrosis and the former can be observed roentgenographically as demineralization or porosity. The endosteal bone trabeculae show thinning with corresponding enlargement of the marrow cavity and dilatation of the Haversian canal system gives rise to a spongy appearance.

Necrotic areas show loss of bone cells and demineralization the cartilage of joints may be affected with subsequent ankylosis.

Tendon is very resistant to cold injury. Connective tissue including fascia is relatively resistant to cold and shows a remarkable ability to proliferate in the healing phase provided some viable cells survive.

Symptoms and Signs. The seriousness of the signs and symptoms depends not only on the severity of the cold exposure but also on the volume of tissue involved. This latter aspect is very important in immersion type injuries. The clinical picture of so-called frost-bite and immersion type injuries is identical, provided the intensity of exposure (time-temperature product) and the amount

of tissue exposed are the same. This is rarely the case, since the majority of so-called frostbite injuries involve parts of fingers or toes whereas immersion type injuries affect much larger areas. Under such circumstances the signs and symptoms are qualitatively but not quantitatively similar. However reports are to be found in the medical literature pointing out identical clinical manifestations in the two types of cases. There is therefore no more reason to describe freezing and nonfreezing injuries separately than to differentiate between the local signs and symptoms of small and large area burns.

Exposure to local cold is as a rule not a painful experience in either freezing or nonfreezing cold injuries. The afflicted individual becomes aware of coldness of the exposed parts and at times has some stinging pain. These symptoms are followed by a feeling of local numbness and, finally loss of all sensations. In immersion type injuries, the involved extremities feel "dead" or "wooden" and swelling occurs because of injury to capillaries. In freezing injuries, the swelling is delayed until thawing has occurred. At first the color of the skin alternates between white and pink, reflecting the state of the cutaneous blood vessels, but after severe exposure remains pale. On walking, some subjects develop severe "pins and needles" sensations.

During and after warming, the signs and symptoms become more alarming. The parts become red or bluish and feel warm with bounding arterial pulse and are painful. Swelling becomes greater with or without bleb formation in the epidermis. The blebs usually contain clear straw-colored fluid which tends to coagulate because of its relatively high protein content. Hemorrhage may occur within the bullae. Injuries insufficiently severe to cause vesicles or bullae often show superficial desquamation of the epidermis within a few days. Those that eventually develop deep gangrene have a more severe clinical picture than those that escape necrosis.

In some instances the phase of hyperemia as manifested by local warmth and redness is transient and gives way to coldness and cyanosis. The latter sign is generally accentuated by dependency. Frequently these patients suffer from burning pain, especially at night.

Within one to three days parts destined to become necrotic become dark brown and finally black. The areas of dry gangrene

is likely to develop in severely injured areas. Fortunately, of all the patients treated by radiation only a few will require surgical treatment for the effects of the irradiation.

Causes of Irradiation Injuries. X rays, radium, radon and radioactive isotopes are the sources of radiation and excessive exposure to these is the cause of the local injury. Alpha particles do not penetrate deeply and therefore exert their greatest effect on the skin and mucous membranes. Beta and gamma rays have more penetrating power.

Beta ray burns of the skin of atomic research workers have been reported. Extensive radiation sloughs have occurred as the result of excessive exposure to gamma rays. Acute or chronic exposure to x rays however is still the greatest cause of local radiation injuries.

Overexposure beyond tissue tolerance may be caused by a single massive dose of irradiation or by the cumulative effect of frequently repeated small or adventitious exposures. Usually acute radiation burns are caused by a single massive overdosage such as might occur with a prolonged fluoroscopic procedure. Chronic irradiation injuries occur more commonly after lesser but frequently repeated radiation exposures in excess of tissue tolerance. Physicians, dentists and technicians may be exposed unwittingly to adventitious doses of radiation over a prolonged time and develop pathologic changes in the skin of their hands after a lapse of many years.

Often repeated dosages of irradiation for the treatment of some skin ailments, usually of the dermatitis group may be followed occasionally by irradiation damage requiring excision and skin grafting. The use of irradiation for the destruction of verrucae, superficial tumors, keloids, hair and scars has resulted in both acute and chronic irradiation damage to the skin. Growing bone and cartilage are extremely vulnerable to irradiation and the treatment of overlying skin lesions with improperly applied irradiation has caused injuries to the underlying bone and cartilage. Although it is not common to remove excess hair by x ray treatment, many of the patients who had this type of epilation twenty years ago are now showing the signs of chronic irradiation damage.

Radioactive cobalt "bombs" are being used increasingly for therapeutic irradiation of certain cancers which are beyond the scope of surgical therapy. Although arrest of

the cancer may result from such treatment extensive and progressive radiation damage to deeper tissues has been observed. The problem of closure of the defect after surgical excision of the damaged tissue is far from simple because vulnerable structures such as the pericardium, visceral pleura and neurovascular bundles in the axilla and neck may be exposed.

The administration of heavily filtered gamma particles may cause serious damage to deep structures. Viscera, bone, cartilage, muscle and subcutaneous tissue have been involved. The degree of damage is dependent on the radiosensitivity of the tissue, the dosage of the radiation and the amount of absorption by overlying tissue.

The first report of human damage from atomic radiation appeared in 1919. Although the hazards of working with atomic radiation are well known and extreme precautions are taken, accidents can and do occur. With the increasing employment of atomic radiation for military research and industrial uses, acute radiation burns have to be anticipated.

Pathology of Irradiation Injuries. Irradiation injuries may be either acute or chronic. The first signs of the acute irradiation reaction are indistinguishable from those of other acute inflammatory processes. Sequelae are dependent upon the severity of the dosage and the radiosensitivity of the tissue. Although the majority of acute irradiation burns gradually subside and the skin may resume a fairly normal appearance, the injury causes damage which is progressive even though the course may take many years to become clinically apparent.

The essential pathologic processes in delayed or late irradiation reaction include alterations in the architecture and cellular composition of the epidermis, obliterative vascular changes in the corium and subcutaneous tissues, necroses in the corium leading to stromal changes and partial or complete destruction of the skin appendages.

In the usual case, the epidermis exhibits atrophy with loss of rete pegs. Focal areas of hypertrophy of the epithelium characterized by acanthosis and hyperkeratosis are common. The melanin content of the basal layer of the skin is increased and accounts for the faint brown pigmentation observed commonly in chronic radiodermatitis. Dysplasia and dyskeratosis are prevalent in severe injuries and are the precursors to carcinoma.

The vascular changes probably rival in

factory. The local application of ointment is not indicated except as treatment for secondary infection.

Amputation of necrotic tissue is to be delayed until the final line of demarcation is stabilized, unless severe infection renders immediate amputation desirable. Constricting eschars should be bivalved.

It has generally been recommended, on theoretical grounds, that cold injured patients refrain from smoking, but experimental evidence does not support the idea that nicotine increases the extent of gangrene following cold exposure.

After the acute phase has passed and healing has begun, care must be taken to avoid or minimize contractures, joint stiffness or ankylosis. Adequate physiotherapy will do much to alleviate these disabling sequelae.

With extensive gangrene skin grafting of the exposed deep surfaces will shorten convalescence considerably and result in a better epithelized stump.

Except for rapid warming, specific procedures aimed at decreasing the extent of gangrene have given such equivocal results as to be of doubtful value. Thus vasodilating drugs, pressure dressings sympathetic block or extirpation and anticoagulants are not indicated.

The treatment of most of the disturbing sequelae is, as a rule unsatisfactory owing largely to the fact that their pathogenesis has not been determined. Excess sweating may be helped by practicing good foot hygiene and by the introduction of a 1 per cent solution of formalin into the skin by electric current six times daily. Hyperhidrosis, as well as edema, pain and chronic ulcers, has at times been reported to be

successfully treated with sympathetic block or extirpation, but the numerous failures reported suggest that these procedures are of doubtful value or at best should be used only in very carefully selected cases.

Severe pain in the balls of the feet on weight bearing may be alleviated by orthopedic appliances to the shoes such as the placing of thick leather or rubber in the instep to act as weight bearing areas.

READING REFERENCES

- Brownrigg, C. M. Frostbite Classification and Treatment. *Am J Surg.* 67:370 1943.
- Crimmon, J. M., and Fuhrman, F. A.: Studies on Gangrene Following Cold Injury: VI. Capillary Blood Flow after Cold Injury: The Effect of Rapid Warming and Sympathetic Block. *J. Clin. Invest.* 28:468, 1947.
- Davis, L., Scarrif, J. E., Rogers, N., and Dickinson, M. High Altitude Frostbite Surg. *Gynec. & Obst.* 77:561 1943.
- Finerman, J. C. and Shumacker H. B., Jr.: Studies in Experimental Frostbite V. Further Evaluation of Early Treatment. *Surg. Gynec. & Obst.* 90:430 1950.
- Lewis, R. B. Pathogenesis of Muscle Necrosis Due to Local Cold Injury. *Am. J. M. Sc.* 222:300 1951.
- Lewis, R. B. and Moen, P. W. Further Studies on the Pathogenesis of Cold-Induced Muscle Necrosis. *Surg. Gynec. & Obst.* 95:543, 1952.
- Pichotka, J. and Lewis, R. B. Effect of Rapid and Prolonged Rewarming on Local Cold Injury. *Armed Forces M. J.* 2:1293, 1951.
- Pichotka, J., Lewis, R. B., and Freytag, E.: Sequence of Increasing Local Cold Injury. *Texas Rep. Biol. & Med.* 9:813, 1951.
- Rosenfeld, L., Langohr, J. L., Owen, C. R., and Cope, A. Correlation of the Blood and Lymph to Frostbite and Influence of Therapeutic Cold and Warmth. *Arch. Surg.* 59:1045, 1949.
- Ungley, C. C. and Blackwood, W.: Peripheral Vaso-neuropathy after Chilling "Immersion Foot and Immersion Hand. *Lancet* 2:447 1942.

Irradiation Injuries

By JOHN L. BELL, M.D.

Tissues subjected to excessive irradiation, whether for diagnostic or therapeutic purposes may develop pathologic changes which require surgical treatment. The sequelae may appear early, delayed or many years following exposures to radiation en-

ergy. Irradiation damage to the skin is more common than injury to deeper structures such as bone cartilage, muscle and tendon. The clinical and pathologic courses following irradiation injury are progressive and, if the patient lives long enough, carcinoma

gentleness aseptic surgical care and the avoidance of irritating topical medicaments. Further irradiation or ultraviolet therapy is contraindicated because of the danger of aggravation of the injury. Because acute irradiation burns are painful the application of bland soothing medicaments may be of value but local anesthetics are usually not effective. In some instances, sedation and hospitalization may be necessary to help control the severe pain accompanying acute burns. Exclusion of air from the burned surfaces is of definite value in alleviating pain. In burns of the hands and feet immobilization of the injured part in a functional position and elevation are advocated to aid in the process of repair and the control of pain.

If a single cauterizing dose of radiation has caused a fairly well delimited burn which does not appear to have a chance of healing spontaneously the area may be excised and a free skin graft applied. One can not always be certain of the depth of the injury however and the take of the skin graft may be jeopardized by inadequate excision of the burn. Excision and closure by suturing are often fraught with breakdown of the wounds.

Following adequate excision sliding pedicle flaps from adjacent healthy tissue or the application of pedicle flaps from distant areas, may be necessary to cover defects which expose bone tendon and joint surfaces.

Treatment of Chronic Irradiation Burns. The surgical treatment of chronic irradiation injuries should be instituted before malignant changes, ulceration and intractable pain occur. It is unfortunate that many patients do not seek surgical care before these changes are prevalent. Surgical therapy for chronic irradiation burns is too often necessary because of malignant changes. The combination of atrophy telangiectases and keratoses is a forerunning sign of the inevitable development of carcinoma. Adequate excision and repair are the essentials of the proper surgical management for chronic irradiation burns.

Chronic ulcerated areas are usually accompanied by anemia and hypoproteinemia. In chronic irradiation injuries, it is important to correct anemia and place patients on high caloric and protein vitamin intake before undertaking extensive surgical corrections.

Atrophy and faint telangiectasis may not require treatment if there is no progress in clinical or pathologic changes, but some pa-

tients will seek surgical treatment to improve appearance. In areas of abundant telangiectasis which are exposed to the possibilities of repeated trauma or to sunlight, early surgical excision is advisable because of the likelihood of detrimental pathologic changes. Surgical excision and repair should be performed while the areas are still in relatively good condition. Because ulceration is invariably accompanied by stubborn infection the problem of repair following excision of such areas is compounded.

The local application of various drugs has no lasting beneficial effect on the pathologic course of chronic irradiation burns. Further radiation from any source is contraindicated because of the danger of acceleration of the pathologic changes. It has been established that patients with chronic irradiation injuries do not tolerate strong exposures to either sunlight or ultraviolet. Carcinomas resulting from irradiation should not be treated by more radiation of any kind.

The surgeon confronted by an irradiation injury has a complex problem in that it is difficult to determine the severity of the injury by clinical appearance. The surface lesion is not indicative of the degree of deeper involvement in most cases. When excision is contemplated, the problems of repair should be anticipated. Although most superficial irradiation injuries may be surgically excised and repaired by the application of a free skin graft, excision of an apparently superficial lesion may leave exposed avascular structures which require more adequate coverage for restoration of function and appearance. When bone, tendon, viscera or cartilage is uncovered by the excision, a protecting layer of subcutaneous tissue is required in order to preserve function. Pedicle flaps of whole thickness skin and subcutaneous tissue are fashioned from adjacent healthy tissue or from distant areas to provide necessary coverage in these situations.

Deep necrosis of tissues occurs frequently after intensive radiation therapy for malignant conditions of the chest and neck. Surgical excision of the damaged tissues may be necessary to prevent erosion of large vessels or sloughing of viscera. Closure of the resulting defect may be difficult and should be anticipated prior to excision of the irradiation damage. Pedicle flaps which carry a permanent blood supply to the area may be required to sustain closure and provide adequate protection to the viscera and neurovascular structures.

importance those alterations in the epidermis. Obliterative endarteritis with resulting diminution in blood supply accounts in part for the marked fibrosis and degenerative changes in the stromal elements. Occlusive endophlebitis may be more severe than the arterial involvement and leads to back pressure with the development of telangiectases in the corium.

The early changes in the corium are those of any acute inflammatory process. As vascular changes progress, secondary degenerative changes occur in the stromal elements of the corium. Collagenous fibers become thickened, fibrosed and densely hyalinized. Alterations of elastic fibers are varied, depending on the chronicity and severity of the irradiation damage.

Partial or complete destruction of hair follicles and sebaceous glands takes place in most irradiation injuries. In the milder type the epilation may be temporary whereas in severe cases permanent alopecia may result. Sebaceous glands are equally or more sensitive than hair follicles to irradiation. Sweat glands have a considerable degree of resistance to radiation and are a good histologic index of the severity of the injury.

Marked dysplasia of epidermal elements should be considered as precancerous lesions. Many subjects show the presence of carcinoma *in situ*. When invasive carcinoma occurs it is usually of squamous cell type although basal cell carcinoma is not uncommon. Distant metastasis can occur with squamous cell carcinoma, but if the invasive process is superficial the extreme fibrosis in the subcutaneous tissue and corium usually holds the carcinomatous cells in abeyance. Wide excision is therefore required in such cases. Extensive local carcinomatous infiltration is commonly observed, especially with basal cell carcinomas. Necrosis of structures deeper than skin and subcutaneous tissue can be produced by the use of large quantities of heavily filtered gamma particles. Radionecrosis of bone especially the mandible, has occurred, accompanying the treatment of overlying soft tissue malignant disease. The condition called *radiacerosis* denotes the involvement of dense fibrotic changes in the underlying joint capsules, tendons and muscles without the presence of an overlying skin ulceration.

Clinical Course. Certain pathologic changes occur in the skin in any person subjected to irradiation which proceeds to an erythema reaction. As in thermal burns, the degree of the reaction is dependent

largely on the severity of the dosage and the tolerance of the tissue which is involved. In contrast to thermal injuries the depth and extent of the irradiation damage are not as apparent in the early stages following the exposure.

Usually erythema of the skin commences within one to two days after sufficient exposure to alpha particles. This subsides only to reappear in from one to three weeks accompanied by varying degrees of vesiculation, swelling, exudation and desquamation. Pain is an outstanding accompaniment to acute radiation burns.

The degrees to which the pathologic stages advance in the early stages following exposure are dependent on the severity of the injury. These inflammatory signs are characteristic of the acute reaction to single massive overexposures or closely repeated excessive exposures. The acute inflammatory phase may subside gradually but incomplete repair leaving the characteristic marks of skin atrophy and dryness, hyperkeratoses and telangiectases, is common. Changes in the pigmentation of the involved area frequently are observed.

In more severe reactions epilation occurs invariably and may be either temporary or permanent. If the destructive process is of sufficient severity necrosis and ulceration of the skin ensue. Acute irradiation ulcerations are extremely painful, indolent and often intractable. Persistent, virulent, localized infection is characteristic of irradiation ulcers.

The chronic burn of the skin is characterized by atrophy, telangiectasis and keratosis. In the majority of patients the scar following irradiation may be inconspicuous and cosmetically acceptable. Previously stable scars, however, have broken down and carcinoma has resulted. Among the factors leading to ulceration of an irradiation scar are slight mechanical trauma, the performance of surgical procedures in the scarred area and the use of further radiotherapy in scarred areas. In chronic repeated exposures to irradiation such as seen in physicians' hands or in patients who have been treated with small doses for acne and other benign skin conditions telangiectasis takes from three to ten years to appear. Keratosis occurs in ten to fifteen years and carcinoma may take as long as twenty years to appear.

Treatment of Acute Irradiation Burns. Early surgical intervention is seldom necessary or indicated in the management of the majority of acute irradiation burns. The essentials of treatment of the acute burn are

rays emitted by residual radioactivity or by fission products. The syndrome may occur as a result of any sort of atomic explosion—air burst, surface burst or underwater burst of either a fission (conventional A bomb) or a thermonuclear (H bomb) weapon or the uncontrolled excursion of a nuclear reactor. In the case of the initial radiation from an air burst or a surface burst, persons who are in the open unshielded by buildings or natural objects will constitute the most exposed group at a given distance. Those who are shielded by natural objects or who are in buildings made of brick, concrete or metal will be less exposed and those who are in tunnels, caves or air raid shelters will be the least exposed. It is evident from animal experiments that effective shielding of some portion of the body, such as the spleen, the head, the abdomen or limbs, may reduce the severity of the injury. Only those individuals who were certainly in the open, unshielded, or were in wooden buildings can be assumed to have received the maximum dose of initial nuclear radiation which could be expected at a given distance. It is proper to assume that all others have received less than the dose that is theoretically possible. It is apparent that it will not be possible to classify casualties solely on the basis of their distance from the explosion. The severity of the exposure to residual radioactivity and/or fission products will depend upon the degree of contamination of the area and the length of time an individual remains there. In most cases, the dose will not be known with any degree of certainty, although a maximum estimate may be available. In the case of area contamination, members of the group who remain indoors or in shelters will be less exposed than those who remain in the open throughout the time they are in the radioactive region.

On the basis of the study of the Japanese casualties during World War II and by analogy with other forms of trauma, radiation casualties can be divided into two classes: (1) those with uncomplicated radiation injury and (2) those having radiation injury complicated by burns and wounds. It is not known with certainty how important such complications as the latter may be but it appears that the effect may be considerable.

Symptoms of Whole Body Radiation Injury. The syndrome of radiation injury due to exposure of the whole body to initial nuclear radiations at the time of an atomic explosion occurs in three fairly distinct

forms. It seems quite likely that exposure to comparable amounts of radiation over a period of ninety six hours or less as in a radioactively contaminated area would cause comparable clinical findings.

Very severe radiation injury occurs when the dose of radiation is in excess of 600 r (or rep i.e. roentgen equivalent physical). In this form vomiting commences shortly after the exposure occurs and may persist for several days. Progressive malaise leading to prostration begins soon after exposure and these symptoms continue unabated until death. Diarrhea usually appears within a few days and persists. Fever of the continuous type appears within the first few days and increases progressively. Leukopenia is present from the first day and becomes progressively more severe so that terminally the total white count may be less than 100 cells per cu. mm. Hemorrhagic manifestations such as purpura, may appear shortly before death, but they do not appear to be responsible for the fatal outcome. Epilation may be noted during the second week. Without therapy death is inevitable and may occur at any time from the first day to the end of the second week.

Severe radiation injury occurs when the dose of radiation is of the order of $400 \text{ r} \pm$ about 200 r (or rep). In this form, vomiting commences shortly after the exposure occurs, but it seldom persists beyond the first day. A latent period follows during which the only symptom is increasing malaise. The end of the latent period occurs during the second or third week after exposure and is marked by the appearance of one or all of the following characteristic symptoms of radiation injury: epilation, purpura, other manifestations of the hemorrhagic state and painful inflammatory lesions of the mouth and throat. Concurrently a variety of conditions may develop such as diarrhea, infection and the breakdown of healing wounds. The patient rapidly becomes quite ill and has fever and prostration and a severe hemorrhagic state may occur. If death follows it is usually due to acute blood loss, septicemia or hemorrhage into a vital structure. Death may occur at any time between the third and the sixth week after exposure. Lymphopenia is evident from the outset, but the hematologic findings become most pronounced during the third to the fifth week and consist of severe leukopenia, thrombocytopenia and anemia, and prolonged bleeding and clotting time. As recovery occurs the hemorrhagic manifestations subside and spontaneous defer

READING REFERENCES

- Brown, J. B., and Fryer, M. P.: Report of the Surgical Repair in the First Group of Atomic Radiation Injuries. *Surg. Gynec. & Obst.* 103:1 1950.
- Brown, J. B., McDowell, F. and Fryer, M. P.: Surgical Treatment of Radiation Burns. *Surg. Gynec. & Obst.* 88:608, 1949.
- Knowlton, N. P., Jr., and others: Beta Ray Burns of the Human Skin. *J.A.M.A.* 141:239 1949.
- Mason, M. L.: Irradiation Injuries of the Hand. *Quart. Bull. Northwestern Univ. M. School* 25:51 1951.
- Mason, M. L.: Irradiation Dermatitis of the Hands. *Am. Surgeon* 17:1121 1951.
- Routledge, R. T.: The Surgical Problem of Local Post Irradiation Effects. *Brit. J. Plast. Surg.* 7:134, 1954.
- Teloh, H. S. Mason, M. L., and Wheelock, M. C.: A Histopathologic Study of Radiation Injuries of the Skin. *Surg. Gynec. & Obst.* 90:335, 1950.

Nuclear Radiation Injuries

By GEORGE V. LEROY, M.D.

GEORGE VEACH LEROY attended the University of Pittsburgh and the University of Chicago. Trained in internal medicine, he taught and pursued his research work at Northwestern University. He served in the Pacific Theatre of World War II and the circumstances peculiar to military medicine directed his energies to a study of the effects of the atomic bomb. He returned to the University of Chicago where he continues his interest in the effects of atomic radiation as Professor of Medicine.

The syndromes which result from accidental exposure to harmful amounts of nuclear radiations are properly designated as *radiation injuries*. It is convenient to classify such injuries into the following categories:

1. *Whole body radiation injury* (also called radiation sickness, and the acute radiation syndrome) due to exposure to large amounts of penetrating gamma and/or neutron radiation at the time of the explosion of a nuclear weapon. Comparable injury may be sustained as a result of accidents with nuclear reactors or following sufficient exposure to residual radioactivity or the fall out from a nuclear weapon.

2. *Surface radiation injury* due principally to exposure to beta rays from contact, or near contact, with radioactive material, such as fission products and induced radioactivity. Comparable injury may be sustained as a result of accidental overexposure to ordinary x rays or to the electron beams from particle accelerators.

3. *Internal radiation injury* resulting from the selective deposition, such as in bone or thyroid gland, of radioactive material inhaled or absorbed through the gastrointestinal tract or wounds. Comparable injury has resulted from the therapeutic administration

of radium salts and from the poorly supervised employment of radioactive material in industry.

The syndrome of whole body radiation injury should be clearly distinguished from irradiation sickness which is the term commonly applied to the symptom complex that occurs during therapeutic irradiation with x rays or gamma rays. Whole body radiation injury has been observed and studied after four laboratory accidents in the United States in the Japanese at Hiroshima and Nagasaki, and in American military personnel Marshall Islanders and Japanese fishermen exposed to radioactive debris which fell out after the field test of a nuclear device. In addition to these studies in human subjects, the syndrome has been produced experimentally in a number of species (monkeys, dogs, pigs, goats, burros, rabbits, rats, mice and bats) in attempts to define the pathogenesis and to develop rational, effective therapy.

The exposure which is responsible for whole body radiation injury may consist of high-energy gamma rays and neutrons emitted during the first few minutes after the atomic explosion or it may consist of exposure for several hours or days to gamma

the Japanese physicians that recovery was unlikely if the total leukocyte count was less than 500 per cu. mm. The approximate time-trend of the total leukocyte count in the three grades of injury are shown in Figure 6.

In the first few days after exposure the lymphocyte count has greater diagnostic value than the total leukocyte count. Lymphocytes start to decrease immediately after exposure. Pronounced lymphopenia appears within twenty-four hours. A total lymphocyte count less than 500 per cu. mm. forty-eight hours after exposure is presumptive evidence of severe radiation injury. A lymphocyte count in excess of 1500 per cu. mm. forty-eight hours after an atomic bombing indicates that radiation injury of the severe grade is unlikely.

With severe radiation injury the neutrophils decrease less rapidly than the lymphocytes. With doses of radiation which are uniformly fatal to experimental animals, the total granulocyte counts are less than 1000 per cu. mm. within a week. With lesser doses of radiation such low levels may be attained, but it takes much longer, perhaps ten to twenty days.

For the first three to four days following

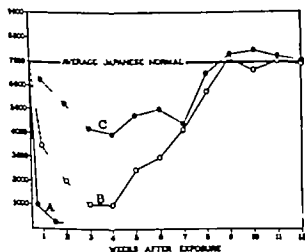


Figure 6 Time-trends of total leukocyte counts observed in Japanese patients after exposure to atomic bombs. A, Approximate average values in patients with very severe radiation injury, twenty-one of whom were studied in Hiroshima, and twelve in Nagasaki. B, Pooled data for 287 patients in Hiroshima with specific signs of severe radiation injury (i.e., epilation, purpura and oropharyngeal inflammation) who survived twenty-one days or longer. C, Pooled data for 554 patients in Nagasaki whose exposure was sufficient to cause moderately severe to mild radiation injury. The broken portions of the two latter curves represent extrapolated data based on a few Japanese subjects and experience with accidental exposure in laboratory accidents. The course of the leukocyte count in the most severely exposed Marshallese was similar to curve C.

severe radiation injury the thrombocyte count remains fairly stable. Beginning four to five days after exposure the thrombocyte count drops rapidly, attaining values close to zero during the second week. Thrombocytes may remain at this low level for two to four weeks. In the less severe grades of radiation injury the lowest values for the thrombocyte count occur in the fourth week.

In severe radiation injury anemia develops slowly. It may be two to three weeks before it is evident. The lowest red cell counts are observed four to five weeks after exposure. A rapidly decreasing blood hemoglobin, red blood cell count or hematocrit reading is a serious sign and suggests rapid blood loss.

Reticulocytes decrease in number promptly and are almost absent for three to four weeks. Any subsequent increase in reticulocytes is a good prognostic sign.

A prominent clinical symptom of radiation injury is the hemorrhagic state which begins between the second and fourth week. The severity of this hemorrhagic state is closely correlated with the level of thrombopenia. It is characterized by an increased capillary fragility, prolonged bleeding time and prolonged clotting time.

The diagnosis of the syndrome of whole body radiation injury and the estimation of its severity depend on a proper evaluation of three factors: (1) The probability that nuclear radiation sufficient to cause injury reached the patient. This will depend on a knowledge of the patient's location at the time of the incident and an estimate of the geographical area at risk. If personnel dosimeters are available the data obtained from them must be correlated not only with the foregoing, but also with the clinical symptoms. (2) The existence or the development of any or all of the typical symptoms of radiation injury. On the day of the incident it is proper to attach great significance to the presumptive diagnostic sign of vomiting. Particular attention should be directed to the tempo at which typical symptoms develop. Early appearance is indicative of greater severity of radiation injury. (3) The occurrence and the time-trend of leukopenia and lymphopenia are significant and should aid materially in the accurate evaluation of the severity of the injury. In conclusion, the diagnosis of radiation injury and the estimation of its severity constitute, essentially, a clinical problem and should be possible on the basis of a brief history, physical examination and a minimum of laboratory studies.

vescence takes place. Among the Japanese the mortality rate for patients with severe radiation injury varied from 25 per cent to more than 50 per cent. It is likely that aggressive therapy is capable of salvaging many patients with this degree of injury even though animal studies demonstrate that all cannot be saved. It is convenient to consider that severe radiation injury in man is equivalent to the type of injury which occurs in experimental animals given an LD₅₀ exposure.

Mild to moderately severe radiation injury occurs when the dose of radiation is in the range from 300 to 200 r to 75 to 50 r (or rep). In this form vomiting may occur once or twice on the day of exposure. During the next two or three weeks there may be no symptoms at all or at the most, moderate progressive malaise. During the third week or later any of the characteristic symptoms of radiation injury (epilation, purpura, inflammation of mouth and throat) may appear but it is distinctly unusual for all of them to be present or to be present in so severe a degree as occurs in the other forms. Constitutional symptoms and fever are not seen except in association with obvious complications. The changes in the blood picture are less profound and occur somewhat later in the course of the disease. Among the Japanese during World War II recovery was the rule for more than 90 per cent of the patients with this form of radiation injury. The deaths which occurred were the result of complications such as tuberculosis, chronic abscesses, bronchiectasis, refractory anemia and the like, and occurred as a rule later than the sixth week.

Whole body radiation injury of approximately this degree of severity occurred in some of the Marshall Islanders and the Japanese fishermen exposed to radioactive fallout following the detonation of a nuclear device at Bikini in 1954. In the case of the sixty-four residents of the island of Rongelap, the estimated dose was 175 r of gamma radiation. Constitutional symptoms and disability were minimal, even though skin lesions due to beta ray injury affected 90 per cent of this group. There were no fatalities among the Marshallese.

In general, patients with this form of radiation injury require no therapy except for well defined and obvious complications. Since in any situation in which atomic weapons are used, the mild or moderately severe cases of radiation injury will far outnumber the severe ones, it is important that

these be diagnosed correctly so that they will not monopolize the medical facilities.

Diagnosis of Whole Body Radiation Injury The diagnosis of whole body radiation injury should be entertained for all individuals who were within the area of destruction or fall-out of a nuclear weapon or who were in the vicinity of a reactor explosion. Under most circumstances, it is probable that military or civil defense authorities should be able to define a zone of potential injury beyond which there is small probability of the occurrence of significant radiation injury. In the case of accidents involving nuclear reactors, competent authorities should be able to assess the risk to individuals whose approximate position at the time of the incident is known. On the foregoing basis, it should be possible to separate survivors and casualties into two groups: those in whom exposure is possible and those in whom it is unlikely.

Among the group who may have been exposed to injurious amounts of nuclear radiation, the occurrence of vomiting on the day of the incident is most significant. In fact, this symptom may be considered presumptive evidence of radiation injury. It is of interest that many Japanese physicians attributed the vomiting on the day of the bombing to psychologic causes, but subsequent analysis of the clinical data demonstrated that the relation between this symptom and exposure was indubitable. Vomiting on the day of the incident is an important factor in evaluation and in triage, since it occurs soon after exposure, and is not likely to be overlooked by the patient. Except for patients with the very severe forms of whole body radiation injury, no reliable clinical symptoms are evident until the second to third week when epilation, purpura, and oropharyngeal inflammation make their appearance. In general, the earlier the occurrence of these specific symptoms the severer the radiation injury.

There is no single laboratory procedure which is, by itself, diagnostic for radiation injury. Exposure to harmful amounts of any sort of nuclear radiation produces leukopenia (lymphopenia and granulopenia), thrombocytopenia and anemia.

Leukopenia invariably follows whole body radiation injury. In the very severe type, the leukocyte count falls to less than 1000 per cu. mm. within the first week. With less severe degrees of radiation injury the lowest white cell counts are seen two to seven weeks after exposure. It was the opinion of

the Japanese physicians that recovery was unlikely if the total leukocyte count was less than 500 per cu. mm. The approximate time-trend of the total leukocyte count in the three grades of injury are shown in Figure 6.

In the first few days after exposure the lymphocyte count has greater diagnostic value than the total leukocyte count. Lymphocytes start to decrease immediately after exposure. Pronounced lymphopenia appears within twenty-four hours. A total lymphocyte count less than 800 per cu. mm. forty-eight hours after exposure is presumptive evidence of severe radiation injury. A lymphocyte count in excess of 1500 per cu. mm. forty-eight hours after an atomic bombing indicates that radiation injury of the severe grade is unlikely.

With severe radiation injury the neutrophils decrease less rapidly than the lymphocytes. With doses of radiation which are uniformly fatal to experimental animals, the total granulocyte counts are less than 1000 per cu. mm. within a week. With lesser doses of radiation such low levels may be attained, but it takes much longer—perhaps ten to twenty days.

For the first three to four days following

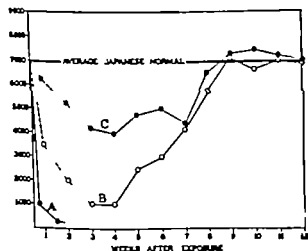


Figure 6 Time-trends of total leukocyte counts observed in Japanese patients after exposure to atomic bombs. A, Approximate average values in patients with very severe radiation injury (twenty-one of whom were studied in Hiroshima, and twelve in Nagasaki). B, Pooled data for 287 patients in Hiroshima with specific signs of severe radiation injury (i.e., epilation, purpura and oropharyngeal inflammation) who survived twenty-one days or longer. C, Pooled data for 554 patients in Nagasaki whose exposure was sufficient to cause moderately severe to mild radiation injury. The broken portions of the two latter curves represent extrapolated data based on a few Japanese subjects and experience with accidental exposure in laboratory accidents. The course of the leukocyte count in the most severely exposed Marshallese was similar to curve C.

severe radiation injury the thrombocyte count remains fairly stable. Beginning four to five days after exposure the thrombocyte count drops rapidly, attaining values close to zero during the second week. Thrombocytes may remain at this low level for two to four weeks. In the less severe grades of radiation injury the lowest values for the thrombocyte count occur in the fourth week.

In severe radiation injury anemia develops slowly. It may be two to three weeks before it is evident. The lowest red cell counts are observed four to five weeks after exposure. A rapidly decreasing blood hemoglobin, red blood cell count or hematocrit reading is a serious sign and suggests rapid blood loss.

Reticulocytes decrease in number promptly and are almost absent for three to four weeks. Any subsequent increase in reticulocytes is a good prognostic sign.

A prominent clinical symptom of radiation injury is the hemorrhagic state which begins between the second and fourth week. The severity of this hemorrhagic state is closely correlated with the level of thrombopenia. It is characterized by an increased capillary fragility, prolonged bleeding time and prolonged clotting time.

The diagnosis of the syndrome of whole body radiation injury and the estimation of its severity depend on a proper evaluation of three factors: (1) The probability that nuclear radiation sufficient to cause injury reached the patient. This will depend on a knowledge of the patient's location at the time of the incident and an estimate of the geographical area at risk. If personnel dosimeters are available, the data obtained from them must be correlated not only with the foregoing, but also with the clinical symptoms. (2) The existence or the development of any or all of the typical symptoms of radiation injury. On the day of the incident it is proper to attach great significance to the presumptive diagnostic sign of vomiting. Particular attention should be directed to the tempo at which typical symptoms develop. Early appearance is indicative of greater severity of radiation injury. (3) The occurrence and the time trend of leukopenia and lymphopenia are significant and should aid materially in the accurate evaluation of the severity of the injury. In conclusion, the diagnosis of radiation injury and the estimation of its severity constitute, essentially, a clinical problem and should be possible on the basis of a brief history, physical examination and a minimum of laboratory studies.

vescence takes place. Among the Japanese the mortality rate for patients with severe radiation injury varied from 25 per cent to more than 50 per cent. It is likely that aggressive therapy is capable of salvaging many patients with this degree of injury even though animal studies demonstrate that all cannot be saved. It is convenient to consider that *severe radiation injury* in man is equivalent to the type of injury which occurs in experimental animals given an LD₅₀ exposure.

Mild to moderately severe radiation injury occurs when the dose of radiation is in the range from 300 to 200 r to 75 to 50 r (or rep). In this form vomiting may occur once or twice on the day of exposure. During the next two or three weeks there may be no symptoms at all or at the most, moderate progressive malaise. During the third week or later any of the characteristic symptoms of radiation injury (epilation, purpura, inflammation of mouth and throat) may appear but it is distinctly unusual for all of them to be present or to be present in so severe a degree as occurs in the other forms. Constitutional symptoms and fever are not seen, except in association with obvious complications. The changes in the blood picture are less profound and occur somewhat later in the course of the disease. Among the Japanese during World War II recovery was the rule for more than 80 per cent of the patients with this form of radiation injury. The deaths which occurred were the result of complications such as tuberculosis, chronic abscesses, bronchiectasis, refractory anemia and the like, and occurred as a rule later than the sixth week.

Whole body radiation injury of approximately this degree of severity occurred in some of the Marshall Islanders and the Japanese fishermen exposed to radioactive fallout following the detonation of a nuclear device at Bikini in 1954. In the case of the sixty-four residents of the island of Rongelap the estimated dose was 175 r of gamma radiation. Constitutional symptoms and disability were minimal, even though skin lesions due to beta ray injury affected 80 per cent of this group. There were no fatalities among the Marshallese.

In general, patients with this form of radiation injury require no therapy except for well defined and obvious complications. Since, in any situation in which atomic weapons are used, the mild or moderately severe cases of radiation injury will far outnumber the severe ones, it is important that

these be diagnosed correctly so that they will not monopolize the medical facilities.

Diagnosis of Whole Body Radiation Injury The diagnosis of whole body radiation injury should be entertained for all individuals who were within the area of destruction or fall-out of a nuclear weapon or who were in the vicinity of a reactor explosion. Under most circumstances it is probable that military or civil defense authorities should be able to define a zone of potential injury beyond which there is small probability of the occurrence of significant radiation injury. In the case of accidents involving nuclear reactors competent authorities should be able to assess the risk to individuals whose approximate position at the time of the incident is known. On the foregoing basis it should be possible to separate survivors and casualties into two groups: those in whom exposure is possible and those in whom it is unlikely.

Among the group who may have been exposed to injurious amounts of nuclear radiation, the occurrence of vomiting on the day of the incident is most significant. In fact, this symptom may be considered presumptive evidence of radiation injury. It is of interest that many Japanese physicians attributed the vomiting on the day of the bombing to psychologic causes, but subsequent analysis of the clinical data demonstrated that the relation between this symptom and exposure was indubitable. Vomiting on the day of the incident is an important factor in evaluation and in triage, since it occurs soon after exposure, and is not likely to be overlooked by the patient. Except for patients with the very severe forms of whole body radiation injury no reliable clinical symptoms are evident until the second to third week when epilation, purpura, and oropharyngeal inflammation make their appearance. In general, the earlier the occurrence of these specific symptoms, the severer the radiation injury.

There is no single laboratory procedure which is by itself, diagnostic for radiation injury. Exposure to harmful amounts of any sort of nuclear radiation produces leukopenia (lymphopenia and granulopenia), thrombocytopenia and anemia.

Leukopenia invariably follows whole body radiation injury. In the very severe type the leukocyte count falls to less than 1000 per cu mm. within the first week. With less severe degrees of radiation injury the lowest white cell counts are seen two to seven weeks after exposure. It was the opinion of

THE HEAD

The Scalp and the Skull

By DANIEL RUGE, M D

DANIEL RUGE is a Nebraskan by birth and a graduate of North Central College and Northwestern University Medical School. He is a product of the residency training program in Neurological Surgery of his alma mater and is an Attending Neurological Surgeon at Chicago Wesley Memorial Hospital. Dr. Ruge has had fundamental training in the field of pharmacology and his investigative interests are concerned with the effect upon gastric secretion of various drugs introduced into the ventricular and subarachnoid systems.

The advancement of the machine age has subjected the scalp to an increasing incidence of trauma. The scalp serves as a covering of the skull and is a frequent site of infections and tumors of varied types. Scalp injuries and infections may result in osteomyelitis of the skull which may be further complicated by intracranial septic processes of the meninges and brain. Various cysts have a predilection for the scalp and hematomas of particular layers of the scalp are frequently encountered.

The scalp consists of five layers: the skin, subcutaneous tissue, the galeal aponeurosis with the epicranial muscle, subaponeurotic connective tissue and the pericranium, or external periosteum of the skull.

The skin of the scalp is very thick. Fibrous septa traverse the underlying subcutaneous tissue to join the skin to the galeal aponeurosis. There are numerous sebaceous glands in the true skin which may become cystic. The fibrous septa give the subcutaneous tissue a toughness and form an inelastic layer.

As a result, the numerous blood vessels in this layer do not contract when divided and this is the reason for the considerable hemorrhage which results from injuries to the scalp. Bleeding may be controlled by passing sutures through the subcutaneous layer and the galeal aponeurosis or by heavy pressure. Suture of the aponeurosis is essential in closure of scalp wounds and incisions, particularly when there is a defect in the underlying skull.

HEMATOMAS

Hematoma is a circular extravasation of blood in the subcutaneous tissue around the site of impact. At the center the skin may become detached from the galeal aponeurosis and this will permit an effusion, around which is a zone of hemorrhagic infiltration in the subcutaneous tissue. The hematoma will present a soft compressible center surrounded by a hard peripheral subcutaneous layer. It may mimic a depressed fracture, but it should be recalled that in an adult a

Treatment of Whole Body Radiation Injury At present (1959) there are available no specific agents of proved therapeutic value either for prophylaxis or for treatment following exposure to nuclear radiation. Treatment, therefore, must be symptomatic. In spite of the indifferent results of experimental therapy in animals it is not proper to conclude that all efforts at treatment are a waste of time and material. Certain general principles which seem reasonable for the three grades of severity may be followed.

Very severe cases. Such patients will have received upward of 600 rcp of whole body radiation. There is no satisfactory method for treating patients who have received the certainly fatal doses which produce this syndrome. Nausea and vomiting appear in one or two hours, followed by prostration within the first twenty-four hours. The experience of the Japanese indicated that these patients were very sensitive to physical activity so that they should not be transported long distances. Palliation may be attempted by maintaining fluid balance and by adequate sedation. There does not appear to be any advantage in the use of antibiotics and blood transfusions merely on general principles.

Severe cases. In general, this includes patients in whom in the neighborhood of 400 r of whole body radiation was received. These persons need and profit most from good medical treatment. Although in the absence of trauma and burns they do not require active care in the first few days strenuous activities should probably not be permitted.

Therapy should be directed at restoration of water and electrolyte balance and maintenance of general nutrition, control of infection and treatment for hemorrhage and anemia. The diet should be bland and low in residue. Intravenous fluids and parenteral alimentation should be given in the usual manner when indicated and when feasible, in an effort to maintain good nutritional status and to restore water and electrolyte balance in the presence of severe gastrointestinal symptoms. Infection can be controlled, at least in part, by the use of antibiotics. There is no reason to begin antibiotic therapy sooner than forty-eight hours after exposure unless required by wounds, burns or infectious disease. When there is

little doubt that a victim has been exposed to several hundred roentgens it is wise if adequate supplies are available to start oral maintenance doses of an antibiotic by the third day.

Sulfonamides are probably not drugs of choice in radiation injury. However if antibiotics are not available, they would undoubtedly prove lifesaving in certain instances.

The only effective treatment for hemorrhage and anemia is replacement therapy by blood transfusion. It would seem desirable whenever possible, to maintain blood hemoglobin at a level of 10 gm or more per 100 cc.

Mild to moderately severe cases. In the absence of complicating burns or wounds patients with this grade of severity should not require treatment. It is desirable to limit their activity and maintain alert, expectant supervision of them. A firm policy with respect to this class of patient is imperative since they may well be the most numerous and the most demanding group of survivors. They should not be allowed to monopolize the medical services and they should not be given antibiotics or blood transfusions without very clear indications.

READING REFERENCES

- Dunham, C. R., Cronkite, E. P., LeRoy G. V., and Warren, S. Atomic Bomb Injury. *Radiation J.A.M.A.* 147:50, 1951.
- Hempelmann, L. H., Libco, H. and Hoffman, G. F. The Acute Radiation Syndrome: A Study of Nine Cases and a Review of the Problem. *Ann. Int. Med.* 36:281, 1951.
- LeRoy G. V. The Medical Sequelae of the Atomic Bomb Explosion. *J.A.M.A.* 134:1143, 1947.
- LeRoy G. V. Hematology of Atomic Bomb Casualties. *Arch. Int. Med.* 90:601, 1950.
- Medical Report of the Joint Commission for the Investigation of the Effects of the Atomic Bomb in Japan, 1946, Atomic Energy Commission Document A.M. 2837 now issued by the Technical Information Service, U.S. A.E.C., Oak Ridge, as "Medical Effects of Atomic Bombs," April, 1951.
- Oughterson, A. W. and Warren, S. Medical Effects of the Atomic Bomb in Japan. New York, McGraw-Hill, 1950.
- Some Effects of Ionizing Radiation on Human Beings, A Report on the Marshallese and Americans exposed to Radiation from Fallout and a Discussion of Radiation Injury in the Human Being. U.S. Atomic Energy Commission, July 1956 (T.I.D. 5358) U.S. Government Printing Office, Washington, D.C.

subcutaneous tissue. After the tumor has been present for some time, there may be irritation of the epidermis resulting in ulceration. Tumors of the scalp as a rule follow a benign course. Occasionally malignant degeneration takes place in which event either basal or squamous cell carcinoma may result.

Often these benign epithelial tumors are grouped together and in neglected cases they may become so numerous as to cover the entire scalp giving the appearance of a turban. They may become lobulated varying greatly in size. The large growths or those which become ulcerated require surgical excision. In all instances one must keep in mind that malignant degeneration can occur.

MALIGNANT NEOPLASMS OF THE SCALP

Malignant lesions of the skin are easily accessible for careful examination, early diagnosis and proper treatment. If proper therapy is to be administered, the patient should be seen when the lesions are small and the treatment given at a time when the disease can be eradicated.

It is helpful to differentiate between histologic types and the following classification is of clinical value: basal cell carcinoma, squamous cell carcinoma and adenocystic basal cell carcinoma.

Basal cell carcinomas are frequent. As a rule, there is a history of the lesion having been present for a period of months or years before it becomes active. They rarely result in regional or distant metastases but it is believed by many that squamous cell changes may develop and these then gain access to lymphatic spread.

Next in frequency are squamous cell carcinomas. They grow rapidly and are subject to regional and distant metastases.

Adenocystic basal cell carcinomas clinically behave in a manner less active than the squamous cell but more active than the basal cell. There is considerable cellular differentiation.

While it is granted that malignant lesions of the scalp are rare, it must be remembered that they do occur. For this reason, patients with papillomas or other scalp tumefactions are often advised to have them removed. Least chronic irritation, such as may result from combing or brushing the hair or pressure from a hat, may result in a malignant lesion.

Diseases involving the skull are for the most part similar to diseases involving other

osseous structures. Absorption of bone may result from general causes or from local pressure. Osteoclasts may be absent or they may dominate the process. Deposition of bone is on the whole dependent upon osteoblasts which line the deep layer of the periosteum, the endosteum and the haversian canals. Specific etiologic factors in aberration are often unknown. Different authorities may implicate infection, faulty metabolism or neoplasia in discussing the same disease.

CONGENITAL DISEASES AFFECTING THE SKULL

Craniolethalodysostosis is a congenital condition in which there is a deficient formation of the clavicles and imperfect ossification of the cranium. Heredity is a large factor in etiology. It occurs equally in both sexes. Because there are great variations from case to case it may be discovered at any age.

Röntgen ray examination reveals imperfect ossification of the membranous calvarium. The sutures often fail to close and the anterior fontanel may remain very large throughout life.

Craniosostenosis results from premature closure of one or more sutures making the skull too small for its contents. There is frequently an increase in the depth and number of the convolutional impressions. The shape of the skull will vary according to which sutures are involved in the premature closure.

Megalocephaly (hydrocephalus) implies a head which is unusually large; the condition may be either congenital or acquired. The vault is unusually large as compared to the facial bones. All the bones are thin and the sutures may or may not be separated. If there is a marked intracranial pressure prior to closure of the sutures, there will certainly be a separation of the sutures.

Platybasia a flattening of the base of the skull, is usually associated with a deformity of the foramen magnum. *Platybasia* may occur with basilar invagination, in which case the odontoid process projects above a line drawn from the hard palate to the posterior margin of the foramen magnum.

ACQUIRED AFFECTIONS OF THE SKULL OF UNKNOWN ORIGIN

Leontiasis ossea is hyperostosis of the skull, the etiology is obscure. It is considered by some to be neoplastic rather than dysplastic. The bones most often affected are the nasal, malar, maxilla, mandible and the

localized depressed fracture rarely occurs in the absence of an open wound. The infiltration is compressible and the hard edge of the swelling can be dispersed by pressure.

Subaponeurotic hematoma may result from rupture of subaponeurotic vessels. The hematoma may extend from the temporal crest and zygoma on one side to the temporal crest and zygoma on the other and from the upper eyelids in front to the superior nuchal line behind. Rarely does infection occur. When it does pus can spread in all directions and elevate the scalp. It may destroy the pericranium and cause necrosis of the calvarium. Thrombi may enter the dural sinuses via emissary veins. Incisions for evacuation of subaponeurotic abscesses are made parallel to the large vessels in the temporal area.

Subpericranial hematoma (cephalhematoma) is the most common form of hemorrhage in the young infant and the age incidence is believed to be due to the great vascularity of the pericranium in early childhood. In the fetal skull the pericranium, or external periosteum, lies in direct contact with the dura mater at the fontanels. When the fontanels are obliterated the pericranium becomes firmly attached to the skull at the suture lines.

A subpericranial effusion of blood limited by the suture lines may result following rupture of some of the blood vessels which run between the pericranium and the skull and is usually due to an injury during labor. Because of the deformity of the scalp it may be mistaken for a fracture of the skull. Like the other hematomas of scalp cephalhematoma is usually absorbed gradually and completely. The hematoma may ossify partially if absorption fails. In rare cases only the periphery ossifies and the center becomes cystic. Such a bony prominence may persist for years; it is symptomless and as a rule requires no treatment.

SCALP WOUNDS

Every scalp wound may be a penetrating wound with injury to the brain. The most innocent-appearing wound requires proper attention for it may be the avenue to intracranial surgery should signs of intracranial hemorrhage occur subsequently. No scalp surgery should be undertaken without proper cleansing and adequate shaving of a wide area of the scalp around the wound. Control of hemorrhage is achieved by placing hemostats closely together in the subcutaneous galeal layers rather than by atten-

tion to the many anastomosing blood vessels.

Débridement of the wound edges should be accurate and conservative. Meticulous closure of a scalp wound involves suture layer by layer of the galea as well as the skin. Sutures may be removed after several days because the rich blood supply makes for early healing. Drainage is seldom necessary and when used should be discontinued after twenty-four hours. If a defect in the scalp exists, there is recourse to sliding grafts or pedicle flaps and again suture of both the galea and skin is essential. Care must be taken to have a good blood supply into the base of the flap. If this is certain one can expect good healing from a large variety of scalp flaps. Split thickness skin is a feasible covering for denuded scalp.

EPITHELIAL CYSTS OF THE SCALP (WENS)

This type of cyst may be caused by occlusion of the orifice of the ducts of sweat glands, fat glands or hair follicles. The enlarging cyst flattens the secreting epithelium until it resembles squamous epithelium. The cystic substance consists of cast-off epithelium, fat and cholesterol crystals and may be solid, soft or liquid. The size is variable and frequently the cysts are multiple. They may undergo malignant degeneration forming either squamous or basal cell carcinomas. They are attached to the true skin but are freely movable over the underlying connective tissue. Complete excision is therefore facilitated and should be done not only for cosmetic reasons but to avoid possible malignant degeneration.

Excision is performed after sterile preparation of the skin. A sufficiently wide area may be anesthetized with local anesthesia. An elliptical incision including the area of attachment of the cyst to the skin is usually made. The cyst wall is removed, preferably without rupture. The incision in the scalp should be sutured in layers. Care must be exercised to leave no part of the cyst wall to avoid recurrence.

CYLINDROMAS

This group of tumors of the skin occurs on the head and neck but more frequently involves the scalp in which case they are referred to as turban tumors.

The tumor is well demarcated in the corium. It may be entirely separated from the epidermis by a connective tissue layer or it may be diffuse with strands extending out into the surrounding corium or the

pelvis. Lesions may appear later in the long bones. Spread is principally to the other bones. Metastasis to the internal organs is very rare.

The roentgen ray appearance is characteristic. The bone lesions are purely destructive but localized so that they appear as punched-out round areas in a number of bones. Roentgen irradiation may cause prompt disappearance of the lesions but rapid recurrence is common.

METASTATIC TUMORS

Secondary tumors of the skull are most likely to occur in carcinomas of the breast

prostate kidney (hypernephroma) and lung. The usual mode of infection is by the blood stream. The roentgen ray evidence shows that most of the tumors are purely osteolytic the destruction not being associated with new bone formation. Tumors arising from the prostate however are osteoblastic as well as osteolytic and there have been instances of osteoblastic tumors arising from breast metastases.

READING REFERENCE

Ward, C. E. and Hendrick, J. W. *Diagnosis and Treatment of Tumors of the Head and Neck*. Baltimore: Williams & Wilkins Company 1930.

The Eyes

By DERRICK T. VAIL, M.D.

DERRICK TILTON VAIL was born in Cincinnati and was educated at Yale and Harvard Universities. He chose to follow in the footsteps of his father, a distinguished ophthalmologist. He supplemented his training by work in India and Oxford. He was the Senior Consultant in Ophthalmology in the European Theatre of Operations in World War II and has held the Chair of Ophthalmology at the University of Cincinnati and Northwestern University.

From a general surgical viewpoint, the anatomy of the eye and its adnexa is relatively simple. However a good working knowledge of it is essential to the proper understanding of surgical conditions pertaining to these structures.

Unless the general surgeon is equipped with special ophthalmic training and is familiar with the use of the small and delicate instruments, needles and suture material required in operations upon the eye, the ocular surgical conditions which he may encounter had better be referred to an ophthalmic colleague. There may be occasions, however, when the referral of a patient with an ophthalmic lesion cannot be done and the surgeon must therefore do the best he can with the tools he has.

The eyeball is well protected in front by the rim of the bony orbit, lids, orbicularis oculi, extraocular muscles and behind by a cushion of muscle tissue, fat and blood vessels. The area of greatest vulnerability is on

the temporal side and slightly below the horizontal. The wink reflex is exceedingly fast and this forms a surprisingly good protection to the eyeball because the rapidly closed lids receive the brunt of flying foreign bodies and burns. In addition the lids are very rich in blood supply and thus resist infection, heal rapidly when lacerated or burned and, even when they appear gangrenous, often make an excellent recovery. This is important to know in order to avoid unnecessary loss of important tissue when débridement is considered.

The sinuous course and adequate length of the optic nerve, permitting wide rotations of the globe without stretching, also is on the side of the surgeon when the eyeball suffers a severe contusion. Indeed, patients were encountered during World War II in whom a blow to the lids and eyeball was strong enough to fracture the floor of the orbit by transmission of the force through these soft tissues, and yet the eyeball had

frontal and parietal bones. At first the changes are unilateral but may eventually involve the entire skull. Signs and symptoms depend upon which of the structures are compressed by the overgrowth.

Roentgen ray examination reveals irregular and mottled increased densities. The outer table is more affected than is the inner table.

Page's disease (osteitis deformans) is characterized by slowly spreading changes in one or more bones. Decalcification combines with hyperostosis. Heredity may be a factor. The majority of patients seen are between fifty and seventy years of age. Males are affected more frequently than females. The femur, tibia, lower spine and skull are common sites.

In the skull there is a thickening of the outer table and irregular increased density. The sutures are obliterated. The changes are more likely to be generalized than in leontiasis and are less likely to involve the maxilla.

DISEASES AFFECTING THE SKULL RESULTING FROM ABNORMAL METABOLISM

Hand-Schüller-Christian disease an uncommon condition, consists of craniohypophyseal localization of a lipoid granuloma of bone. There are multiple defects in the skull and other bones, exophthalmos and diabetes insipidus. There may or may not be other signs of pituitary dysfunction. It occurs typically in childhood, affects males more than females and there is no apparent hereditary influence.

Roentgen ray examination reveals a sharply outlined defect. As a rule there is no sclerosis in the periphery.

Letterer-Siwe disease (reticuloendotheliosis) is seen in infants and may be regarded as an acute form of Hand-Schüller-Christian disease. Lesions are found in the liver, spleen, glands, lungs, skin and skull. Radiographic appearance is similar to that in Hand-Schüller-Christian disease.

EOSINOPHILIC GRANULOMA

This is an inflammatory condition which is similar to lipoid granulomas but is rich in eosinophils and contains no lipoid elements. Males are affected five times more than females. The etiology is obscure. Lesions may be single or multiple. The bones most often affected are the ribs, skull, vertebrae, pelvis and humerus.

There are frequently no symptoms. Pain

is rarely severe. There may be a soft fluctuant swelling.

Roentgen examination reveals oval or circular translucent and cyst like punched-out and sharply defined areas.

The lesion is amenable to surgery. There is also value in radiotherapy.

OSTEOMYELITIS

Osteomyelitis is an inflammation of bone. In children the infection is usually hematogenous in origin. In adults the infection is usually from without and may result from spread from the sinuses or from a fracture. As the infection progresses the dead bone becomes separated from the living by the action of the osteoclasts and forms a sequestrum. When osteomyelitis appears in the skull or other membranous bones there is practically no reproduction as there may be in the long bones.

Treatment of osteomyelitis should include specific antibacterial medication with antibiotics or chemotherapeutic agents as well as surgical removal of sequestra and devitalized tissue. Open drainage of the soft tissue is essential to cure.

BENIGN TUMORS

Osteomas of the skull are compact outgrowths from the surface and are probably formed by the periosteal osteoblasts. It is proper to think of them as exostoses and not as true tumors. The most frequent site is in the frontoethmoidal or orbitoethmoidal region, but they may be found elsewhere. On roentgen examination there is a homogeneous density.

Osteochondromas are very similar to osteomas except that, as the name implies, they contain cartilaginous elements. They are apt to be pedunculated and may appear in the roentgenogram as masses of irregular density but with clearly defined outlines. They are not associated with increased vascularity and produce no local bone reaction.

MALIGNANT TUMORS

Malignant tumors of the skull may be either primary or secondary. There are three principle types of primary bone tumor: osteogenic sarcoma, Ewing's tumor and multiple myeloma. Only the latter which occurs in flat bones is commonly seen in the skull. Multiple myelomas occur in patients over forty years of age. A striking feature is the multiplicity of the lesions. The flat bones containing red marrow are first involved, i.e., sternum, ribs, vertebrae, skull and

retained in the orbit as far as possible and the lid is closed over the eye with a wire suture until a fracture of the bony wall of the orbit has occurred in the lower wall.

Wounds of the orbit are a rare cause of proptosis, the eye is usually in the orbit in such cases, the eyeball is usually in the orbit, or occasionally a very large fracture of the orbital wall may result if the eye is not removed and can be left alone. If infection occurs, treatment with an antibiotic with or without drainage of the orbit is usually effective.

Of the trauma is accompanied by hemorrhage within the orbit, subperiosteal or within the orbital tissues, sometimes enough to cause an alarming ocular proptosis. In this event, a firm pressure bandage should be applied and maintained until bleeding has ceased. Care must be taken that the lids are closed as far as possible and the cornea protected by an antibiotic ophthalmic ointment, preferably 10 per cent sodium sulfathiazole. Once there is no further bleeding, the pressure bandage can be removed and a protective transparent plastic shield can be placed over the eye.

Operative interference to drain the blood is most disappointing and introduces the serious danger of infection, although in alarming cases, when the vision is threatened because of extravagant proptosis lateral decompression of the orbit may be necessary. It is usually better to watch and wait, however. The orbital blood is slow to absorb but generally the proptosis has subsided by the end of three or four weeks.

In the absence of a perforating wound, but when the skull is injured, hemorrhage into the orbit is a sign of fracture of its bony walls. The most serious danger to vision is a fracture through the optic foramen. The optic nerve can be hopelessly injured in this event, either through actual laceration of the nerve by sharp bone fragments or most commonly as the result of compression due to hemorrhage in the meningeal sheaths of the optic nerve. While it seems obvious that immediate decompression of the orbit foramen in this event ought to result in saving vision, very few of such maneuvers have been successful.

Early reduction of the fractured wall or walls of the orbit should be performed. In many of these injuries of the more severe type, the general condition of the patient may seem not to warrant early reduction, but, even so unless there is cerebrospinal rhinorrhea, repositioning should be

performed in the period of 24 hours or even in the presence of marked swelling and clear evidence of direct fracture can usually be accomplished by closed methods of manipulation within the first 24 hours. Although the approach may tax the ingenuity and skill of the surgeon, it must be borne in mind that orbital fractures heal very quickly and an eye reduced early seldom deforms. Even a complete reduction is hopeless to try after three weeks.

Ocular proptosis or exophthalmos can be due to a great many things. It may be lateral or unilateral, apparent or true, and an accurate diagnosis must be made in order to give proper treatment. Most often the diagnosis is difficult and requires most careful studies, repeated observations and consultation with colleagues in many fields of medicine.

Apparent proptosis may be the result of orbital fat, a familial feature or facial asymmetry. In the presence of swelling, edema of the lid or inflammation, proptosis may be simulated. Purely ocular causes, such as iritis or glaucoma, and inflammatory lid conditions may yield an apparent proptosis. There are also rare cases of voluntary or semivoluntary forms of ocular proptosis.

In some cases in which there is an exophthalmos or retraction of the globe within the orbit, as in Horner's syndrome or fracture of the floor of the orbit, for example, the opposite eye by comparison may appear to be proptosed. Likewise when there is a slight ptosis of the upper lid, the other eye may appear to be exophthalmic. Nor is it unusual to see a patient who shows a retraction of the upper lid without exophthalmos, which may be unilateral, and is due to past or present thyrotoxicosis.

The determination of true proptosis is made by measurement of the degree of exophthalmos either with the Hertel exophthalmometer or by means of a transparent Lucite millimeter ruler. These instruments permit one to make a measurement of sufficient clinical accuracy to record the variations of degree of proptosis from time to time. A difference of 3 mm between the eyes is of significance.

Inspecting the two eyes in a good light and noticing whether the eyeball is displaced forward vertically or laterally is of importance because the direction of the proptosis is often helpful in deciding its cause. In order to discover slight degrees, it is sometimes necessary to elevate both of the lids simultaneously and have the patient

suffered surprisingly little permanent damage and sight was little affected, although the intraocular coats particularly the choroid, might have been ruptured.

EMERGENCY SURGICAL CONDITIONS

When a person is seriously injured, unconscious and in shock, it is rare that the surgeon pays any attention to the condition of the eyes. If the eye is injured, this can have most serious consequences to sight. An inspection of the eyes must always be made as a routine part of the study of such a patient and, if the eyes are found to be injured, they should be treated as soon as the general condition of the patient will permit. Since much of the surgical repair can be done with the patient under local anesthesia, there is no need to postpone such repair unduly.

The inspection of an eye should be made in a good light, preferably a beam of light focused onto the eyeball with a condensing lens. A good flashlight is often most adequate. If the lids are swollen and in spasm it may be necessary to open them with lid hooks or small retractors gently inserted beneath them. One should observe the cornea and corneal reflex, the anterior chamber the pupil and, upon gently rotating the eye the bulbar conjunctiva and sclera and the insertions of the four rectus muscles. The condition of the red reflex, as seen with the ophthalmoscope, should be ascertained. Gentle finger palpation of the eyeball in the absence of a perforating wound of the globe will give a rough idea as to the state of the ocular tension—whether the eyeball is soft, indicative of a rupture or perforation of the sclera, or hard, indicative of intraocular hemorrhage.

Lacerations of the lids are common. Although they are usually the results of injury by a sharp object, they can also be caused by contusions and concussions. The lids should be cleansed with soap and water and the extent of the laceration and the structures involved ascertained. Infiltration of the affected area with procaine solution gives efficient anesthesia. Primary closure of the laceration, preferably with 5-0 or 6-0 silk sutures spaced closely together can then be done without difficulty. If the laceration is deep and extends to the orbital periosteum or through the tarsal plate, repair of this part of the wound with 3-0 or 6-0 gut sutures should be carefully performed before the skin is closed. The tissues should be

replaced as accurately as possible in their original position.

Lacerations of the lid margin present slightly more difficulty. In order to prevent an eversion of the lid, the margins must be exactly approximated. The key suture here is through the intermarginal areas one to the other tightly tied so that a slight elevation of the joined edges occurs. This always flattens out later. The remainder of the laceration is then sutured, usually quite easily.

A laceration through the lids involving the upper or particularly the lower canaliculus of the lacrimal apparatus (nasal side of the lids) is one of the most difficult and at the same time the most important to handle properly at the first instance. It is necessary to identify the two cut ends of the canaliculus exactly. A sterile probe of small wire no. 1 catgut, preferably chromicized, or heavy linen thread is inserted into the punctum, then through the canaliculus, joining both ends and either well into the nasolacrimal duct or at least into the tear sac, and held in place by whip sutures. The laceration is then properly sutured. The guide suture or wire is left in place for at least four days or longer if it is not causing a tissue reaction. If suturing is not done at once later when scar tissue forms, it is usually impossible to identify the ends of the cut canaliculus and to have a patent functioning tubule.

Ten per cent sodium sulfacetamide ophthalmic ointment, or other antibiotic ophthalmic ointment, is then placed on the lids and a firm eye pad, pressure bandage combination is applied. There is generally no need to inspect the wound for two or three days. The sutures should be removed in four to five days.

Burns of the lids may either be thermal or chemical in origin and present the same problems as do burns of the skin elsewhere. Gentle but thorough cleansing with soap and water and the occasional application of an ophthalmic antibiotic ointment may be all that is necessary in mild injury. In severe third degree burns, however, it is essential that the eyeball be protected by suturing the lid margin together or if there is too extensive loss of tissue to insure this, by covering the cornea with a conjunctival flap or in desperate cases by bringing a sliding skin flap over the eyes from above or below or even by cutting all four of the rectus muscle tendons at their insertions and

removal of a tumor which lies in the middle part of the orbit is best accomplished by the Kronlein approach.

Severe progressive (malignant) exophthalmos may require unroofing of the orbit in order to save the eye and sight by permitting herniation of the enormously thickened muscle cone and the relief of the eyeball from pressure. The operation is futile when the muscles are completely livalinized and no longer are elastic.

Tarsorrhaphy or the sewing of the lateral third or half of the eyelids together is a useful operation designed to protect the eyeball from dangerous exposure in moderate degrees of exophthalmos.

Wounds of the globe may be penetrating or perforating, single or multiple or due to concussion. Perforating wounds of the cornea may extend into the sclera or may remain localized in the cornea. When such a wound occurs, there is immediate loss of aqueous humor and the iris pushes forward to plug the wound or prolapse through it. If the injury is severe enough, loss of the lens and vitreous humor occurs and thus total loss of the eye may result.

It is for these reasons that a most careful examination of a corneal injury should be made. Not only should the eye be handled most gently, but the patient should not be ambulatory until after the wound has been repaired.

If the examination of the eye shows that iris or ciliary body prolapse has occurred, the prolapsed tissue must be excised and the wound closed with direct appositional sutures. Care must be taken not to place the sutures too deeply in the cornea, because of the danger of fistulization of the wound or growth of epithelium into the anterior chamber. This procedure requires great skill and delicacy, for undue pressure upon the eyeball may express the lens and vitreous humor.

It is sometimes easier to close the wound by covering it with a conjunctival flap. In this procedure, the conjunctiva is separated from the limbus and sclera in the area adjacent to the wound. Following excision of prolapsed uveal tissue, the loose conjunctiva is then brought over the wound and sutured in the limbic zone on each side like an apron.

Or peritomy of the corneoscleral margin can be done and the entire bulbar conjunctiva separated from the sclera by blunt dissection. The loose conjunctiva is then

brought over the cornea and closed with a purse-string suture.

An x ray photograph must be taken of every eye which has had a perforating or penetrating injury for this constitutes a medicolegal responsibility. It is surprising how often the x ray photograph reveals the presence of an opaque intraocular or intra orbital foreign body that is usually metallic and fortunately generally magnetic.

However a simple anteroposterior or frontal x ray photograph is not enough, for the removal of a magnetic foreign body with minimum additional trauma to the eye, is one of the most difficult ophthalmic operations to perform successfully.

For proper removal of a magnetic intraocular foreign body it is entirely necessary to know its exact location within the eye and its exact size. This requires most expert skill in localization with x ray photographs and accurate charting of the result. The Sweet method of localization is the one most frequently used and has stood the test of time. Many roentgenologists are quite skilled in its technique and are able to localize the foreign body within a fraction of a millimeter. Others unfortunately are less skilled and have reported the foreign body as outside the eyeball when it is within it, or vice versa, errors which can be most dangerous, or at least embarrassing.

A new and valuable adjunct to the exact localization is the use of the Berman localizer, an instrument the principle of which on a minute scale is that used in the detection of land mines.

Having determined the exact size and position of the foreign body the surgeon makes an incision into the sclera, choroid and retina in the area closest to the foreign body. The incision must be large enough to permit withdrawal of the object with the sterile tip of a strong hand magnet. The wound area then is stippled with diathermic ignipuncture to prevent secondary retinal separation, and the scleral wound is closed with fine silk sutures or other sutures, as required.

One of the most valuable lessons learned in World War II was the fact that there is no great urgency in removing an intraocular foreign body. It is far better to wait until accurate x-ray localization can be obtained and the services of an expert ophthalmic surgeon are available than to cut into the eyeball and blindly apply the magnet.

The removal of a nonmagnetic foreign

gaze directly forward. It is often better to stand behind the patient, who is seated on a low chair or stool and look down across the plane of the orbits from above. It is also wise to sight along the plane of each orbit from the lateral or profile view.

Ocular motility is then tested and limitation of movement in any direction of gaze is noted. An analysis of the limitation of ocular motion is of value in the diagnosis of the site of the lesion and frequently of its cause.

Palpation and stethoscopic examination of the orbit give useful information at times. Palpating the orbital contents may reveal the presence of a tumor. In addition, one can obtain a rough estimate of the degree of the orbital pressure. It is always necessary to have x-ray photographs made. These should include plates of the skull as a whole, the orbits, sinuses and the optic foramen of both sides. A nose and throat examination is frequently advisable. Blood studies may be indicated and other laboratory tests such as a determination of the basal metabolic rate or iodine uptake studies are often advisable in order to arrive at the proper diagnosis.

True proptosis may be classified for the purpose of differential diagnosis in the following manner:

I. Monocular

A. Rapid proptosis with edema of the lids

1. Ocular (glaucoma, iridocyclitis, panophthalmitis, tenonitis)
2. Orbital (cellulitis, cavernous sinus thrombosis, hemorrhage)

B. Gradual proptosis without edema

1. Congenital malformation of the orbit (encephalocele, meningocele)
2. Ocular enlargement (high myopia, buphthalmos, anterior megalophthalmos, intraocular neoplasm perforating posteriorly)

C. Unilateral proptosis with central displacement

1. Thyrotoxic or progressive exophthalmos
2. Vascular causes (arteriovenous aneurysms or pulsating exophthalmos)
3. Tumors of the optic nerve or those arising in the optic foramen
4. Cysts of the orbit
 - a. Congenital (meningocele se-

rous cysts seen in congenital microphthalmos, dermoid cysts)

- b. Acquired (cysts which are developed as the result of exudation and the inclusion of epithelial tissue as a result of trauma or around neoplasms, parasitic cysts [hydatid or cysticercus])

5. Neoplasms of the orbit

- a. benign as in plexiform neuroma
- b. Malignant (mixed tumor of the lacrimal gland, primary sarcomas, metastatic neoplasms, chloroma, pseudo tumor, malignant lymphoma)

6. Foreign bodies

D. Unilateral proptosis with lateral displacement

1. Mucocele from the sinuses, neoplasm from the sinuses, bone exostosis from the orbital walls (osteoma, meningioma) the result of nasal surgery (radical frontal sinus surgery) orbital osteoperioritis, meningocele, emphysema of the orbit

II. Binocular

1. Congenital or as a familial trait
2. Tower skull
3. Graves disease including progressive (malignant) exophthalmos
4. Neoplasms arising in the ethmoid or sphenoid sinuses extending into both orbits
5. Double-sided sinusitis (pansinusitis)
6. Neurofibroma
7. Myasthenia gravis
8. Cavernous sinus thrombosis
9. Mikulicz's disease
10. Sarcoidosis
11. Cranio-orbito-facial malformations

Thus we see that the sign of exophthalmos is a complex matter which demands careful investigation.

The removal of an orbital tumor especially when it is decided that it lies in the posterior third of the orbit, is best accomplished through frontal craniotomy and unroofing of the orbit. Many of these tumors are found to arise within the skull and invade the orbit by direct extension. The

found the first eye muscle and pulled it easily forth with my Mauldheim made instruments. Soon all the eye muscles were cut. The most dramatic moment arrived when I felt for the optic nerve the only sound being the clicking from the film camera in the background. Successfully severing the nerve I then extracted the eye globe and tied the eye muscles together closing the wound with conjunctival membrane. The operation was successfully over after an exciting two hours and forty minutes.

In enucleation of the globe first a lid speculum is inserted then the conjunctiva is incised at its insertion at the cornea the incision is enlarged all around the cornea (peritomy) and, with blunt dissection the conjunctiva is undermined and separated from the sclera as widely as possible. This will expose the rectus muscles. A muscle hook is then inserted beneath the tendon, beginning with the superior rectus muscle and going on to the medial and inferior rectus muscle cutting as closely as possible to the globe and thus avoiding some bleeding. The last muscle to be severed is the lateral rectus. Enough of a stump of this muscle is left attached to the eyeball so that it can be strongly grasped with tooth forceps or hemostat. The eye is rotated medially. A half-curved, blunt pointed scissors is inserted along the temporal side of the eyeball, between it and the separated conjunctiva, and when the scissors is behind the globe search is made for the optic nerve which is a firm cordlike structure. The nerve is then placed between the blades of the scissors and cut. At the same time the eyeball is pulled and tags of restraining tissue (superior and inferior oblique muscle tendons) are cut off. There will be a smart hemorrhage when the nerve is cut and the eye removed. This is controlled by a cone-shaped tampon of cotton placed beneath the conjunctiva into the socket and held with firm finger pressure directed toward the apex of the orbit. Bleeding should stop well within ten minutes. When it stops the tampon is removed and the conjunctiva is sutured together with three well placed 3-0 to 4-0 interrupted silk sutures. Sulfacetamide ophthalmic ointment is placed in the eye and the lids are closed eye pads and a firm elastic or adhesive plaster bandage are applied. The sutures can be removed in five days and an artificial eye inserted in ten days.

In the event of panophthalmitis the eye

should be eviscerated. The cornea is completely excised and the contents of the eye are scooped out with a blunt curet. Care should be taken to remove all of the pigment tissue. The inner walls of the scleral cavity must be white everywhere. There will be little or no bleeding when the cavity is entirely clean. Sulfacetamide ophthalmic ointment is inserted and the lids are closed and covered with an eye pad and a firm bandage.

Thermal and actinic burns of the eyeball are relatively rare. Most commonly they occur as the result of arc welding or long exposure to harmful ultraviolet light. In these conditions the eyes are usually quite painful. The application of cold compresses and sulfacetamide ophthalmic ointment and the occasional instillation of a drop of 1:1000 solution of Adrenalin chloride usually relieve the patient and the eye heals readily in twenty four hours.

Severe burns of the eyeball affecting the bulbar tarsal conjunctiva and cornea are most difficult to treat because the cornea generally perforates with loss of the intraocular contents. However ice compresses of saline solution, atropine and sulfacetamide ophthalmic ointment and frequent separation of the lids from the bulbar conjunctiva to prevent adhesions are employed. In deep bulbar conjunctival burns immediate graft with a suitable piece of buccal mucous membrane should be made.

Chemical burns are not rare and each employee of a chemical plant or workers in chemical laboratories should receive instruction in first aid treatment of this condition. The opened eye is irrigated copiously with tap water preferably using a fountain syringe with a rubber tip for a full five minutes. The eye is then carefully inspected, a drop of 0.5 per cent solution of Pontocaine is instilled, followed by a drop of 2 per cent fluorescein solution to stain the burned or abraded cornea in order to reveal the extent of the damage. All foreign bodies are removed with a toothpick swab and the eye is gently irrigated for fifteen minutes with warm normal saline solution. The eye is again stained with fluorescein and irrigated. If the greenish stain is retained by the cornea, it indicates a definite chemical burn and the patient should be referred to an ophthalmologist for definitive care.

In the past, attempts have been made to neutralize the responsible chemical by suitable chemical agents but experience has shown that simple but thorough flushing of

body is exceedingly difficult, often impossible. If the foreign body is near to the retina and choroid it sometimes can be grasped through a scleral incision with suitable forceps and withdrawn. Such an event is most fortunate.

Of all the innumerable kinds of nonmagnetic foreign bodies reported, those composed of copper or its alloys are the most dangerous to leave within the eye for the metal undergoes oxidation and produces copper oxide a substance that is highly toxic to the intraocular contents. Stone, glass, wood and plastic material can often be well tolerated sometimes for a long time provided no infection occurred in the beginning.

Injuries to the lens are common and generally produce complete opacification (cataract) of it in a short time, for when the capsule of the lens is opened, aqueous humor enters into the lens fibers and leads to cloudy swelling. The cataractous lens is removed by several methods depending on the age of the patient and the condition of the eye.

When the lens is removed (aphakia) there is no focusing power remaining in the eye and the patient must wear a substitute lens in a spectacle frame. If a contact lens is tolerated a host of difficulties is avoided. Usually however when the uninjured eye is sound and has good vision, correction of the refractive error in the aphakic eye causes confusion between the two and binocular single vision is impossible to attain for a number of optical reasons.

However an aphakic eye without correction, if otherwise sound, still has most useful peripheral vision and in addition is a reserve eye in case anything should happen to its fellow.

Injuries to the choroid, a highly vascularized tissue, generally consist of ruptures of this ocular coat. Severe intraocular hemorrhages either into the retina or breaking through into the vitreous always occur in this event. The location of the rupture, which is generally in the posterior pole of the eye, will determine whether or not useful vision is retained after the hemorrhages are absorbed.

If the macula of the retina, the fixation point of vision, is damaged, the resultant central scotoma in the field of vision is permanent. If the periphery of the retina is affected, central vision may be retained and may even be normal.

Injuries to the retina may or may not

result in a tear or tears of this membrane. If the retina is torn, separation or detachment of it almost always ensues. In this event, surgery is necessary and consists of first localizing the tear as exactly as possible with the ophthalmoscope and then treating this area with perforating diathermic ignipunctures, in order to produce points of adhesive chorioretinitis, thus sealing the retina back in its place. Obviously the exact localization of the tear in the retina requires utmost skill in the use of the ophthalmoscope and the technique of diathermy. Coagulation of the retina is one that demands much experience and special training in its use. About 70 per cent of the patients having retinal detachment, both traumatic and idiopathic in origin, are successfully treated by this method in the hands of experts.

If a perforating injury of the eyeball occurs, particularly when it involves the iris or ciliary body the risk of subsequent sympathetic ophthalmia is ever present and is always to be considered. This is a most difficult problem to solve. Fortunately sympathetic ophthalmia rarely occurs before the tenth day following injury so that there is a certain leeway in deciding whether or not an injured eye should be removed. Also it is consoling to know that sympathetic ophthalmia is quite rare for example very few of these cases occurred during World War II. Thus a conservative approach is always best unless it is obvious that the eyeball is hopelessly injured and that there is no prospect of useful vision. Therefore if expert ophthalmic care is available the general surgeon is never justified in removing an injured eye but leaves it to his colleague to assume the grave responsibility of decision.

If however such is not the case and if the injured eye remains inflamed and soft, with vision markedly reduced for more than a week, it may be necessary for the general surgeon to remove the eye. This emergency happened to Dr. Ove Wilson, the only surgeon with the joint British-Scandinavian Antarctic Expedition in Vaudheim, Antarctica, on July 21, 1953. He was faced with the necessity of removing the injured right eye of a fellow member of the group, an operation that he had neither seen before nor ever performed. In writing to the *London Times* about this experience later he said, "Waiting for the operating moment I recapitulated all details of the operation in my mind, wondering whether I should be able to find all the different eye muscles and the right place to cut the optic nerve." 1

normal mucous membrane cannot meet the burden of added humidification and drying increases the viscosity of the mucous blanket. Irritants and bacteria are not carried away and, under these circumstances the mucous blanket becomes a medium actually encouraging bacterial growth. Prolonged congestion and irritation caused by the infection produce hyperplasia and permanent respiratory obstruction results. Numerous local and general symptoms are produced and the opportunity exists for extension of the infection to other organs.

The original obstruction may be produced by either functional or structural changes in the nasal cavities. Allergy, chemical irritants, dust, fumes, swimming and diving, menstruation, endocrine imbalance, dietary deficiencies and any condition which produces circulatory changes may be the etiologic factor. The structural changes are of more surgical significance. These include deviations of the nasal septum, septal spurs, hyperplasia of the nasal turbinates, synechiae, bone cysts, exostoses, nasal polyps or other nasal tumors, choanal atresia, hypertrophied or infected adenoids, rhinoliths and other foreign bodies. Any or all of these conditions can usually be corrected by surgery.

Perhaps the most frequently found cause of varying degrees of nasal obstruction is a *deviation* or deflection, of the *nasal septum*, the result of an asymmetrical malformation of the cartilaginous or osseous or both, portions of this structure. In some instances, such a deformity is the result of trauma to the nose and septum but most often is the end result of overgrowth of the septal components. Since insufficient space is available to accommodate the overgrowth, buckling, bulging, twisting and overriding of portions of the septum occur. At times a high, arched palate is responsible for the lack of space available for septal growth, but such a palate deformity is not invariably accompanied by a septal deflection. This adds credence to the view that overgrowth, or redundancy of the septal parts is responsible for most septal deformities, with trauma second in importance.

There is wide variation in the form and degree of deviation produced, but the types of deflections seen tend to fall into certain patterns. The normal septum is essentially straight from top to bottom and from front to back and the variations from this normal pattern can be classed as convex deviation, sigmoid deviation and septal ridge. The convex deviation produces a unilateral bulge more or less continuous from top to bottom,

which narrows one nasal cavity and produces overpatency of the opposite side. The sigmoid deviation is an S-shaped deformity that blocks one side above and the other side below. An S shaped buckling can also occur that blocks one side anteriorly and the opposite side posteriorly. The septal ridge is a prominence running horizontally from front to back. A more severe form of buckling occurs in which both bone and cartilage are crumpled and irregular ridges and sulci on both sides of the septum are produced. A very common type of deformity is one in which the lower end of the cartilage is displaced from its groove in the nasal spine of the maxilla and presents in the nostril. All of these deformities are often associated with spurs, crests and ridges of various shapes and external deformity is frequently produced.

The symptoms produced by deflection of the nasal septum depend upon the degree of distortion and the area in which obstruction is produced. This may vary from mild interference with nasal breathing to obstruction severe enough to occlude sinus drainage. Pressure of the deflection, spur or ridge on nerves may produce neuralgia referred to areas about the head, face, eye and ear. Overventilation of the unobstructed side may produce drying and infection, resulting in disease and symptoms in the overpatent side.

There are few conditions which can be confused with a deflected nasal septum if the diagnosis is made by direct examination. Symptoms of nasal obstruction give no clue as to the condition responsible.

If the obstruction produced by a deflected septum requires treatment, the treatment is of necessity surgical. The results of surgical correction are uniformly good when the procedure is done by an adequately trained surgeon. In most cases, a satisfactory airway can be restored. The effect of the restored airway on the secondary complications of the pre-existing nasal obstruction depends on the length of duration, severity, extent of changes produced, the physical condition of the patient and the persistence of after treatment of the secondary condition.

The operation for correction of a septal deviation is known as a submucous resection and is accomplished by incising the septal mucosa and perichondrium to the cartilage just posterior to the anterior nares usually on the convex side of the deflection. The perichondrium and periosteum are carefully elevated from the cartilage and bone of the septum care being taken to avoid tearing

the conjunctival flap with tap water for five minutes is much more simple and efficient. Occasionally the neutralizing solution is quite irritating, even harmful. Besides, the neutralizing solution is rarely at hand for immediate use.

NONEMERGENCY CONDITIONS

Surgical conditions which are nontraumatic in origin, such as the removal of cat-

aract correction of ocular muscle anomalies, operations for glaucoma and detachment of the retina, keratoplasties and surgery of the lacrimal apparatus, are properly the domain of the ophthalmic surgeon. Plastic surgery of the lids and orbit is a highly specialized procedure and requires special training and experience in order to obtain the best results.

The Nose, Nasal Accessory Sinuses and the Pharynx

By FLETCHER AUSTIN M D

FLETCHER AUSTIN is the son of a physician who practiced in Illinois and later moved to Ohio. After graduating from Ohio State University, Doctor Austin attended Northwestern University Medical School and there earned a Master of Science degree in Anatomy. He received his training in otolaryngology in Iowa and New York. He believes that the problems of the ears, nose, larynx and pharynx have not all been solved by the advent of chemotherapy and the antibiotics and that one should have a broad base in surgical training before extending the scope of any particular surgical specialty. Doctor Austin is an Associate in Otolaryngology at Northwestern University.

THE NOSE

The rhinologist is consulted primarily because of impaired function, disease or cosmetic appearance of the nose. The nose is the starting point of many infections which may subsequently involve the sinuses, pharynx, ear, eye, gastrointestinal tract, bronchopulmonary system, the kidneys and the blood and lymph circulation. In addition, it is affected by congenital and acquired malformations, specific infections and neoplastic involvement. There are some common intranasal conditions that impair the function of the nose, in which surgical treatment may be reasonably expected to aid in restoring the normal physiology.

The basic function of the nose is respiration and olfaction is second in importance. The nasal cavity also acts as a resonating chamber for the voice and is the origin and recipient of numerous reflex arcs.

The secretions of the nose possess a bacteriostatic property. The vestibule of the

nose is lined by skin containing hairs, or vibrissae which defend the nasal cavities against the intrusion of foreign material. The nasal structures are richly vascular and the inferior turbinates have a cavernous structure that is essentially erectile tissue. The mucous membrane of the nasal cavity is ciliated and the action of the cilia constantly moves a thin blanket of mucus toward the nasopharynx. These are the mechanisms which clean, warm and moisten the inspired air preparing it for reception by the lower respiratory tract and the lungs. In order to properly accomplish this, the entire nasal mucosa must function normally. If, for any reason, the ventilation and drainage of any part of the mucosa are interfered with, symptoms will develop. The normal movement of the blanket of mucus is impaired, dust and bacteria are not removed and the membrane becomes infected. The resulting congestion causes increased obstruction and a vicious cycle is established. The remaining

small superficial vessel Digital trauma to the area is the most common inciting cause The bleeding vessel in many instances is so close to the anterior nares that compressing the nostrils between the thumb and index finger may stop the hemorrhage. A small pack, moistened with an ephedrine Adrenalin or cocaine solution and firmly but not forcibly placed against the area may be necessary if the bleeding is brisk Once the bleeding point is located, the offending vessel may be destroyed by the application of chemical or high frequency cautery

Severe persistent hemorrhage and inability to find the bleeding vessel may require firm packing of the entire nasal cavity and the introduction of a postnasal pack. It is often helpful to pack the nose in segments, which aids in determining from what area the bleeding is arising.

If nasal hemorrhage cannot be controlled or recurs at frequent intervals, a careful study must be made to determine if a blood dyscrasia, neoplasm or some condition other than simple rupture of a vessel may be responsible. Ligation of the external carotid artery is sometimes necessary to control the hemorrhage and save the life of the patient

Formation of a *furuncle* is a common, painful and potentially dangerous condition which occurs about the nose, forehead, upper lip and in the nasal vestibule. Venous drainage from these areas forms a fairly direct route through the ophthalmic vein to the cavernous sinus and fatality from cavernous sinus thrombosis has followed improper treatment of a seemingly simple, circumscribed infection in one of these locations. Such an infection should be treated by warm fomentations and the patient confined to bed. These furuncles should not be incised but should be allowed to resolve or drain spontaneously Picking and squeezing of the lesion should never be permitted. Early use of antibiotics is indicated if the infection is at all severe.

Fracture of the nasal bones is due to direct violence and the displacement which occurs usually depends upon the direction of the blow producing the fracture The bones may be pushed directly backward, crushing or buckling the septum beneath them, or the displacement may be in a lateral direction. Such fractures may be compound or comminuted. The fracture frequently occurs at the junction of the nasal bone with the nasal process of the superior maxilla, because of the relative thinness of the bone at this point. The nasal process of the maxilla is frequently included in the fracture. Frac-

ture at the glabella does not often occur because of the thickness and density of the bone in this region Fracture of the nasal bones may occur with no displacement of the fragments and in this case no manipulation is necessary although the nose should be protected from further injury by splinting

If displacement occurs reduction should be done before swelling obscures the deformity The depressed or displaced bones may be brought into proper alignment by introducing a closed forceps or a flat, rigid elevator into the nasal cavity and lifting the bones and molding them into place by counterpressure applied by the fingers externally If they tend to sag back into the nasal cavity petrolatum gauze packing high in the nose will support them in place until the healing processes will maintain the reduction

The nasal septum is often injured by the trauma producing the fracture of the nasal bones or the septum alone sustains the injury If the septum is dislocated from its attachments it may be grasped between forceps blades protected by rubber sheathing and lifted or rotated into place. The septum is sometimes fragmented or so distorted that replacement is difficult. In such cases, a submucous resection done at the time to remove the bone and cartilage so involved, will hasten healing and prevent later obstructive deformities.

At times the nasal bones are so severely fractured and displaced that it is necessary to hold them in place by transfixing wires or sutures or by devising an apparatus which will exert mild sustained traction If reduction of the fracture has been delayed two weeks or more it may be necessary to break up the existing bony union by the use of chisels to achieve restoration of the bones to their normal position. This constitutes an open reduction and is usually accomplished by employing the techniques used in rhinoplasty

An external splint fashioned from dental compound, or of metal, shaped to fit the nose and extending from the forehead well down past the nasal bones is held in place by adhesive strapping to protect the reduction until union of the fragments is firm Loose intranasal packing with petrolatum gauze is also usually required.

THE NASAL ACCESSORY SINUSES

Acute and chronic infection of the paranasal sinuses is almost always bacterial in origin, but the development of inflammatory

or perforating the membrane. This elevation is extended to include the total area of deformity. The cartilage is then cut through, at the point of the initial mucous membrane incision to the perichondrium of the opposite side and that membrane is elevated in a like manner and with the same care. The deformed cartilage and bone are removed in segments; bleeding is controlled, the space between the flaps formerly occupied by the septum is cleansed of fragments of bone and cartilage and the flaps are approximated and held together by firm packing of the nasal cavities for twenty-four hours.

A laceration or perforation of one or both of the membrane flaps during the operation is of little consequence unless the tears or perforation appose when the flaps are closed. In this instance, a permanent perforation of the membranous septum is almost sure to result. Replacing a piece of straight cartilage between the two lacerations may prevent this complication.

Trauma to the nose may produce a *hematoma* beneath the perichondrium or periosteum of the intact septum or a hematoma may occur between the apposing membranes following a submucous resection. It is more commonly the result of trauma and, although it occurs more often in children than in adults, it is seen in persons of all ages owing to the increasing frequency of head injuries. When it occurs it usually produces obstruction to both nasal airways. Palpation will make the diagnosis if it is not recognized by sight, the softness and fluctuation differentiate this lesion from a solid obstructing mass. The treatment is free incision at the base of the hematoma with careful removal of the blood, clots and any bone or cartilage fragments. Gentle suction may be used, but irrigation of the space may cause further elevation of the mucosa and introduce and spread infection. The incision should be kept open by a drain until any danger of recurrence is past. Antibiotic or sulfonamide therapy lessens the danger of supervening infection.

Failure to drain a hematoma may result in marked thickening of the septum and permanent nasal obstruction. The hematoma may become infected and a septal abscess may develop.

In instances in which a septal abscess develops, there is usually a history of trauma followed by nasal obstruction. Development of an abscess produces fever and leukocytosis and local heat and inflammation are evident on examination of the septum. Treat-

ment is the same as for hematoma. Drainage maintained for several weeks may be necessary. Failure to drain or inadequate drainage may result in marked destruction of the cartilage and produce a "saddle-nose" external deformity. Because of the severity of the infection and the possibility of its extension to the intracranial cavity, antibiotics and/or chemotherapy are indicated.

Chronic hyperplastic rhinitis, one form of many acute and chronic infections which may involve the nasal cavities, is produced by constant irritation of the nasal mucosa by one or a combination of numerous agents. The epithelium thickens and fibrosis of the tunica propria occurs. This is prone to affect the erectile portions of the turbinal mucosa. Contraction of the fibrous tissue produces corrugations and irregularities in the thickened, swollen mucosa, causing obstruction to nasal respiration and to sinus drainage. The best treatment is to prevent the changes from occurring by eradicating the cause; for after fibrosis is established, the changes become irreversible. Surgical correction is then necessary and is accomplished by removing all, or the affected part, of the turbinates involved or by destroying the thickened tissue by actual or high frequency cautery. Introduction of a cautery needle submucosally preserves the ciliated epithelium and is the preferred method of application. Subsequent scarring and contraction reduce the size of the turbinate and relieve the obstruction.

A rather rare cause of nasal obstruction is *atresia* of either the anterior or posterior nares. This is usually congenital but may be acquired as a result of syphilis, tuberculosis or injury. The anterior atresias are membranous and those involving the posterior choanae are bony or membranous or both. They are either bilateral or unilateral and, if unilateral, may escape detection. The treatment is surgical removal. The approach to the posterior atresias is difficult and the surgery is often unsuccessful, owing to the difficulty of removal and the tendency for the surgically created opening to close.

Spontaneous hemorrhage from the nose, or *epistaxis*, is a common and frequent occurrence which is often difficult for the surgeon to bring under control and, if prolonged, results not only in considerable blood loss but almost invariably produces some degree of shock, and an emotionally disturbed and frightened patient. The commonest site from which bleeding occurs is at the anterior inferior end of the nasal septum, the bleeding is due to the rupture of a

though headache is sometimes a prominent symptom in sinus disease any attempt to diagnose the sinus involved by the location or character of the headache can be most inaccurate.

Redness and swelling may occur over any acutely involved sinus which lies close to the skin and is frequently seen at the inner angle of the orbit in acute ethmoiditis in infants or children. Edema can occur without inflammation.

Transillumination gives additional diagnostic information about the frontal and maxillary sinuses but cannot be applied to the ethmoid or sphenoid sinuses because of their anatomic location. The light fails to pass through, or passes through with diminished intensity those sinuses which are involved. Transillumination is affected not only by the condition of the sinus itself but also by the thickness of the bone and other tissues through which the light must pass.

Failure of a frontal sinus to transilluminate may indicate a small or absent sinus rather than infection. Transillumination is presumptive evidence of normality or abnormality of the sinus thus examined and is valid only when considered with the objective clinical findings and the history. It is of questionable value in children, and in adults additional x-ray evidence is often necessary to determine the location and the severity of involvement of the diseased sinus.

The x-ray evidence of sinus disease must also be interpreted in connection with the history and the clinical findings. The instillation of radiopaque oil into the sinuses, followed by x-ray examination, outlines the cavity of the sinus and gives valuable information concerning the thickness of the lining membrane and the presence of a polyp cyst or tumor. The oil is introduced into the ethmoid cells by the displacement technique of Proetz and into the others by cannulating the natural opening, whenever possible.

If, after these diagnostic procedures the diagnosis is still uncertain, it may be necessary to irrigate the sinus to obtain direct washings for study. This procedure should also be done through the natural opening, if conditions permit.

The complications of sinusitis may vary from some inconsequential local condition to the most desperate intracranial or systemic involvement. The diseased sinus may act as a focus of infection or the infection may spread by direct extension through continuity of structure along fascial planes, through

dehiscences of the bony walls along perineural sheaths and by the blood and lymph streams. Thrombophlebitis of a perforating vein is common and infection may reach the general circulation in the form of bacteremia.

Optic neuritis and retrobulbar neuritis not infrequently follow infection of the ethmoid or sphenoid sinuses either by direct extension of the infection by vascular congestion secondary to the sinusitis by pressure on the optic nerve by bone or by periosteal changes induced by the sinus infection. Toxemia may produce loss of vision when the sinus serves as a focus of infection.

Orbital cellulitis and orbital abscess are commonly produced by extension of infection from the sinuses most commonly from the ethmoid cells or the maxillary sinus. They are most often seen in children. The infection reaches the orbit through a dehiscence in the wall or it is due to thrombophlebitis. Pus may burrow forward along the periorbita and present in the lid without involving the orbital contents or burrow posteriorly along the periorbita to the sheath of the optic nerve and produce intracranial involvement. If an orbital cellulitis occurs, thrombophlebitis of the orbital vein may result and the infection extend to the cavernous sinus. Other vascular channels can be involved by the same mode of extension.

Osteomyelitis of the skull may result from the spread of infection, usually by thrombophlebitis, into the bones of the calvarium. The outer table of the skull usually breaks down first, resulting in the formation of a subperiosteal abscess. Breaking down of the inner table does occur and an intracranial complication results. Necrosis of the bone of the roof cells of the ethmoid labyrinth not uncommonly occurs with direct extension of the infection into the anterior cranial fossa. Meningitis and brain abscess are the common intracranial complications.

Respiratory system complications are a common result of sinusitis and range from tonsillitis, pharyngitis and laryngitis to bronchitis, bronchiectasis, asthma and lung abscess. Generalized systemic complications and various forms of renal involvement can sometimes be traced to an active sinus infection.

The use of antibiotics and/or chemotherapy has had a pronounced effect on both the medical and surgical treatment of sinus disease. When this therapy is employed, the attack of acute sinusitis usually subsides quickly with much less deleterious effect on the sinus mucosa than was formerly obtain-

sinus disease is markedly influenced by the presence of conditions in the nose or sinuses which interfere with drainage and ventilation of the sinuses through their natural openings. The sinuses may become infected when no such obstruction exists if the invading organism is of particular virulence or if the body resistance of the host is lowered. Chronic sinus disease however is not so apt to develop in those sinuses which suffer no interference with aeration and drainage.

The most commonly encountered obstructive lesions are a deflected nasal septum or a septal spur an enlarged or cystic middle turbinate nasal polyps or other nasal tumors, foreign body and chronic congestion of the nasal mucosa and nasal turbinates secondary to a chronic tonsil, adenoid or nasopharyngeal infection.

Obstruction in the region of the middle turbinate is of particular significance, owing to the fact that the frontal maxillary and the anterior ethmoid cells drain into a relatively restricted area of the middle meatus and infection of one or all of these sinuses may result.

Allergic rhinitis is often followed by secondary sinusitis, owing to blocking of the sinus ostia by the marked changes in the nasal and sinus mucosa. The floor of the maxillary sinus is in direct relation to the roots of the upper teeth and dental sepsis may extend to that sinus or infection may be introduced when the sinus cavity is unavoidably or accidentally opened during the extraction of an upper tooth. Trauma to the bony framework of the sinuses is increasingly common and the disruption produced by fractures and penetrating injuries is often followed by a severe infection. Destructive new growths originate in or extend into the sinuses and the effect of the tumor on the mucosa, plus the interference with drainage results in sinusitis which may be the complaint which leads to discovery of the tumor.

It is generally agreed that some factor or factors other than the presence of bacteria is necessary to produce sinus disease. In trauma, the injury to the sinus plus the introduction of bacteria may be sufficient. The obstructive lesions have been noted. In sinus disease, if the etiologic basis is not evident, more obscure factors must be considered. These include general debility lowered resistance from emotional stress, overwork or exposure dietary deficiencies, endocrine imbalance and hereditary or acquired pathological and anatomic factors. With or without

the presence of any of these contributing factors, the most common cause of sinus infection is bacterial invasion of the sinus following the common cold.

The pathologic changes occurring in the mucous membrane lining the affected sinus show a wide variation and thus there is a tendency to complex classifications. The changes can be conveniently and simply grouped into four types *acute edematous acute suppurative chronic suppurative and chronic hyperplastic*. In the acute changes, the membrane is hyperemic, edematous and covered with a fibrinous exudate. The degree of these changes can further be classified as catarrhal, mucopurulent and gangrenous. Chronic suppurative changes can be described as edematous, infiltrative, fibrotic, cystic and degenerative. Any combination of these changes may exist in the same diseased membrane. Ulceration and involvement of the underlying bone are not unusual in the chronic suppurative type. This may lead to the formation of fistulous tracts which, by their extension, may cause serious intracranial, ocular or dental complications.

The chronic hyperplastic form is characterized by marked thickening and edema leading to the formation of polypoid masses. This polyp formation is common in the maxillary sinuses and the ethmoid cells. The chronic hyperplastic form is often associated with an allergic rhinitis, but the exact relationship between the two conditions has not yet been finally determined.

The symptoms of infection of any sinus depend upon the sinus involved, and upon the character duration and intensity of the infection. In general, the more acute the infection the more typical the symptoms. Acute sinusitis is accompanied by pain, fever general malaise, a feeling of fullness or pressure in the head or face and serous mucopurulent or purulent discharge from the nose. Tenderness and pain on pressure over the involved sinus are usually present in the acute phase, but seldom in the chronic. The diagnosis is made from the history and the objective clinical findings. If the sinus is draining, the source of the drainage can usually be seen. Pus in the middle meatus indicates involvement of the frontal or maxillary sinuses or of the anterior ethmoid cells, alone or in any combination. Discharge in the superior meatus or in the sphenoethmoid recess, which can be seen by posterior rhinoscopy, is indicative of posterior ethmoid or sphenoid involvement. Al

sisted conservative treatment and repeated irrigations often respond favorably to this treatment.

A more extensive operation is indicated when the entire membrane lining the maxillary sinus must be removed because of marked hyperplastic polypoid or cystic changes or when a chronic infection has produced a thickened fibrous ulcerated, suppurating or degenerated mucous membrane. The operation most frequently used for this purpose is the Caldwell-Luc procedure. An incision through the mucous membrane and periosteum in the sulcus between the gum and the upper lip above the bicuspid and first molar teeth allows the bone of the canine fossa to be exposed. The anterior wall of the sinus is perforated here and the greater part of the wall removed. The lining membrane and any other contents of the cavity are removed and an opening is made through the medial (nasotransverse) wall of the sinus into the inferior meatus of the nose. Epithelialization of the denuded bony walls is accomplished by regrowth of epithelium from the nasal cavity through the antrotomy and the natural openings.

The surgical attack on a diseased frontal sinus may be done by either an intranasal or an external approach. The removal of the anterior end of the middle turbinate followed by irrigation of the sinus through the nasofrontal duct is effective treatment and should be tried before more extensive surgery is decided upon. If this treatment proves to be insufficient, the nasofrontal duct may be enlarged by an intranasal procedure. The middle turbinate is fractured toward the septum to increase the space in the middle meatus. Bone is removed by biting forceps passed upward through the anterior ethmoid cells to the crista nasalis and the floor of the frontal sinus is entered. Ethmoid cells lying behind the nasofrontal duct are removed in the same manner and all irregularities and projecting edges are smoothed. In this procedure, the medial wall of the ethmoid capsule is avoided, since it is a continuation of the cribriform plate fracture of which could allow extension of infection to the cranial cavity.

Frontal sinus disease is often so severe, with fistula formation, intracranial or orbital complications, excessive bone necrosis or mucocele formation that an external approach is required to expose and eradicate the pathologic process. The least extensive external approach consists of removal of part of the anterior wall essentially a trephining,

and insertion of an external drain. Its application is limited. It is used most often in patients with acute fulminating frontal sinusitis with pus under pressure in whom serious consequence may result if drainage is not prompt and adequate.

The Lynch frontoethmoid operation is a safe and effective external approach to the frontal sinus for patients in whom extensive surgical interference is required. The procedure gives excellent exposure not only of the frontal sinus and the anterior ethmoid cells but with modifications of the posterior ethmoid cells and the sphenoid sinus as well. In advanced disease of the frontal sinus the anterior ethmoid cells are almost invariably diseased. They should be removed at the time of the operation on the frontal sinus and in adults these two procedures are almost always combined. Excellent visualization of the surgical area is obtained by this approach. Exposure is obtained by an incision made through the eyebrow which extends medially well down over the nasal process of the superior maxilla. The soft tissues are retracted down and away from the bone. The floor of the frontal sinus and the contents of the sinus cavity are removed. The nasofrontal duct is enlarged and the anterior ethmoid cells are everted. If indicated, the medial wall of the orbit can be entered and the entire ethmoid labyrinth and the middle turbinate removed. This gives access to the sphenoid sinus which can be opened if necessary.

In cases of orbital abscess or when an external fistula from the ethmoid has developed, particularly in children, external drainage of the ethmoid labyrinth alone may be indicated. The incision is the same as for the frontoethmoid operation and care is taken to avoid injury to the lacrimal sac and ducts during elevation and retraction of the soft tissues. The ethmoid sinus is entered through the lamina papyracea and sufficient exposure is obtained for removal of both anterior and posterior cells. External drainage is maintained until all drainage ceases.

Intranasal operations on the ethmoid labyrinth require surgical skill and, because the ethmoid is less frequently operated upon than in former years many surgeons prefer the external approach because of easier visualization and orientation. When the intranasal approach is employed, the middle turbinate may be removed in whole or part to allow better visualization of the operative field. The ethmoid labyrinth is entered through the ethmoid bulla and the cells re-

able. The rapid and more complete clearing of the acute phase results in fewer cases entering the chronic state of infection and the need for surgical interference is of less frequent occurrence. The danger of an early severe complication is abated and in the longer period available for conservative therapy many cases go on to complete resolution that would have required surgery in the past. Antibiotic therapy and chemotherapy are not without danger. Symptoms may be masked, the infection may become indolent and confusion and indecision for the necessity of surgical interference may result.

The development of strains of bacteria resistant to antibiotics is increasing. Antibiotic therapy and chemotherapy alone are not adequate in most cases of chronic sinusitis. The use of these forms of therapy does not supersede the basic principles involved in the treatment of sinus disease. Measures necessary to improve ventilation and establish free drainage of the diseased sinus are still required.

Local treatment during an acute attack consists of shrinking and decongesting the nasal mucosa and turbinates by the application of vasoconstricting agents, such as ephedrine compounds, Adrenalin or cocaine. These are applied to the nasal structures by spraying or placing cotton tampons moistened with the substance in the region of the middle meatus. Mild suction cautiously applied, is helpful. Some form of heat during the acute phase gives comfort to the patient. General supportive measures and symptomatic treatment are given as required. As the acute stage of the infection passes and fever subsides, the sinus may be irrigated if resolution seems delayed. This irrigation can, in many subjects, be done by cannulating the natural opening of the sinus. If the maxillary sinus cannot be cannulated easily through its ostium, the nasointral wall beneath the inferior turbinate can be pierced by an entral trocar and the sinus irrigated. Warm normal saline solution is a suitable irrigating agent. If desired, an antibiotic solution can then be instilled and left in the sinus. Repeated washings may be necessary.

When frontal and maxillary sinusitis co-exist, treatment and irrigation of the maxillary sinus may lessen swelling in the middle meatus sufficiently to allow the frontal sinus to drain normally. Irrigation of the frontal sinus may be performed by cannulating the nasofrontal duct unless bony obstruction or septal deviation prevents the procedure. The

frontal sinus does not often require lavage because the nasofrontal duct opening at the bottom of the sinus promotes dependent drainage.

The sphenoid sinus may be irrigated through the natural ostium, although it may be necessary to first remove an obstructing part of the middle turbinate. If the natural ostium cannot be entered, the anterior wall can be punctured.

The ethmoid sinuses cannot, in actuality, be irrigated, but secretion and exudate can be displaced by the Proetz technique. With the head tilted back, 0.25 per cent ephedrine in saline solution is introduced into the nose and nasopharynx. Intermittent negative pressure is exerted by an instrument devised for the purpose and the ephedrine solution enters the ethmoid cells and the sphenoid sinus displacing any secretion or exudate in those sinuses.

No surgery of the sinuses is attempted during the acute phase of infection unless a complication of some seriousness threatens. In such case, the surgery done is the least necessary to solve the particular problem and extensive or definitive surgery should not be undertaken.

The surgical treatment of inflammatory sinus disease includes those procedures necessary to correct any intranasal deformities which interfere with sinus drainage, and/or opening of the sinus involved for drainage or removal of its contents by either an intra-nasal or external surgical approach. The decision to perform sinus surgery and the choice of the procedure to be done depend on the location and type of involvement and are influenced by the age, physical condition and emotional status of the patient.

The simplest surgical procedure for draining and ventilating the maxillary sinus, excepting sinus irrigation, is the creation of a permanent opening into the sinus through the nasointral wall in the inferior meatus of the nose. By this antrotomy or antrum window operation, an opening of generous size is created. Care is taken to avoid injury to the nasolacrimal canal which opens into the inferior meatus under the anterior end of the inferior turbinate. The sinus cavity can be directly inspected through the opening or a nasoscope can be introduced through the aperture and all areas of the sinus inspected for pathologic changes. It is possible to remove small amounts of diseased tissue through the new opening. Many cases of chronic maxillary sinusitis which have re-

rior portion of the ring. In addition to these well defined lymphoid structures, there is a considerable amount of irregular shaped and sized lymphoid tissue scattered about on the posterior pharyngeal wall, which may undergo rather pronounced hyperplasia under certain conditions.

The *adenoid* is present at birth but under goes atrophy at puberty although remnants are often found in adults. It consists of masses of lymphoid cells supported by a fibrous connective tissue framework. The stroma forms pockets or spaces each one of which is called a lymph follicle. The total mass is divided by deep vertical clefts or fissures, the central one of which ends in a fossa known as the median recess. The clefts in the adenoid mass run so deep that upon removal, the mounds between the clefts can be separated like the leaves of a book.

The *faucial tonsils* are formed of the same type of lymphoid tissue supported by a connective tissue reticulum but where the adenoid contains clefts or folds the faucial tonsil is canalized by epithelial ingrowth to form a complex system of crypts. The openings of the crypts on the surface of the tonsil are seen as small indentations and the crypts collect and hold cellular debris inspissated secretion and dead and viable bacteria. Exudate in these crypts during an acute inflammation gives the surface of the tonsil a characteristic follicular appearance. The tonsil lies in a fossa created by the anterior and posterior pillars and the so-called capsule of the tonsil rests against the superior constrictor muscle. The lingual tonsil has the same characteristic internal structure as do the adenoid and faucial tonsils but does not develop clefts or branching crypts.

The chief functions of the pharynx are concerned with respiration, deglutition, voice resonance and articulation. There is a minor gustatory function owing to the presence of a few scattered taste buds. Because of the location of the orifices of the eustachian tube, the pharynx is also concerned in hearing. It is probable that there is a considerable immunologic function since the lymphoid structures in the pharynx are a part of the reticuloendothelial system and form a first line of defense against invasion of the body by infection. Phagocytic activity of the cells probably plays a part in natural and acquired immunity.

Inflammatory disease of the lymphoid structures of the pharynx, or spread of such infection to contiguous or distant organs, may necessitate surgical therapy to erad-

icate the disease. The faucial tonsil is representative of all the lymphoid structures composing Waldeyer's ring, as concerns reaction of this tissue to infection and for practical purposes the description of acute tonsillitis can stand for acute infection of the adenoid, the lingual tonsil and the scattered pharyngeal follicles. The main difference lies in the symptoms and the complications produced.

Acute tonsillitis is characterized by redness, pain upon swallowing, fever and leukocytosis and general malaise. There is marked hyperemia and swelling of the tissue and small punctate yellow white spots are scattered over the mucous membrane surface. In some subjects pseudomembranes are formed. The bacteria commonly found are streptococci pneumococci and staphylococci. The crypts of the faucial tonsil fill with desquamated epithelium, phagocytes and bacteria and pus may often ooze from the crypt.

The complications of acute tonsillitis include peritonsillar abscess, tonsillar abscess, toxemia, septicemia, endocarditis, arthritis, pneumonia, suppurative otitis media and nephritis. The infection may spread to the neck and produce a deep cervical abscess that can extend to the base of the skull or into the mediastinum.

The treatment of acute tonsillitis is medical and includes bed rest, adequate fluid intake and general supportive and symptomatic treatment. Hot normal saline throat irrigations or gargles are beneficial. The disease is self limited, but severe systemic reaction or threatened complication is probably best treated by the administration of antibiotics or sulfonamides. Topical use of antibiotics is not helpful and may result in sensitizing the patient to the antibiotic so used.

Septic sore throat is an acute primary streptococcal infection of the pharynx and tonsils, and has a much more violent inflammatory reaction than does acute tonsillitis. There is a diffuse fiery redness of the entire pharynx, tonsils and pillars and soft palate, and patches of gray pseudomembrane usually appear. The rapid formation of an inflammatory edematous swelling with extension to the surrounding parts is typical of this disease. The exciting organism is a streptococcus of the beta hemolytic type. The condition sometimes occurs in epidemics and is often milk borne. There is high fever prostration and toxemia. A skin rash may be produced as in scarlet fever but the rash is more variable and less pro-

moved by one or a combination of several methods. The posterior outer and upper angle of the posterior ethmoid cell is a region of extreme hazard because of its anatomic relations.

The intranasal approach to the sphenoid sinus is one of marked surgical risk. Perfect vision of the entire surgical field is necessary at all stages of the operation. The superior wall of the sinus is in close relation with the optic nerve, the pituitary gland, the brain, the lateral wall with the cavernous sinus, and the internal carotid artery. All obstructing conditions of the nasal septum and the nasal cavity must be corrected. The posterior half of the middle turbinate is removed and the posterior ethmoid cells everted. A sphenoid punch forceps or a sphenoid elevator is inserted into the ostium and the opening cautiously enlarged by removing part of the anterior wall. Exploring, curetting or packing the sinus is extremely hazardous.

THE PHARYNX

The pharynx is a musculomembranous tube, wide above and narrow below extending from the basal portion of the occipital bone to the level of the sixth cervical vertebra. It communicates with five cavities: the right and left nasal cavities through the posterior choanae; the right and left eustachian tubes, the oral cavity, the vestibule of the larynx, and the esophagus. The musculature of the pharynx consists of three pairs of constrictor muscles: the superior, middle and inferior; and two pairs of elevator muscles: the stylopharyngeus and the palatopharyngeus. The constrictors are outer circular muscles and the elevators are longitudinal, inner muscles: a reverse order of arrangement to that found in the esophagus.

The nasopharynx is that portion of the pharynx above the level of the soft palate; it is immobile except for the soft palate which, in contraction, forms a floor for the space. The oropharynx extends from the inferior border of the soft palate to the lingual surface of the epiglottis. The hypopharynx extends from the tip of the epiglottis to the esophagus. The anterosuperior part of the hypopharynx is often designated as the laryngopharynx.

The eustachian tubes open into the anterolateral wall of the nasopharynx just behind the posterior ends of the inferior nasal turbinates. Above and behind the tubal orifices the wall expands to form a cul-de-sac, the pharyngeal recess or Rosenmüller's fossa.

The wall here is in close relationship to the internal carotid artery.

The hypophysis cerebri develops as an evagination from the nasopharynx at a point in the midline high on the posterior wall. Rarely, a vestige of the stalk remains and forms a cyst, the pharyngeal bursa, which extends in the median plane to the pharyngeal tubercle of the occipital bone and lies under the mucous membrane. The adenoid develops in this area and the pharyngeal bursa, when it exists, lies immediately above and behind the median recess of the adenoid. When an adenoid remnant persists in an adult, the central cleft of the adenoid, which leads into the median recess of that structure, is often seen as a slitlike opening. This cleft may harbor infection and an abscess of the median recess can develop. This is known as *Thornwaldt's disease*. If a pharyngeal bursa exists and becomes infected, it is often erroneously diagnosed as *Thornwaldt's disease* and the two are often difficult to differentiate by examination. Since abscess of the median recess or *Thornwaldt's disease* lies in adenoid tissue, it can be opened by a probe, but the pharyngeal bursa, which lies behind the pharyngeal mucous membrane, requires incision to effect drainage.

The oropharynx opens into the oral cavity and is bounded above by the soft palate and laterally by the glossopalatine and pharyngopalatine muscles, constituting respectively the anterior and posterior pillars between which lie the palatine or faucial tonsils. The lingual tonsil is a collection of lymphoid tissue situated on the base of the tongue and is bisected by the glossoepiglottic ligament.

The hypopharynx, that portion of the pharynx below the tip of the epiglottis, enlarges laterally into the pyriform sinuses. During deglutition, the larynx is raised and closed and the bolus is directed into the pyriform sinuses and on into the esophagus. The anterosuperior portion of the hypopharynx communicates with the vestibule of the larynx and is designated as the laryngopharynx.

The pharynx contains masses of lymphoid tissue so arranged as to form a ring about the pharynx. These masses form the combined structure known as *Waldeyer's ring*. The adenoid, or pharyngeal tonsil in the nasopharynx forms the posterior portion; the palatine, or faucial tonsil in the oropharynx forms the lateral part, and the lingual tonsil on the base of the tongue forms the ante-

occur in adults but is considered to be a disease of infancy and early childhood. Occurrence is usually preceded by an upper respiratory infection and the abscess is produced by infection and breaking down of a prevertebral lymph node. It can be of tuberculous origin. The symptomatology is that of an upper respiratory infection followed by dysphagia and inspiratory dyspnea. Nursing or feeding is difficult and the voice is muffled or changed in quality. The location of the abscess may be high and easily visible above the base of the tongue or so low as to be difficult to palpate. Increasing dyspnea is a serious symptom and is due to pressure against the larynx and trachea by an abscess deep in the pharynx. This cause of respiratory obstruction in an infant must always be kept in mind. If the diagnosis is in doubt, a lateral x-ray view of the cervical region will show the location and size of the abscess, if one exists.

The prognosis is good if the condition is recognized. The abscess is incised and drained with the child on his back with the head extended and the shoulders elevated. The tongue is lifted up to expose the posterior pharyngeal wall and to insure an adequate airway. A mouth gag should not be used, since forcible opening of the jaw may rupture the abscess and result in a fatality. With good illumination and under direct vision, the abscess is incised and the contents are aspirated. Incision at the lowest point of the abscess may prevent it from burrowing lower in the pharynx. One draining usually suffices although the wound may require reopening for two or three days.

The pharynx is at times involved by specific infections, such as syphilis and tuberculosis. Syphilis may be seen in any of its three phases. Tuberculosis involves the pharynx and its structures in both the acute and chronic forms. The first symptoms of leukemia, granulocytopenia, infectious mononucleosis, pernicious anemia and other blood dyscrasias often appear in the mouth or pharynx.

The pharynx is often involved by carcinoma. The primary sites occur in the nasopharynx, the tonsil, the lateral wall of the hypopharynx and in the laryngopharynx. Other tumors found in the pharynx include sarcoma, fibroma, papilloma, polyps, cyst and angioma. The pharynx is also subject to various sensory and motor neuroses.

Most foreign bodies which enter the pharynx pass on to enter the esophagus or the larynx and tracheobronchial tree. Those

which lodge in the pharynx proper are usually sharp-pointed objects such as pins, tacks and small fish bones. These usually lodge in the faucial tonsil or the tonsil fossa, the lingual tonsil on the base of the tongue or in the lateral pharyngeal wall behind the posterior pillar. Occasionally one will lodge in the vallecula between the base of tongue and the anterior surface of the epiglottis. Careful search will reveal them and they can usually be grasped by an appropriate forceps and removed. Caution and skill are necessary in order that they are not dislodged and converted into an esophageal, laryngeal or pulmonary foreign body.

Pulsion diverticulum has its origin from the pharynx but is often referred to as of esophageal origin because of the symptoms it produces.

The surgical operation most frequently done on the pharynx is removal of the tonsils and adenoids. The diversity and severity of the complications produced by acute and chronic disease of the tonsils make the removal of these diseased organs of great benefit. Indiscriminate or incomplete removal by inadequately trained surgeons has no doubt caused harm to some individuals, but, when the proper indications exist, the operation can be recommended as being as necessary to the health and well-being of the individual as is the removal of a diseased organ elsewhere in the body.

The indications for tonsillectomy and adenoidectomy are usually quite evident. In children, gross hypertrophy is sometimes so great as to prevent nasal respiration and the tonsils meet in the midline producing an actual block against the passage of food. The child is a poor eater and the obstruction to respiration when prolonged, produces a typical "adenoid facies." The child tends to be dull and apathetic, nourishment is poor and general growth and development are delayed. Marked hypertrophy can develop without the stimulus of infection and constitutes a positive indication for surgical intervention.

Recurrent otitis media in children is most often secondary to disease of the adenoids and tonsils and the removal of these tissues has more chance of preventing and eradicating this complication than has any other single procedure. Control of the otitis media minimizes the incidence of future deafness and sharply reduces the chance of mastoid disease and its complications.

Persistent enlargement of the cervical glands which drain the tonsillar area is often

tracted. Since this infection is violent, and more apt to produce systemic complications than is acute tonsillitis, appropriate antibiotic and sulfonamide therapy should be given early and continued, if possible, until the patient is symptom free.

Acute infection caused by fusiform bacilli and spirochetes in combination frequently affects the pharynx and is known as *Plaut-Vincent's angina*, or *trench mouth* when there is involvement of the mouth and gums. This infection rather typically produces ulcerations of the mucous membrane and pseudomembrane is often formed. Despite the ulcerations and the presence of pseudomembrane, the surrounding tissues usually do not show as severe involvement as occurs in acute bacterial pharyngitis. Examination of smears from the infected areas will usually establish the diagnosis.

Diphtheritic tonsillopharyngitis does not now occur frequently but, when it does occur is most often seen by the pediatrician. It must be suspected in any pharyngitis with membrane formation and smears should be made for microscopic study. The onset may be mild with only slight soreness of the throat or the onset may be sudden with severe toxemia and prostration. Swollen, tender cervical lymph glands are usually found. The breath has a foul, peculiar odor. The throat may be red and no membrane present, but usually there are patches of adherent gray yellow or greenish membrane that leave a bleeding, raw surface when removed, and which quickly re-form. Differentiating a true diphtheritic membrane from the pseudomembrane produced by the coccal forms of bacteria and by fusiform-spirochete infection may be difficult, particularly if there is ulceration under the pseudomembrane. The treatment of diphtheria is medical except in those instances when membrane forms in the larynx, producing obstruction that may require tracheotomy for relief or when the tonsils may require removal to eradicate a "carrier" state of infection.

Peritonsillar abscess occurs as a complication of acute tonsillitis. Several days after the onset of the acute tonsil infection, and frequently when the tonsillitis seems to be clearing, there is a return of pain and inflammation more severe than the original. Swallowing becomes difficult or impossible; the jaw can be opened only slightly and with great pain and speech becomes muffled and thick. Examination reveals a unilateral swelling near the upper pole of the tonsil involving the anterior pillar and the soft palate.

The tissues are shiny fiery red and edematous. The uvula may be pushed to the opposite side and the usual landmarks of the area are obscured by the swelling. Fever may be absent. The abscess is produced by extension of the acute infection into the loose peritonsillar tissue first as cellulitis and progressing to abscess formation. Administration of antibiotics in the cellulitis phase may prevent abscess development, but the complication not infrequently occurs during antibiotic or sulfonamide therapy for acute tonsillitis. During the stage of cellulitis, throat irrigations with hot normal saline solution should be given at frequent intervals. Incision at this stage is not wise but, when an abscess is formed, surgical drainage is required. An incision is made through the anterior pillar or through the supratonsillar fossa until pus is reached. A blunt forceps is introduced through the incision to the abscess cavity and forcibly opened to enlarge the drainage tract. Relief is so immediate that the patient does not resent the momentary pain this procedure causes. Care is taken that the patient does not aspirate the pus. Peritonsillar abscess, or quinsy sore throat, does not often occur in children.

An abscess within the substance of the tonsil may follow or complicate acute tonsillitis but is of infrequent occurrence. In the acute process, the patient is toxic, the tonsil is large, hyperemic and firm but not tender to touch. The surrounding tissues are not markedly involved. The treatment is incision and drainage. A retention cyst of the tonsil occurs more frequently than an acute abscess and is found in a superficial area. It is probably produced by the closing off of a crypt.

A *lateral pharyngeal abscess* may be produced by acute tonsillitis or a peritonsillar abscess. The abscess forms external to the superior constrictor muscle, just lateral or posterior to the faucial tonsil. If posterior to the tonsil, it may bulge into the pharynx and be drained by incision through the pharyngeal mucosa. If lateral to the tonsil, removal of the tonsil will give access for draining through the pharynx. External drainage through the neck may be necessary. Such abscesses can extend along fascial planes to the base of the skull or to the mediastinum. Extension of a lateral pharyngeal abscess may require extensive external drainage of either the pharyngomaxillary fossa or the neck spaces.

Retropharyngeal abscess is a collection of pus behind the posterior pharyngeal wall anterior to the prevertebral fascia. It may

teeth, bits of tonsil or adenoid tissue, pieces of instruments, sponges and blood. General complications include thrombophlebitis and embolism, septicemia, otitis media and its complications, atelectasis, lung abscess, exacerbatation of organic disease, meningitis and death.

TUMORS

The nose, nasal sinuses and the nasopharynx may be involved by benign or malignant tumors arising from any of the types of tissue found in, or going to make up these structures. Included, therefore, are tumors derived from epithelium, cartilage, bone, connective tissue, lymphoid elements, endothelium, pigment cells and neural tissue. The same areas may be invaded by tumors extending from adjacent structures and, in rare instances, be the site of metastases from distant primary lesions.

Although the majority of the tumors encountered in the nose, sinuses and the nasopharynx are benign, any tumor in this area must be considered as malignant until proved otherwise. The gross appearance of the lesion can be very misleading. Malignant lesions frequently simulate the appearance of the simplest benign tumor common to the area. Certain tumors, although histologically benign, have malignant characteristics in that they erode or invade adjacent structures, produce destruction and deformity and result, ultimately, in death. The treatment necessary to eradicate a tumor of this type approaches or equals that required in the attack on a malignant one. These facts emphasize the importance of accurate diagnosis. Biopsy of the tissues is the only means of achieving proper identification and classification. Repeat biopsies are necessary when clinical judgment is not satisfied by the first or subsequent tissue reports. The specimen of tissue removed should be generous.

The symptoms of tumor in the nose, sinuses or the nasopharynx depend largely on the location, size and growth characteristics of the neoplasm. The most usual symptoms include nasal obstruction, nasal discharge, pain referred to the teeth and various areas of the head, paresthesia of the cheek, bloody discharge or hemorrhage from the nose or nasopharynx, eye or visual disturbances and external deformity. Most of the above symptoms depend on erosion, pressure by or invasion of the tumor and are, unfortunately, late symptoms. Although many of these symptoms occur after the tumor has reached considerable size or extent, none of them

gives any hint of the pathologic condition responsible for symptoms which are of such common and widespread occurrence. Neither are the symptoms helpful in determining the site of origin, since primary nasal tumors invade the sinuses and tumors arising in the sinuses may cause no symptoms until they encroach on the nasal cavity. Tumors of the maxillary, frontal and ethmoid sinuses may invade the orbit and produce visual disturbances. Sphenoid sinus neoplasms can also produce visual symptoms by pressure on the optic nerve or by invasion of the cranial vault.

BENIGN TUMORS OF THE NASAL CAVITY AND PARANASAL SINUSES

By far the most commonly encountered benign tumor of the nasal cavity and of the paranasal sinuses is the mucous polyp. These are usually multiple but they occasionally occur as a solitary growth. A question remains as to whether these are true tumors or only edematous hypertrophies of the nasal or sinus mucosa resulting from frequent congestions and a rarefying osteitis of the underlying bone. The maxillary and ethmoid sinuses are most often involved in this type of reaction. Polyps from these sinuses may present in the nasal cavity or even in the nasopharynx, attached to their point of origin by a long narrow pedicle.

A polyp has the appearance of a smooth, rounded, grayish-pink, slimy tumor. It is covered by respiratory epithelium and many eosinophils and blood vessels are found in the loose, edematous stroma. Polyps are classified as edematous, fibrous, angiectatic, glandular or cystic, depending on the relative amounts of these elements making up the stroma. The appearance and consistency of the polyp vary with the type. The edematous polyp is the most common.

The symptoms produced by nasal polyps are nasal obstruction, profuse watery to purulent discharge, sneezing and loss of the sense of smell. The severity of the symptoms depends on the number and size of the polyps. Headache as a result of the severe nasal congestion is common.

The treatment of nasal polyps is surgical. Medical treatment alone is not effective. In all cases, infection must be cleared, allergies controlled or eliminated and other irritants avoided. The use of corticosteroids appears to be of benefit in some cases but, since they are not universally helpful and their use is not without certain hazards, they cannot be recommended for routine treatment. Re-

an indication of active tonsil infection, which may require tonsillectomy. The occurrence of a peritonsillar abscess is an absolute indication for tonsillectomy since the abscess is prone to recur with subsequent attacks of tonsillitis and because the potentials are so serious. The crypts in some tonsils become enlarged and contain so much debris and bacteria that, while not producing marked infection of the tonsil, they are responsible for foul breath and local irritation. Tonsillectomy is the only sure means of correcting such a condition.

In addition to these rather clear-cut indications are many lesser indications which vary with the feelings and beliefs of the surgeon, pediatrician or internist concerned. They are usually based on the effect of removal of diseased tonsils or tonsils and adenoids on respiratory tract and lung disease, rheumatic fever, arthritis, pyelitis, nephritis and any other conditions to which the tonsil infection is felt to be a contributory factor.

Recently the correlation between removal of the tonsils and adenoids and increased susceptibility to poliomyelitis has been under investigation. Statistical evidence shows the attack rate of poliomyelitis following adenoidectomy and tonsillectomy to be markedly less than the attack rate in the general population. There is evidence that if poliomyelitis develops soon after removal of the tonsils and adenoids there is a greater chance of it being the bulbar type. The development of a successful vaccine has made these arguments of little importance. Despite the vaccine the operation should not be done when the incidence of poliomyelitis in the community is high.

Conscientiously to recommend surgical removal of the tonsils as a procedure which will benefit the patient is a decision arrived at after obtaining a careful history and a thorough physical examination to exclude the possibility of other diseases which may be responsible for the symptoms and after a period of observation of the course of the tonsil disease. The physical appearance of the tonsil, in most cases, is the least important factor in making the decision.

The technique of surgical removal of the tonsils and adenoids has infinite variations and modifications, but, in general, these fall into two types: that of dissection and snare and the guillotine method. There is a choice as to the positioning of the patient during the operation each having proponents for its superiority.

In the dissection and snare method, the tonsil is grasped by a tonsil forceps and dissected away from the peritonsillar tissues by blunt or sharp dissection with knives or scissors or other instruments devised for this purpose. After being freed superiorly and from the anterior and posterior pillars a cold wire snare is passed over the tonsil and it is severed at the base by closing the snare. The dissection is against the so-called capsule of the tonsil and avoids the aponeurosis covering the fibers of the constrictor muscles.

The guillotine method utilizes an instrument incorporating a ring which can be closed by a blade or cold wire snare. The tonsil is pushed through the ring, held by a blunt hemostatic blade and then severed by a sharp blade or the tonsil is engaged by the snare as it is pushed through the ring and then severed from its attachments by slowly closing the snare. Proponents of the guillotine method cite lessened bleeding and less trauma to the surrounding tissues as its main advantages.

The adenoid is removed by the use of an adenotome or is scraped from its attachments by special curets. The adenotome is a boxlike instrument designed to fit the nasopharynx, the lower side of the adenotome can be closed by a flexible cutting blade. The adenotome is placed over the adenoid with the blade opened, pressed down firmly and the adenoid severed by closing the blade. The closed instrument prevents pieces of adenoid tissue from entering the lower airways. Lateral extensions and small remnants can be removed by curetting, or by using punch forceps.

During the performance of the operation one of the greatest responsibilities of the surgeon is the maintenance of a clear airway at all times. Another as important, and interrelated, is the control of hemorrhage. Inspired blood is one of the frequent causes of embarrassed respiration. A free and clear airway at all times during the operation is of primary importance in preventing the occasional but too frequent, surgical death. It is for this purpose that intubation of the larynx to administer the anesthesia has been recommended.

The complications of tonsillectomy and adenoidectomy include hemorrhage at the time of the operation or shortly after secondary hemorrhage many hours or days postoperatively, edema of the palate and uvula, acute inflammation with delayed healing or more serious complication pulmonary foreign body from the inspiration of

teeth bits of tonsil or adenoid tissue pieces of instruments sponges and blood. General complications include thrombophlebitis and embolism septicemia, otitis media and its complications, atelectasis lung abscess, exacerbation of organic disease meningitis and death.

TUMORS

The nose nasal sinuses and the nasopharynx may be involved by benign or malignant tumors arising from any of the types of tissue found in or going to make up these structures. Included therefore are tumors derived from epithelium cartilage bone connective tissue lymphoid elements endothelium pigment cells and neural tissue. The same areas may be invaded by tumors extending from adjacent structures and, in rare instances, be the site of metastases from distant primary lesions.

Although the majority of the tumors encountered in the nose sinuses and the nasopharynx are benign, any tumor in this area must be considered as malignant until proved otherwise. The gross appearance of the lesion can be very misleading. Malignant lesions frequently simulate the appearance of the simplest benign tumor common to the area. Certain tumors, although histologically benign have malignant characteristics in that they erode or invade adjacent structures, produce destruction and deformity and result, ultimately in death. The treatment necessary to eradicate a tumor of this type approaches or equals that required in the attack on a malignant one. These facts emphasize the importance of accurate diagnosis. Biopsy of the tissues is the only means of achieving proper identification and classification. Repeat biopsies are necessary when clinical judgment is not satisfied by the first or subsequent tissue reports. The specimen of tissue removed should be generous.

The symptoms of tumor in the nose, sinuses or the nasopharynx depend largely on the location size and growth characteristics of the neoplasm. The most usual symptoms include nasal obstruction, nasal discharge pain referred to the teeth and various areas of the head, paresthesia of the cheek, bloody discharge or hemorrhage from the nose or nasopharynx, eye or visual disturbances and external deformity. Most of the above symptoms depend on erosion, pressure by or invasion of the tumor and are, unfortunately late symptoms. Although many of these symptoms occur after the tumor has reached considerable size or extent, none of them

gives any hint of the pathologic condition responsible for symptoms which are of such common and widespread occurrence. Neither are the symptoms helpful in determining the site of origin since primary nasal tumors invade the sinuses and tumors arising in the sinuses may cause no symptoms until they encroach on the nasal cavity. Tumors of the maxillary frontal and ethmoid sinuses may invade the orbit and produce visual disturbances. Sphenoid sinus neoplasms can also produce visual symptoms by pressure on the optic nerve or by invasion of the cranial vault.

BENIGN TUMORS OF THE NASAL CAVITY AND PARANASAL SINUSES

By far the most commonly encountered benign tumor of the nasal cavity and of the paranasal sinuses is the mucous polyp. These are usually multiple but they occasionally occur as a solitary growth. A question remains as to whether these are true tumors or only edematous hypertrophies of the nasal or sinus mucosa resulting from frequent congestions and a rarefying osteitis of the underlying bone. The maxillary and ethmoid sinuses are most often involved in this type of reaction. Polyps from these sinuses may present in the nasal cavity or even in the nasopharynx, attached to their point of origin by a long narrow pedicle.

A polyp has the appearance of a smooth, rounded, grayish-pink, slimy tumor. It is covered by respiratory epithelium and many eosinophils and blood vessels are found in the loose edematous stroma. Polyps are classified as edematous, fibrous angiectatic glandular or cystic, depending on the relative amounts of these elements making up the stroma. The appearance and consistency of the polyp vary with the type. The edematous polyp is the most common.

The symptoms produced by nasal polyps are nasal obstruction, profuse watery to purulent discharge sneezing and loss of the sense of smell. The severity of the symptoms depends on the number and size of the polyps. Headache as a result of the severe nasal congestion is common.

The treatment of nasal polyps is surgical. Medical treatment alone is not effective. In all cases infection must be cleared, allergies controlled or eliminated and other irritants avoided. The use of corticosteroids appears to be of benefit in some cases, but, since they are not universally helpful and their use is not without certain hazards, they cannot be recommended for routine treatment. Ro-

removal of the polyps by a cold wire snare under light topical anesthesia gives immediate relief from the symptoms. In severe or long-standing involvement the sense of smell may remain impaired or lost. Removal of polyps usually needs to be repeated at varying intervals of time. Severe involvement of the sinuses may require extensive sinus surgery with complete removal of the diseased membrane.

The importance of the polyp as a nasal and sinus tumor does not rest in its frequency of occurrence but in the tendency of malignant conditions to induce a polypoid tissue response and stimulate the polyp in appearance. All nasal polyps removed surgically should be submitted to microscopic examination. Malignancy is so often discovered in supposedly simple nasal polyps as to not only warrant, but require, such examination.

Primary fibroma, osteoma and chondroma of the nasal cavity are not common. Small papillomas which are actually cutaneous warts occur at the mucocutaneous junction in the nasal vestibule. Their location leads to constant irritation and they should be removed by electrosurgery. Papilloma durum, which is a true papilloma, occurring in the nasal cavity requires differentiation from a papillary carcinoma. Complete removal is required and the tissue must be examined microscopically for malignant changes. If these papillomas are widespread, even though they are benign, their removal should be followed by irradiation.

Syphilis and tuberculosis produce nasal lesions which require biopsy for identification.

Rhinoscleroma, a granulomatous disease rare in this country produces nasal tumors.

Extreme hypertrophy of a turbinate or a cystic or pneumatized turbinal bone may cause an obstructing mass which requires differentiation from a neoplasm.

The benign tumors common to the paranasal sinuses are the mucous polyp, osteoma, fibroma, angioma, papilloma, cyst and mucocele. The pathology of these tumors is identical no matter which sinus is affected. The symptoms depend on the size, primary location and the adjacent structures involved.

Most benign tumors except nasal polyps which tend to occur bilaterally affect only one nasal cavity. Despite this, a growth obstructing one nasal cavity requires a provisional diagnosis of malignancy because unilateral involvement is a characteristic of carcinoma of the nose and the accessory sinuses.

Mucous polyps, most common to the ethmoid and maxillary sinuses, may block sinus drainage and result in suppuration.

If these tumors are present in the nasal cavity they produce the same symptoms as nasal polyps. Osteoma does not produce symptoms unless the tumor occludes the sinus ostium or becomes large enough to produce pressure on a sinus wall. The frontal sinus is a very common site for osteoma. The ethmoid and maxillary sinuses seem to be less often involved. Papilloma durum requires thorough removal and tissue examination. Cysts other than those produced by degenerated polyps are usually of dental origin. These cysts usually grow rapidly and may erode a sinus wall or produce deformity of the hard palate or face. The surgical approach necessary for their removal depends on their location.

The frontal sinus, and less often the ethmoid sinus, may become involved by a mucocele. A mucocele is not a true tumor but is a retention cyst produced by a closure of the sinus ostium with the accumulation of secretions and exudate within the mucosal lining of the sinus cavity. Gradual distention leads to erosion of the sinus wall and, in both the frontal and ethmoid location, the orbit may be encroached upon with displacement of the eyeball downward and outward. This produces diplopia which may be the only symptom from which the individual seeks relief. The treatment is surgical removal of the diseased tissue by an external approach and re-establishment of normal sinus drainage.

The sphenoid sinus is most often affected by mucous polyps or by an osteoma. These may produce symptoms of suppuration or cause pressure which can result in optic neuritis or even produce exophthalmos.

MALIGNANT TUMORS OF THE NASAL CAVITY AND THE PARANASAL SINUSES

Malignant conditions of the nasal cavity and of the accessory nasal sinuses should always be considered to exist as a single entity. The characteristics of the disease make it highly improbable that one structure is involved without some involvement of the other and the successful eradication of the tumor may depend on this concept. For all practical purposes, malignant disease of the nose and sinuses implies carcinoma. All other varieties of malignant tumors found in these areas constitute a very small percentage of the total as little as 1 per cent in some series of cases. The sarcomas constitute

the majority of the small percentage not carcinomas.

The symptoms produced by malignancy occurring in this area are not unusual or much different from those produced by a number of benign nasal sinus or dental diseases. The basic symptoms are obstruction to nasal breathing, nasal discharge and nasal bleeding. In addition there may be headache or pain referred to various areas of the head, face or to the teeth. Sinus suppuration is common and may have led to repeated sinus lavage. Not infrequently there have been multiple tooth extractions in an attempt to relieve the dental pain. None of the symptoms can be considered to be early since each depends on the enlargement of the tumor or on the erosion or invasion of other structures by the tumor. Fortunately most carcinomas of these areas develop slowly and metastasize infrequently or late in their growth. The symptoms are certainly no indication of the extent of involvement or of the primary site of the lesion.

In sarcomatous involvement of the nose or sinuses, the prognosis depends upon the type of the tumor and the extent of involvement. Biopsy may show it to be a spindle cell large or small round cell or osteoblastic or lymphosarcoma, melanosarcoma, fibrosarcoma, chondrosarcoma or rhabdomyosarcoma. The only effective treatment is early and complete removal. Some of the sarcomas are highly radiosensitive and respond so well to irradiation that it is the treatment of choice. With the more radioresistant sarcomas, the best results are obtained by a combination of surgery and irradiation.

Just as the term malignant disease of the nose and paranasal sinuses implies carcinoma, carcinoma of the sinuses implies involvement of the maxillary or ethmoid sinuses. Primary malignant growth in the frontal sinus is rare. Carcinoma of the sphenoid sinus is also rare and, when it does occur is almost always found in an advanced state with extension to the sella turcica or into the cranial vault. The location of the sphenoid sinus makes the surgical approach in this type of involvement extremely hazardous and impractical and irradiation of this area has not proved a satisfactory mode of treatment. Carcinoma of the ethmoid sinus tends to involve surrounding structures early in its development. This characteristic compromises the chance of recovery no matter what the mode of treatment.

The diagnosis of carcinoma of the sinuses is too often made in the late stages of the

disease. This delay in diagnosis may be due to symptoms that direct attention to other areas or that resemble symptoms of benign disease. It may be entirely due to the late development of the symptoms or to the failure of the individual to present himself for care. In these cases the tumor may have produced external deformity or metastases. Diagnosis by x-ray examination is often made only after bone destruction has occurred. Any tumor found on x-ray examination must be suspect and studied by follow-up films. Of all abnormal densities in sinus x-rays only osteoma produces a density greater than that produced by carcinoma. Tomographic studies are helpful in determining the extent of tumors which have spread beyond their primary location. Exploration and biopsy should be done immediately in any case when the diagnosis is in doubt. Biopsy is the only means of differentiating the benign from the malignant tumor. Repeated biopsies with specimens being taken from several representative areas, must be the procedure when clinical judgment is not satisfied by a negative report. The ubiquitous mucous polyp must not be overlooked as possibly malignant.

The most commonly encountered carcinomas of the nose and paranasal sinuses are of the squamous cell variety which vary from slow growing to highly anaplastic types. An occasional adenocarcinoma is encountered.

The successful treatment of carcinoma of any organ is never easy but when the growth occurs in the sinuses it is particularly difficult because of the preponderance of bone and the relative scarcity of soft tissue. While the bone does act as a barrier to extension of the neoplasm it also quickly becomes infiltrated with tumor. Because of the great variation in the manner in which these tumors develop grow and invade, there can be no real standardization of treatment. The proper attack must be decided in each case and is best a team decision involving surgeon, x-ray therapist, pathologist and internist. The extent and type of the malignancy and the physical and emotional status of the patient are all factors which influence the ultimate choice of therapy. Treatment may be surgery alone, surgery preceded or followed by irradiation, or irradiation therapy alone. Surgical resection should not be attempted when there is no reasonable chance for complete removal of the tumor. If the case is clearly inoperable, nothing but palliative measures should be undertaken.

Contraindications to surgery vary but the commonly accepted ones are destruction of the base of the skull, extensive bilateral involvement, involvement of the nasopharynx and distant metastases. Irradiation therapy alone will give better results in certain anaplastic radiosensitive tumors than can be obtained by surgery and should be the choice in such instances. Surgical removal, sometimes preceded and always followed by irradiation gives the best results in the majority of cases.

The ideal surgery of any malignant growth is wide removal through normal tissue. This requirement can almost never be met in the surgical treatment of malignant disease of the sinuses. The approach may involve the site of the tumor and removal of the growth requires a certain amount of scraping and curetting. There is little doubt that these methods disseminate the cancer cells and may produce distant metastases. The use of electrosurgery (electrodisssection and electrocoagulation) seals off blood vessels and lymphatics and should minimize the spread of cancer cells. The use of electrosurgery has made it possible to attack growths impossible to remove successfully by classical or scalpel surgery. In sinus cancer surgery many surgeons believe it should be used to deal with all soft tissue excepting the skin incision. Thorough diathermy coagulation of the postoperative cavity appears to increase the chances of complete eradication of the malignant growth. Postoperative irradiation, either by radium or external x-ray, should destroy disseminated cells and any microscopic remnants of tumor.

The first step in the treatment of malignant neoplasm of the sinus is surgical exposure of the lesion by whatever approach is necessary to obtain representative tissue for biopsy. The next consideration is the surgery necessary to provide drainage. Superimposed infection must be controlled prior to either definitive surgery or irradiation. Drainage operations should be done even if no infection exists but treatment is to be by irradiation alone. This allows the escape of degenerated and sloughing tumor tissue and provides a window through which the response of the tumor to the therapy can be observed. It also permits the early detection of any recurrence. The common sites for these openings into the maxillary sinus are through the hard palate or sublabially through the canine fossa.

The surgical attack should aim at removal of the tumor with a margin of healthy tis-

sue. This demands an external approach with wide exposure and the sacrifice of normal structures if necessary. The surgical approaches described for the treatment of sinus suppuration and for exposure of benign tumors are completely inadequate in the treatment of malignant ones.

The surgical exposure should be made in the area of greatest involvement. For example if the orbit is involved, the contents of the orbit should be exenterated. This allows easy access to the frontal sinus above, the maxillary sinus below and the ethmoid and sphenoid sinus and the nasal cavity medially. If the hard palate is involved, removal of half of the hard palate gives easy access to the antrum and all other sinuses on that side. Even when it is not involved, many surgeons remove the hard palate as a routine part of the surgery. Irradiation should immediately follow surgery. External irradiation by x ray is preferred in this country but radium may be introduced into the tumor area if for any reason this should be preferable.

Irradiation should never be selected as the method of treatment with the idea that it is easier for the patient than surgery. It is very often the opposite. Irradiation produces reactions which are both painful and disfiguring. Radiation in cancerocidal doses results in definite morbidity. The pain and discomfort of radiation reaction, sloughing of the tumor and secondary infection can disable the patient for a long time. The pain of bone necrosis can be excruciating and may persist for months or years before sequestration finally takes place.

The final choice of the mode of treatment is not an easy decision and subsequent events may prove it not to have been the best. Extremely radical surgery may restore a person to an active, productive life, but it may also result in disfigurement which makes him a social outcast. Despite the probability of the patient surviving extensive surgery it is the responsibility of the surgeon to choose the least surgery that will be effective. The surgery chosen, however, must be adequate to eradicate the malignant growth. There is no partial cure of cancer.

BENIGN AND MALIGNANT TUMORS OF THE NASOPHARYNX

Fibroma of the nasopharynx is an extremely vascular tumor found most often in young males between the ages of five and twenty. It is not of frequent occurrence. The tumor is so vascular that it should properly

be termed an angiofibroma. Though it is benign, it can present the most difficult surgical problems in its removal.

The tumor grows rapidly filling the post nasal space. It may then extend into the nasal cavity or by finger like extensions invade any of the sinuses or the cranial vault. These projections frequently attach at points of contact making it appear that the tumor has multiple points of origin. It is composed chiefly of connective tissue with many thin walled blood vessels which have almost no contractile layer. There is no true capsule. It arises in either a broad or pedunculated base from the fibrous layer of the prevertebral fascia and grows in the line of least resistance.

The symptoms are nasal obstruction and hemorrhage. The bleeding may be severe and persistent. If the tumor invades the skull mental or ocular symptoms may result. Extension into the sinuses and the skull may separate the superior maxillary bones and produce exophthalmos resulting in a "frog face" deformity. A striking characteristic of these tumors is their tendency to disappear spontaneously in early adult life.

Differentiation from the adenoid mass or from a mucous polyp does not present much of a problem if the examination is careful. Sarcoma presents more of a problem and the microscopic differentiation is not always easy or certain.

Removal of these fibromas by classical surgical methods is difficult and hazardous. They cannot be avulsed because of their attachment to fascia or periosteum. Irradiation makes the tumor smaller and more accessible and, perhaps, lessens the tendency to hemorrhage. The introduction of electro-surgery has somewhat simplified the surgical problems posed by these tumors and the combination of preoperative irradiation and electro-surgery is the method of choice when surgery is necessary.

Although various types of sarcomas occur in the nasopharynx, the malignant growth most often encountered is the epidermoid carcinoma. Squamous cell, slow growing or highly anaplastic types and adenocarcinomas are all represented. The incidence of

slow growing and anaplastic types is about equal.

The symptoms depend on the size and location of the tumor. There may be no recognizable symptoms before the tumor reaches a large size or produces metastases. If the lesion is large enough there is nasal obstruction. If the growth encroaches on the eustachian tube, there may be hearing loss and middle ear symptoms. Blood-tinged postnasal drainage is more common than frank hemorrhage. Pain referred to the ear, supraorbital or occipital areas is common. Extension to the base of the skull may produce involvement of cranial nerves most often of the sixth, ninth, tenth, eleventh or twelfth. Metastases, eustachian tube obstruction, middle ear infection or cranial nerve involvement may produce the symptom leading to discovery of the primary lesion. The nasopharyngeal tumor may be so small as to escape detection unless the examination is exceedingly thorough. A remnant of adenoid tissue often persists in the adult as a mass in the midline of the vault of the nasopharynx. Biopsy will differentiate it from a malignant lesion.

The treatment of malignant neoplasms of the nasopharynx, whether carcinoma or sarcoma, is irradiation. Surgery, other than for biopsy, should not be attempted. One of the contraindications to surgical treatment of malignant tumors of the nose and sinuses is involvement of the nasopharynx and this applies equally when the growth is primary in the area.

READING REFERENCES

- Ballenger H. C., and Ballenger J. J. *Textbook of Diseases of the Nose, Throat and Ear* 10th ed. Philadelphia, Lea & Febiger 1957.
- Boke, Lawrence R.: *Fundamentals of Otolaryngology* 3rd ed. Philadelphia, W. B. Saunders Company 1959.
- Jackson, Chevalier and Jackson, C. L. *Diseases of the Nose, Throat and Ear* 2nd ed. Philadelphia, W. B. Saunders Company 1950.
- Lederer, Francis L. *Diseases of the Ear, Nose and Throat*, 8th ed. Philadelphia, F. A. Davis Company 1952.
- Proetz, Arthur W. *Essays on Applied Physiology of the Nose* 2nd ed. St. Louis, Annals Publishing Company 1953.

The Ears

By THEODORE E. WALSH, M.D.

THEODORE EDWIN WALSH was born in Calcutta and educated at King's College Cambridge University. He served as a medical officer in Borneo and came to the United States in 1929 when he joined the faculty of the University of Chicago. After nine years, he became the Professor and Chairman of the Department of Otolaryngology at Washington University, the chair he now occupies. His many contributions in his field of surgery have received laudatory recognition.

ACUTE OTITIS MEDIA

Acute otitis media is an acute inflammation of the mucosa of the middle ear space. It is the result of a direct spread of infection from the nasopharynx via the eustachian tube and is secondary to acute infection of the upper respiratory tract.

The pathologic changes observed in the ear vary with the severity of the infection and the virulence of the invading micro-organism. In mild infections there is edema of the mucosa with an intact epithelium. The swelling of the mucosa may lead to blockage of the eustachian tube and some exudation of fluid into the middle ear. The fluid is mucoid and contains few cells.

When the infection is severe, marked edema of the mucosa with necrosis of the epithelium occurs. The tympanic membrane becomes swollen, thickened and necrotic, leading to perforation. There is considerable exudation of fluid containing many cells. In the more severe infections, direct spread through the aditus ad antrum leads to infection of the mastoid air cells, i.e., *acute mastoiditis*. The continuity of the mucosa of the middle ear and of the mastoid antrum and air cells makes the concept tenable that in any instance of acute otitis media there is some degree of mastoiditis.

Pain of sudden onset, referred to the ear and spreading to the parietal and occipital areas is the most common symptom. The pain may be steady or lancing and severe. It increases in intensity until the drum ruptures or until paracentesis relieves the pressure of the fluid in the middle ear.

In the more severe infections, when the mastoid is involved, tenderness is present

behind the ear and is most common over the region of the mastoid antrum, on the tip of the mastoid process and over the area of the emissary vein.

Swelling behind the ear may occur and is due to inflammation of the postauricular lymph nodes or to edema of the soft tissues. When acute mastoiditis is present and breakdown of the bone occurs subperiosteal accumulations of pus may result.

Elevation of temperature is usual in all of the more severe infections. A fever may range from 38 to 41 C. It may be accompanied by chills.

Hearing loss is present early and the loss is of a conductive type. It is usually not severe and a threshold loss of approximately 10 to 30 decibels is common.

The ear drum shows progressive signs of inflammation. Early dilatation of vessels is noted especially at the periphery of the pars tensa and along the handle of the malleus. As fluid accumulates in the middle ear bulging of the posterior half of the drum occurs and the normal landmarks become less easily recognized. Later there is complete bulging and reddening of the drum. When perforation occurs spontaneously it is usually in the anterior inferior quadrant.

At the onset of pain and loss of hearing, appropriate antibiotic and/or sulfonamide therapy should be instituted. Until the infecting organism can be identified, broad spectrum drugs are advisable and should be administered in adequate dosage. If there is any evidence of fullness in the drum early myringotomy is indicated. Since the advent of the antibiotics and sulfonamides, the tendency has been to refrain from myrin-

gotomy. The physician has been lulled into a sense of security by the clinical response of the patient to the antibiotics, e. g., by resolution of the temperature, remission of pain and other symptoms. The failure to provide adequate drainage to fluid entrapped in the middle ear leads to increasing permanent loss of hearing and to recurrent otitis media and mastoiditis.

In the presence of inflammation of the drum, myringotomy must be performed with the patient under anesthesia. For small children, a general anesthetic is to be preferred; for adults, a local anesthetic may be adequate provided it is carefully administered. The anesthetic of choice is Bonain's solution but it must be used with care. A small cotton wick with only a small portion of the tip moistened with the solution is placed against the ear drum at a point chosen for myringotomy. Care must be taken to avoid contact of the solution with the skin of the external auditory canal. A crescentic incision in the lower quadrant of the drum is made with a sharp-pointed knife. Local application of boric acid and alcohol and frequent cleansing with sterile cotton tipped applicators under direct vision will usually lead to healing. Resolution of the infection occurs in from one to four weeks. In the event that the mastoid has been involved in the infection, healing may occur with adequate antibiotic therapy. In the process of healing, the fluid in the air spaces of the mastoid process becomes organized by an ingrowth of fibrous connective tissue from the mucosa lining and fibrous tissue is formed in an air space. Osteoblasts lay down new lamellar bone and the air space becomes converted into fibrous tissue and new bone with very small, air-containing cavities. In the event that the infection is not overcome by the antibiotics, decalcification of the bony trabeculae occurs and an abscess forms in the mastoid. This necessitates surgical drainage.

Signs pointing to the necessity for surgical intervention in mastoiditis are

1. Persistent discharge from the middle ear for more than four to six weeks after adequate antibiotic therapy
2. Persistent tenderness over the mastoid area in spite of adequate antibiotic therapy
3. Sagging of the posterosuperior external auditory canal wall indicating bony breakdown in the air cells around the antrum.
4. Persistent elevation of temperature.
5. Evidence of spread of infection to adjacent structures, such as the lateral sinus and the meninges. Confirmatory evidence

of bony breakdown may be obtained from roentgenograms of the mastoids.

The operation for acute mastoiditis is designed to promote drainage and to excise diseased tissues. The incision of choice is a postauricular one which is made through the skin superficial tissues and periosteum to the bone. The periosteum is reflected and the cortex exposed. The cortex is entered with a round cutting burr. The mastoid air cells are thoroughly exenterated and the short process of the incus lying in the aditus ad antrum is visualized, the aditus being slightly enlarged to permit free drainage from the middle ear. After all the cells have been carefully exenterated and the landmarks clearly visualized, the incision is closed in layers, care being taken to approximate the periosteum. A drain is left in the lower end of the wound for a few days.

SEROUS OTITIS MEDIA

Serous otitis media is the result of obstruction of the eustachian tube leading to improper ventilation of the middle ear. This may occur as a result of an acute respiratory infection or from obstruction due to allergy. It may occur in sudden descents in an airplane when the eustachian tube does not open adequately (aero-otitis). Eustachian tube obstruction may occur in association with tumors in and about the region of the pharyngeal orifice of the tube. This should be always borne in mind. The symptoms and signs of serous otitis are a sudden loss of hearing in the affected ear, a sensation of fullness, tinnitus which is described frequently as a roaring sound or as a pulsating sound in the ear and is extremely annoying, and autophony—the patient states that when he speaks his voice sounds to him "as though his head were in a barrel." Examination of the ear reveals a peculiarly clear appearing drum. The drum is slightly retracted in those subjects in whom the middle ear is not completely full of fluid, a fluid level may be seen both in the posterior and anterior parts of the drum. In those patients in whom the middle ear is completely full of fluid, there is a characteristic appearance of the drum in which the short process and the handle of the malleus are peculiarly prominent and seem to be a clear white. The rest of the pars tensa has a distinct lemon yellow color and the drum appears transparent. It is not unusual to be able to see, through such a drum, structures in the middle ear—the niche of the round window, the long process of the incus and

The Ears

By THEODORE E. WALSH, M.D.

THEODORE EDWIN WALSH was born in Calcutta and educated at King's College Cambridge University. He served as a medical officer in Borneo and came to the United States in 1929 when he joined the faculty of the University of Chicago. After nine years he became the Professor and Chairman of the Department of Otolaryngology at Washington University, the chair he now occupies. His many contributions in his field of surgery have received laudatory recognition.

ACUTE OTITIS MEDIA

Acute otitis media is an acute inflammation of the mucosa of the middle ear space. It is the result of a direct spread of infection from the nasopharynx via the eustachian tube and is secondary to acute infection of the upper respiratory tract.

The pathologic changes observed in the ear vary with the severity of the infection and the virulence of the invading micro-organism. In mild infections, there is edema of the mucosa with an intact epithelium. The swelling of the mucosa may lead to blockage of the eustachian tube and some exudation of fluid into the middle ear. The fluid is mucoid and contains few cells.

When the infection is severe, marked edema of the mucosa with necrosis of the epithelium occurs. The tympanic membrane becomes swollen, thickened and necrotic, leading to perforation. There is considerable exudation of fluid containing many cells. In the more severe infections direct spread through the aditus ad antrum leads to infection of the mastoid air cells, i.e., *acute mastoiditis*. The continuity of the mucosa of the middle ear and of the mastoid antrum and air cells makes the concept tenable that in any instance of acute otitis media there is some degree of mastoiditis.

Pain of sudden onset, referred to the ear and spreading to the parietal and occipital areas, is the most common symptom. The pain may be steady or lancinating and severe. It increases in intensity until the drum ruptures or until paracentesis relieves the pressure of the fluid in the middle ear.

In the more severe infections when the mastoid is involved, tenderness is present

behind the ear and is most common over the region of the mastoid antrum, on the tip of the mastoid process and over the area of the emissary vein.

Swelling behind the ear may occur and is due to inflammation of the postauricular lymph nodes or to edema of the soft tissues. When acute mastoiditis is present and breakdown of the bone occurs, subperiosteal accumulations of pus may result.

Elevation of temperature is usual in all of the more severe infections. A fever may range from 38 to 41 C. It may be accompanied by chills.

Hearing loss is present early and the loss is of a conductive type. It is usually not severe and a threshold loss of approximately 10 to 30 decibels is common.

The ear drum shows progressive signs of inflammation. Early dilatation of vessels is noted especially at the periphery of the pars tensa and along the handle of the malleus. As fluid accumulates in the middle ear bulging of the posterior half of the drum occurs and the normal landmarks become less easily recognized. Later there is complete bulging and reddening of the drum. When perforation occurs spontaneously it is usually in the anterior inferior quadrant.

At the onset of pain and loss of hearing, appropriate antibiotic and/or sulfonamide therapy should be instituted. Until the infecting organism can be identified, broad spectrum drugs are advisable and should be administered in adequate dosage. If there is any evidence of fullness in the drum early myringotomy is indicated. Since the advent of the antibiotics and sulfonamides, the tendency has been to refrain from myrin-

ized into fibrous tissue and a true adhesive deafness follows. In these subjects, we have seen strands of fibrous tissue binding down the ossicular chain and giving rise to a conductive deafness resembling that of otosclerosis. Rarely in patients in whom there is a considerable accumulation of mucus in the middle ear with blockage of both of the round window and immobilization of the ossicular chain, hearing loss will be profound with all the characteristics of perceptive deafness.

CHRONIC OTITIS MEDIA

Chronic otitis media is the result of unresolved acute otitis media. The patient complains of a chronically discharging ear with some loss of hearing, acuity. This disease may be classified into three types.

Type I is characterized by an intermittent or constant discharge from the ear which is frequently scanty and becomes more profuse when a respiratory infection is present. The discharge is mucoid and, under ordinary conditions of cleanliness, without odor. The discharge may cease spontaneously only to recur with the next respiratory infection. On examination of the ear there is seen a perforation usually in the anteroinferior quadrant which may be small. The hearing in such subjects is not remarkably depressed commonly from 10 to 30 decibels of conductive loss being found. The discharge from the ear is due to a low grade infection of the mucosa of the middle ear and of the eustachian tube with secretion of mucus from the goblet cells in the eustachian tube and in the mucosa of the middle ear.

Type I chronic otitis media is a condition which is not dangerous to life. Surgery should be aimed at restoration of function rather than excision of disease. Of paramount importance is attention to the nasopharynx with the object to eliminate any chronic upper respiratory infection either in the nasopharynx or in the sinuses and also to prevent recurrent acute infections.

If the ear canal is wide enough to permit adequate operating space the perforation in the drum can frequently be healed by myringoplasty.

In this operation, the superficial layer of epithelium from the drum is removed and the edge of the perforation freshened. The epithelium is removed for approximately 5 mm. all around the perforation. Then a thick split-thickness graft is taken from some area of skin that is not hair bearing and the graft placed over the perforation and lightly

packed in with cotton balls. This should be left without interference for ten days. The objective of closing the perforation is to rehabilitate the patient so that he may get water in the ear with impunity may go swimming, etc. The operation also will often improve the hearing to within normal limits provided there is no actual fixation of the ossicular chain.

Local therapy to the ear consists of cleanliness for which applications of boric acid and alcohol are used.

The patient with type II chronic otitis media complains of a constant foul smelling discharge from the ear. This discharge may be profuse is constant and in spite of all efforts of cleanliness is malodorous. Examination of the ear shows a large perforation frequently occupying most of the pars tensa. The handle of the malleus may be retracted and appear foreshortened. The medial wall of the tympanum may be covered with granulations or one may see polyps filling the external auditory canal. The perforation of the drum is central and does not involve the annulus or margin of the drum. The pathologic process in this instance is one of a low grade osteitis of the ossicular chain or of the bony trabeculae in the epitympanum and hypotympanum and of the peritubal air cells. The hearing is usually markedly depressed and commonly 40 to 50 decibels threshold loss for the pure tones is found. The bone conduction in most instances is good, but if the disease is present for a long time more and more nerve involvement may occur so that bone conduction is depressed. Because of the inaccessibility of the disease process to medication, local treatments to the middle ear are, as a rule ineffective. In some subjects, it is possible to displace antibiotic solutions into the middle ear and effect remission of the disease. As a rule, however cure can only be effected by radical mastoidectomy.

Type III chronic otitis media is a condition which is potentially dangerous to life. The discharge may be intermittent and on occasions scanty. The hearing is usually good, a threshold loss of less than 15 decibels is not uncommon. The perforation, which is marginal involving the annulus, is usually seen in the posterosuperior quadrant of the drum, through the pars flaccida or sometimes extending to the anterosuperior portion of the drum. On occasions, the perforation is so small that it is difficult to visualize without adequate magnification, sometimes it may be covered with a small

the incudostapedial joint, and the tendon of the stapedius muscle.

Treatment consists of evacuation of the fluid from the middle ear by paracentesis. For this no anesthetic is required. It is preferable not to prepare the external auditory canal by any cleansing. If wax and debris obstruct a clear view of the drum, they can be gently removed with cotton tipped applicators, but application of antiseptics or cleansing agents in the canal should be avoided. An adequate speculum and good illumination are essential and with a very sharp paracentesis knife a small puncture is made in the inferior portion of the pars tensa. By the use of a pneumatic otoscope fluid may be aspirated from such a perforation or with the use of a small suction tip the ear may be evacuated. It is essential at this time that the eustachian tube be inflated to insure complete evacuation of fluid from the middle ear. After such a procedure the hearing is returned to normal almost immediately. In most cases the perforation heals by the day following treatment and the condition is cured.

When there is a persistent recurrence of fluid in the middle ear further etiologic factors must be sought. In many of these patients allergy is the cause. It should be repeated that in the presence of serous otitis the greatest attention should be paid to the nasopharynx to eliminate all possibilities of tumor. It is not infrequent that serous otitis is the only early evidence of malignant disease in the nasopharynx.

Examination of the ear requires the use of a pneumatic otoscope and with this instrument it is seen that in serous otitis the ear drum will not move normally. There seems to be always a "splinting" of the drum when fluid occupies the middle ear space. Evacuation of fluid from the middle ear may be accomplished on occasions by eustachian tube inflation alone. If the patient is placed in such a position that the affected ear is upward and gentle inflation done by Politzer's method, fluid may be evacuated from the ear as air replaces it. This method, however, does not always completely evacuate the fluid and paracentesis is the preferable treatment. In some instances, fluid re-forms in the middle ear in spite of paracentesis and in spite of all efforts at medical therapy to control allergic and other factors. In such subjects a simple mastoidectomy has been recommended. We do not recommend mastoidectomy for these patients as it has been our experience that, in spite of all the mas-

toid cells being removed, fluid still accumulates in the middle ear from the middle ear mucosa. In patients with chronic disease in whom fluid reaccumulates in the ear the insertion of a small polyethylene tube through the pars tensa into the inferior portion of the middle ear is recommended and has been found successful in many instances. This tube may be left in place for a week or ten days and, when the ear is dry the tube is removed and the perforation closes spontaneously.

CHRONIC MUCOUS OTITIS MEDIA

Chronic mucous otitis media is the result of a low grade infection in the middle ear or is due to the improper treatment of acute otitis media of low virulence. Nowadays, more and more cases of chronic mucous otitis are observed in children who have had acute otitis media treated with antibiotics without the benefit of myringotomy. The common complaint is difficulty in hearing. The deafness is found to be of a conductive type. In mucous otitis the drum is thickened, dull and has a peculiar creamy appearance. With magnification and with the use of a pneumatic otoscope the posterior half of the drum is seen to be slightly full and does not move adequately on positive or negative pressure. The landmarks are visible but are slightly distorted by the thickened appearance of the drum and its immobility. The treatment is wide myringotomy and this must be done with the patient under general anesthesia. It is not possible adequately to evacuate the middle ear of mucus with the patient under local anesthesia because frequently such evacuation necessitates the use of very strong suction and sometimes of forceps. Any trauma to the promontory causes considerable pain. We have found it necessary on many occasions to make two incisions, one in the lower part of the drum and a counterincision high anteriorly to allow for the entrance of air as the mucus was being evacuated. This mucus is tenacious, sticky and sometimes is extremely difficult to remove. Inflation of the eustachian tube after myringotomy and careful attention to the nasopharynx are important. It is fortunate that in most patients in whom mucus has been evacuated from the middle ear it will not re-form and the patient's hearing is restored. Occasionally, with subsequent mild respiratory infections further accumulation of mucus may occur. In the event that no treatment is provided, it is not infrequent for such mucus in the ear to become organ-

unusual that there will be an intact ossicular chain, so generally the incus and malleus must be removed. This is accomplished while preserving the tympanomeatal skin flap and any of the pars tensa possible. Diseased mucosa is removed from the promontory but the eustachian tube is not curetted. Perforations in the pars tensa are closed by a tympanomeatal flap or by a thin full thickness skin graft. The tympanomeatal flap or skin graft is packed down onto the head of the stapes. An attempt is made to create an air-containing space between the eustachian tube and the round window. This is usually possible by the use of a small amount of Gel foam in the hypotympanum which, when absorbed, leaves an air space. When such an air space can be obtained and good contact is made between the skin graft and the head of the stapes hearing is remarkably restored.

DEAFNESS

Deafness may be conductive or perceptive. Conductive deafness results from interference with sound impulses reaching the inner ear by occlusion of the external auditory canal, by wax, foreign bodies inflammation or tumors by disease of the middle ear structures as in otitis media, either acute or chronic, in which the continuity of the ossicular chain is broken or in which adhesions may form in the middle ear by fixation of the ossicular chain as in otosclerosis. When sound impulses reach the inner ear but are not perceived or correctly interpreted, there is perceptive deafness. This may be due to a lesion in the end organ in the cochlea or to lesions in the cochlear division of the eighth nerve or in the central cerebral pathways.

Tests of hearing are performed with tuning forks and audiometers. Fork tests are qualitative tests and they compare the patient's ability to hear by bone and air conduction.

The Rinne test compares the patient's hearing by bone and air conduction. Normally, when a tuning fork is struck and the stem held on the mastoid process, the sound can be heard in the homolateral ear for a certain number of seconds. After it has ceased to be heard behind the ear and the tines of the forks then are held close to the external auditory canal, the sound is again perceived. This is termed a "positive Rinne." In perceptive deafness the Rinne is positive; in other words, the patient will hear longer by air conduction than by bone conduc-

tion. In conductive deafness the contrary is true and the patient will hear better by bone conduction than he will by air conduction. This is termed a "negative Rinne."

The Weber test compares the ability of the two ears to hear by bone conduction. The tuning fork is struck and the stem held on the vertex of the head or on the upper incisor teeth. When the hearing is unequal in the two ears, the sound will be referred to the better ear in perceptive deafness and to the worse ear in conductive deafness.

The Schwabach test compares the ability of the patient to hear by bone conduction with that of the observer. The tuning fork is struck and held on the observer's mastoid process until he can no longer hear it and then on the patient's mastoid process and vice versa. In perceptive deafness, assuming that the observer's hearing is normal, the patient will not hear as well by bone conduction as will the observer. On the other hand assuming that the observer's hearing is normal in conductive deafness the patient will hear for a longer period than the observer. Fork tests, therefore are qualitative tests of the hearing and differentiate between perceptive and conductive deafness.

Audiograms measure the patient's threshold of hearing and are plotted in relation to the established normal. Audiograms are made either with pure tones usually at octave intervals from 128 double vibrations up to 8192 double vibrations, or the signal instead of pure tones may be speech. A pure tone audiogram measures only the patient's threshold of hearing for pure tones. Speech audiograms measure not only the threshold of hearing for speech, but the ability of the patient to understand speech when the signal is given at an intensity easily heard by the patient. Therefore it measures not only the threshold but the suprathreshold areas of hearing for speech. The importance of speech testing cannot be overestimated in relation to rehabilitation of patients for everyday life. To translate pure tone losses into losses for speech is not justifiable.

Perceptive deafness may be congenital or acquired. In the congenital type the parents usually notice that their child is deaf because he may not develop speech at the normal time or he may not respond adequately to sounds. To dismiss parents of such a child with the remark, "there is nothing to be done" is inexcusable. These parents need encouragement from physicians and they should be told that, although there is no medical treatment which can restore

dry crust which may be mistaken for cerumen. In almost every instance a history of previous ear infection in childhood can be obtained and on careful examination it will be seen that through the perforation one can obtain flakes of desquamated epithelium and sometimes pus. If pus is present, it invariably has an extremely unpleasant foul odor. Occasionally there are granulations and almost always cholesteatosis. Cholesteatosis occurs when there is metaplasia of the squamous epithelium through a marginal perforation of the drum and a continued growth of such epithelium into the epitympanic space, mastoid antrum and middle ear. In the presence of an acute exacerbation in such an ear intracranial complications are frequent and may occur with distressing suddenness. It has been thought that the absorption of bone which accompanies cholesteatosis is due to pressure of desquamating epithelial cells which are unable to be extruded through the small perforation. However, on examination of the sections of ossicles removed at operation in patients with cholesteatosis it is seen that the sub-epithelial connective tissue matrix extends along the haversian canals through the periosteal layer of bone to come to lie adjacent to calcified cartilaginous rests in the enchondral layer. In many instances, these are seen to be undergoing decalcification, possibly from chemical lysis. In these patients an acute superimposed infection may extend through the bone and give rise to labyrinthitis, epidural abscess or lateral sinus thrombosis. In many instances acute infection superimposed on chronic type III otitis may lead to brain abscess presumably by retrograde thrombosis of the vessels in the vicinity. Surgery in the type III ear is aimed at excision of disease and preservation of function. This is usually accomplished by a radical mastoidectomy.

In any patient with chronic otitis media, if there is superimposed an acute infection complications are possible. They are lateral sinus thrombosis and perisinus abscess, meningitis, labyrinthitis and brain abscess.

Pain in the ear and headache, referred to the temporal and occipital areas of the affected side, are indications that infection is extending beyond the ear. Spiking temperature with or without chills, is an indication of lateral sinus involvement. A lack of rise in the cerebrospinal fluid pressure with compression of the jugular vein indicates the probability of lateral sinus thrombosis. Stiff neck, an increased cerebrospinal fluid pres-

sure and the presence of cells in the cerebrospinal fluid are indicative of meningitis. Attacks of vertigo with the production of nystagmus or the ability to produce nystagmus on positive or negative pressure in the ear indicate a labyrinthine fistula.

The surgical treatment of chronic otitis media is aimed at excision of diseased tissue with preservation of function wherever possible.

Tympanoplasty. In those cases of chronic otitis media in which there is a large perforation of the pars tensa, but in which discharge is absent and no active infection is present, the hearing may be improved and the ear made safer by closure of the perforation by a skin graft. A very thin full thickness graft is taken from behind the ear. Through a large ear speculum under magnification, the edges of the perforation are freshened and the epithelium removed from the drum and from the skin of the canal for approximately 0.5 cm from the edge of the perforation. The graft is now placed against the freshened area and held in with appropriate packing for approximately ten days. The patient is cautioned against nose blowing. Such grafts take in a high percentage of cases and, with closure of the perforation, hearing is improved and the likelihood of subsequent acute exacerbations is lessened.

Radical Mastoidectomy. This procedure is performed in those cases of chronic otitis media in which the hearing is so far deteriorated that any restoration is impossible. The endaural approach is preferable as it affords the most direct route to the disease. All diseased cells in the mastoid are exterminated. The bridge over the aditus ad antrum is thinned and removed. The outer attic wall is removed and the epitympanum is exposed. The incus and malleus are removed and the hypotympanic area thoroughly exposed by lowering the facial ridge. The tensor tympani muscle is removed and the eustachian tube and peritubal cells are curetted. All mucosa is removed from the middle ear. The eustachian tube is plugged, preferably with bone taken from the superior iliac crest. A full-thickness graft taken from behind the ear is placed in the middle ear and held in with proper packing.

Modified Radical Mastoidectomy. In those cases of chronic otitis media in which there is little demonstrable perceptive loss of hearing, every effort should be made to restore the conductive loss. The objective of surgery should be to eliminate the disease with preservation of any normal tissue. It is

When this is successfully accomplished, there is an immediate improvement in hearing which can be demonstrated by audiometry in the operating room. Success with this operation is at present possible in only approximately 50 per cent of attempts. Following mobilization of the stapes the tympanomeatal flap is replaced. No packing is necessary in the ear and healing is rapid.

Fenestration Operation An endaural incision is made and sufficient mastoid cells excised to make possible the approach to the horizontal canal and its anterior end. The incus and head and neck of the malleus are removed after the tympanomeatal flap has been mobilized and cut. The flap is thoroughly cleansed of any bone chips and, under high magnification the fenestra is made anteriorly in the amputated end of the horizontal canal. The fenestra usually is approximately 3 to 4 mm. in length by 1 to 1.5 mm. in width. When this has been clearly accomplished, the tympanomeatal flap is placed firmly against the fenestra and held in place with packing. The results from fenestration have been successful in about 85 to 90 per cent of ideal cases and hearing has been maintained for periods up to four teen years.

In comparing the two surgical procedures, it is evident that stapes mobilization is less immediately traumatic to the patient and causes less postoperative morbidity. The chance of success in restoration of hearing, however is infinitely greater with fenestration.

MENIERE'S DISEASE

Meniere's disease is characterized by nerve deafness, tinnitus and vertigo.

The pathologic process in this disease is that of increased endolymphatic pressure in the scala media. Sections of temporal bones from patients with Meniere's disease who have died from other causes, have shown dilatation of the cochlear duct and herniation of the utricle and saccule into the amputated end of the semicircular canals.

Meniere's disease (endolymphatic hydrops) is primarily a cochlear disease. A careful history will reveal that the disturbance of hearing has been present usually a long time before the disabling symptom of vertigo appears. The patient usually presents himself complaining of vertiginous attacks with accompanying tinnitus; the history of deafness has to be elicited.

There is a true aural vertigo. The patient states that the vertigo may come on sud-

denly at any time. Frequently it will occur when he is in bed. There is a sensation of objects moving in a rotary fashion or some times the patient complains that he himself seems to be spinning. The vertigo is usually accompanied by nausea and vomiting and is incapacitating. It may last for a few minutes or for several days. The attacks may occur fairly regularly at biweekly or monthly intervals or occur frequently and at irregular intervals. There may be long periods of freedom from attacks.

The tinnitus is usually a roaring sound likened by some to the sound heard on holding a sea shell to the ear. It may be described as "the sound of surf on the sea shore" or "of a wind in the forest." Or there may be an intermittent and much less bothersome high-pitched ringing. In many instances the tinnitus either disappears just preceding a vertiginous attack or may become very much louder temporarily and be followed by the vertiginous attack.

The patient notices a loss of hearing in the affected ear. He usually can state that although he hears sounds the sounds are distorted and they sound different in the two ears (diplacusis). Although speech can be heard, it frequently cannot be understood. The hearing varies considerably and patients will state that at times they hear "perfectly" while at other times there is a "sense of pressure or fullness" in the ear and the hearing ability is impaired. Those patients who are musical complain bitterly of the difference in pitch appreciated by the two ears. Many patients state that loud sounds hurt the affected ear.

Usually the examination of the ear, nose and throat is unrevealing, but the functional tests will show abnormalities.

Fork tests as a rule give characteristic findings of nerve deafness. The Rinne test is positive. In the Weber test the sound is referred to the better ear. In the Schwabach test the bone conduction is diminished. Comparison of the sounds made by a tuning fork held close to each ear alternately reveals that the same fork may sound higher or lower in the affected ear than in the normal one. The sound, too, is not clear but is described as "fuzzy." Diplacusis may be present only in the low tones or only in the high, or may be confined to only one frequency but it is nearly always present. This diplacusis can be measured.

The pure tone audiogram reveals a threshold loss of hearing more marked in the low tones in the earlier stages, later becoming a

the congenitally deaf patients hearing, he may be taught to speak and be able to go through his educational years provided the training is started early. It is suggested that the parents apply at special schools for the deaf and that the child should start his education when three years of age.

Acquired deafness may be caused by toxic factors or agents. Examples are such drugs as quinine and streptomycin or various virus infections such as mumps, measles and influenza. In toxic deafness the hearing loss is commonly about the same throughout the scale, low tones being affected as much as high tones. There is frequently an accompanying tinnitus which is extremely bothersome. The speech audiogram will show a threshold loss of hearing varying in degree but the discrimination ability of the patient is markedly depressed. He will understand probably less than 40 per cent of the words which he hears. It is important to realize that acquired toxic nerve deafness is not amenable to therapy and the patient should be encouraged to learn "to hear with his eyes and with his brain" rather than with the ears and should be discouraged from continual therapy with vitamins and other nostrums so commonly advised.

Presbycusis occurs in persons of advancing age and is characterized by a loss in the high tones. The threshold of hearing for speech is not greatly impaired as the hearing for 500 and 1000 cycle tones is commonly good. The ability to understand speech is impaired, i.e., there is a marked discrimination loss.

The term *otosomotor deafness* may be used to describe such conditions as Meniere's disease, in which the hearing loss fluctuates from time to time and may be accompanied by tinnitus and dizziness. In early cases it is possible sometimes to arrest the hearing loss by use of vasodilator drugs.

Conductive deafness is the only form of deafness which can be helped surgically. Wax and foreign bodies in the external auditory canal should be removed with care. In most subjects, syringing with warm water is the method of choice for removal. If instrumentation is necessary the utmost care should be taken not to push the foreign body further into the canal or through the drum into the middle ear. If difficulty is encountered in the removal of a foreign body a general anesthetic should be used. Deafness due to accumulation of fluid in the middle ear can be promptly relieved with myringotomy.

Adhesive deafness can be greatly helped by adequate surgery to the middle ear and/or by fenestration.

Otosclerosis is a disease of youth. It is between four and eight times more common in women than in men. The deafness is commonly first noticed when the subject is in the late teens or early twenties and is progressive. Patients with otosclerosis complain of tinnitus, of being unable to hear unless people speak loudly but of being able to hear perfectly when the sound is made loud enough. They hear better in a noisy place than in a quiet one and usually well over the telephone. The patients themselves have a soft, quiet, well modulated voice and good articulation. Examination shows the ear nose and throat to be entirely normal except that, not infrequently, the tympanic membrane is seen to be atrophic and there may be patches of atrophy resembling healed perforations. In some subjects in whom the disease is active, there may be seen a pink glow through the drum which is the result of vasodilatation in the mucosa over the promontory (Schwartz's sign). Hearing tests in persons with otosclerosis demonstrate a negative Rinne, an increased Schwabach and in the Weber test the sound is referred to the ear with the greater hearing loss. With pure tones there is a threshold loss for air conducted sounds but there is normal, or near-normal, bone conduction.

Speech tests show a threshold loss comparable to that of the pure tones and the patient can repeat correctly ± 100 per cent of the words heard if they are given loudly enough (normal discrimination).

Patients with otosclerosis may be helped by mobilization of the stapes or by fenestration.

Stapes Mobilization. The operation for mobilization of the stapes is done under local anesthesia through a large ear speculum. An incision is made approximately 5 mm. from the annulus tympanicus from eleven o'clock to six o'clock on the right side and from one o'clock to six o'clock on the left side and the skin is elevated from the bony canal wall. The drum is lifted out of the annulus and turned forward so that the middle ear is exposed. Further exposure must be obtained by removal of the thin margin of bone of the posterior canal wall until a good view of the incudostapedial joint and the foot plate of the stapes is obtained. With pressure on either the incus or on the margins of the foot plate an attempt is made to mobilize the fixed stapedial foot plate.

underside of the facial nerve or possibly might be caused by heat if the area is not satisfactorily dried before the current is applied, in effect boiling the undersurface of the nerve. In any event if the patient does by misfortune show a facial paralysis following the operation no time should be lost before the nerve is completely decompressed.

Labyrinthotomy is simpler and preferable to section of the eighth nerve. Differential section has no place in the treatment because the hearing is impaired and its preservation is not worth while.

TUMORS

Benign tumors of the middle ear and external ear are commonly the result of supuration in the ear. Fibromas or fibrous granulations and polyps may be seen in the external ear coming through a perforation in the drum.

Osteomas occur in the external bony canal. They seldom give rise to symptoms unless they increase in size sufficiently to occlude the canal and cause deafness. These tumors are small, usually present on the superior and posterior aspects of the external auditory canal and can be removed easily by elevation of the skin in the canal and removal of the mass by cutting burs or curets.

Chemodectomas may arise in the middle ear and may present in the external canal either through a perforation of the drum or they may be present with an intact drum. They are not common tumors.

Carcinoma of the middle ear is rare and the prognosis is poor. Carcinoma of the external canal may occur and invade the middle ear space. If such a tumor is found early and is removed by an extremely radical excision the prognosis is fair but recurrence locally is common. An extension intracranially is usually the cause of death. The results of x-ray therapy of squamous carcinoma of the middle ear are disappointing.

Sarcoma of the external ear with extension to the middle ear is rare but does occur occasionally in children.

MALFORMATIONS

Congenital agenesis of part of the ear may occur. Microtia, with partial or complete atresia of the external auditory canal, with or without agenesis of the middle ear may be present. It may be unilateral or bilateral. In unilateral cases it is usually unnecessary to reconstruct a middle ear as the hearing with one normal ear is not a sufficient handi-

cap to warrant surgery. Plastic reconstruction of the external ear is advisable particularly in boys for cosmetic reasons. In the bilateral cases, it is important to determine early whether there is hearing by bone conduction and if so it is possible to reconstruct the middle ear and improve function and at a later date still further improve hearing by fenestration. The operation for restoring the middle ear function is difficult because the landmarks are distorted. The cortex of the bone is removed and the middle ear space found. Usually the external auditory canal is represented by fibrous tissue and the drum is represented by a bony plate. Frequently the middle ear space is normal except that usually the incus and malleus are distorted, fused and in an abnormal position. They are removed. The bony plate representing the drum is removed to an extent which allows for entrance to the middle ear. A skin graft is placed over the defect so caused and attached to the horizontal canal thus forming a closed tympanic space. The incus may be kept open with skin grafting and with packing. If one is successful in re-forming an external auditory canal and middle ear plastic reconstruction of the external ear may be done at a later date. Reconstruction of the middle ear should be done when the child is from three to five years of age to avoid poor speech habits which result from deafness.

READING REFERENCES

- Baron, S. H. Modified Radical Mastoidectomy. Preservation of the Cholesteatoma Matrix, a Method of Making a Flap in the Endaural Technique. *Arch. Otolaryng.* 49:250, 1949.
- Baron, S. H. Symposium. The Surgical Management of Aural Cholesteatoma. Why and When I Do Not Remove the Matrix. *Tr. Am. Acad. Ophth.* 57:694, 1953.
- Boies, L. R. *Fundamentals of Otolaryngology* 3rd ed. Philadelphia, W. B. Saunders Company 1950.
- Cawthorne, T. E. The Pathology and Surgical Treatment of Bell's Palsy. *Proc. Roy. Soc. Med.* 44:565, 1951.
- Cawthorne, T. E., and Hewlett, A. B. *Ménière's Disease*. *Proc. Roy. Soc. Med.* 47:663, 1954.
- Davis, H. *Hearing and Deafness*. New York, Murray Hill Books, Inc., 1947.
- Davis, H. and Walsh, T. E. The Limits of Improvement of Hearing Following the Fenestration Operation. *Laryngoscope* 60:273, 1950.
- Day, K. M. The Management of Deafness. *Tr. Am. Acad. Ophth.* 55:22, 1950.
- Day, K. M. Aids to Hearing. *Laryngoscope* 64:1, 1954.
- Derlacki, E. L. Non-surgical Management of Meniere's Disease. *Laryngoscope* 64:271, 1954.
- Hoople, G. D. Otitis Media with Effusion. *Tr. Am. Acad. Ophth.* 54:531, 1950.

flat loss throughout the scale and still later showing a greater loss for the high frequencies. The bone and air thresholds are equal. Loudness balance tests reveal recruitment or hyper recruitment.

Speech audiometry characteristically shows a proportionally greater loss for discrimination than might be expected from the threshold loss. We have found in our patients that whereas the loss at threshold for speech is perhaps only 40 decibels, the discrimination score is frequently less than 30 per cent.

Caloric tests are not diagnostic in Meniere's disease. In the early stages one may find that the responses in the affected ear are equal to those of the normal ear. Later as a rule the affected ear becomes hypoactive and one may find a directional preponderance to the unaffected side. In patients with long-standing cases one may obtain no response to stimulation of the affected ear even with maximum stimulation with ice water.

Although the cause of Meniere's disease is not known, clinical evidence is accumulating which points to the possibility of a vasomotor change leading to spasm of the internal auditory artery. Such vasomotor change may be the result of an allergic state; for example a true hypersensitivity to foods or inhalants. Endocrine imbalances or psychogenic stimuli may have an effect. It is of importance to investigate all possibilities and eliminate the possible causative factors. However such investigation is difficult and frequently time consuming, and treatment should be instituted early. If the theory of a vasospastic state is correct, it is important to relieve this spasm and vasodilators have been recommended. The use of RoNiacoI or nicotinic acid (50 mg.) together with thiamine chloride (10 mg.) and ascorbic acid (250 mg.) three times a day with meals is often helpful. In addition, Banthine (50 mg.) or Pro-Banthine (15 mg.) is given every six hours. In some cases atropine (1/50 grains three times a day by mouth) has proved beneficial. In many subjects this has prevented the dizzy spells although others under such therapy have had attacks.

In a few of the patients large doses of vitamin B₁₂ have been used with apparent success. One thousand micrograms are given intramuscularly daily for thirty days, and thereafter as a maintenance dose at intervals to be determined. Although the patients who have benefited from this treatment are few it warrants further trial.

Histamine therapy has proved valuable in some instances. The patient is tested with an intradermal injection of histamine at dilutions of 1:100,000,000, 1:10,000,000, 1:1,000,000 and 1:100,000. If he shows a definite reaction—a wheal with or without erythema—the dilution with which the least positive reaction was obtained is used. Biweekly doses are given starting with 0.1 cc. and increasing by 0.1 cc. each time until there is response. Thereafter weekly injections of the appropriate dose are given. By this means a reversal of the hearing loss and a disappearance of symptoms have been obtained in a number of patients. It should be noted that if too large a dose is given the hearing again becomes worse and the tinnitus and vertigo reappear.

Normal remissions in this disease make it difficult to evaluate therapy and one must be guarded in ascribing to therapy what may be the result of a normal fluctuation in the disease.

When the patient has not been helped with medical management and has become incapacitated or handicapped by his disabling vertigo, destruction of the labyrinth will give complete relief from the symptoms provided only one ear is diseased. It is emphasized that labyrinthotomy is reserved for patients in whom the disease is unilateral.

Labyrinthotomy is a simple procedure performed either through an endaural or a postauricular approach as the surgeon desires. Enough mastoid cells are excised to allow for a direct approach to the horizontal semicircular canal and, particularly, to its anterior end. It is usually advantageous to remove the incus and the head of the malleus to have an easier access to the vestibule. A large opening is made in the vestibule. The endolymphatic labyrinth is avulsed and, after the area is carefully dried, a coagulating current is applied to the vestibule in order to destroy both utricle and saccule completely.

Results from labyrinthotomy are universally good if an adequate destruction is accomplished. If however the utricle and saccule are not completely destroyed, the patient, although he may have no true vertiginous attacks, may be unsteady and have difficulty on forward motion.

There should be no danger from the procedure of labyrinthotomy. There have been reports of facial injury following coagulation of the labyrinth. This is probably either due to improper placement of the coagulating electrode so that it approximates the

relatively mild soft tissue injury to the lips or elsewhere on the face without breaking the skin or mucosa and are familiar to all. They are soft tissue response to trauma. Degree of swelling, ecchymosis and pain are in direct proportion to the extent of the injury. Treatment for the most part is symptomatic. Pain can usually be relieved by the usual analgesics, such as aspirin but cold applications may help to control the swelling and the pain. Caution is indicated in the use of cold applications when the swelling is pronounced enough to cause tenseness of the skin, as a slough may result in the skin with the already compromised circulation. Soft light cool cloths are the most satisfactory. Ice or ice bags are not used on a severely swollen area about the face or mouth because the added pressure may cause necrosis. Occasionally a localized collection of blood may be present in a contused or undermined area, which may be evacuated by needle aspiration, and thereby hasten subsidence. Elastic or adhesive support of contused, swollen lips and undermined areas adds to the comfort of the patient but should be changed frequently enough to be kept clean. The swelling in most contusions subsides in two weeks, but discoloration may persist considerably longer. Persistent discoloration at this stage can be covered by cosmetics.

Lacerations and tears of the skin or mucosa may result from sharp cuts or from a blunt force and need no aids to diagnosis except in the latter to recognize that there are varying degrees of soft tissue contusion and explosion which may proceed to deep or superficial necrosis. Simple lacerations of the lips and elsewhere about the face, resulting from sharp objects such as glass, will usually heal if further trauma is not added by the repair. Treatment of simple lacerations consists of adequate cleansing of the surrounding area, irrigation of the wound, minimal debridement, exploration for and removal of foreign bodies which constitute the debridement, ligation of individual bleeding points and careful approximation of the wound edges with deep sutures whenever possible. No viable tissue is cut away. Local anesthesia should be used. The patient should not be hurt. The injection may be made through the open wound with topical application to start with. Facial wounds are never packed open. Dirt blasted into wounds is completely removed to prevent permanent tattooing. Superficial sutures of the finest unabsorbable material practi-

cable (3-0 to 7-0 silk) are placed close to the edges of the wound and a supporting pressure dressing is applied.

Repair of more complicated tears and trap-door lacerations should be made as soon as the patient's general condition permits and follows in general the procedure outlined for simple lacerations. General anesthesia may facilitate the repair and be to the patient's advantage. Known points are approximated first as they are recognizable such as lip, nose and eyelid border and the remainder of the laceration is closed by successive bissection of the wound. Minimal debridement in facial wounds is of particular importance as features are involved and cutting debridement may create deformities for which innumerable secondary operations cannot compensate (Fig. 1). It is preferable to leave areas of questionable blood supply for later excision in hope they may survive. Packing wounds open or doing cutting debridement is a general surgical rule mainly for the extremities, to prevent gas gangrene. As gas gangrene does not occur in the face and as features may be lost by either of these procedures they are not carried out in this region. The debridement is thorough as far as cleansing is concerned, but ragged, torn wounds which elsewhere would be excised are cleansed, sorted out and carefully put back together in their entirety. Trap-door type of lacerations tend to heal irregularly and require a most accurate approximation of all edges. Tears of the lips or lining of the mouth require careful closure of both skin and mucosal surfaces and stay sutures should be put on the inside to try to convert the skin surfaces to the simplest possible closure.

Loss of tissue in facial wounds usually does not require immediate rotation of flaps for closure because of the addition of scarring in the face and possible loss of motor and sensory nerve supply. Partial closure in areas of actual loss is usually preferable. Healing is encouraged and secondary repair is done for a superior final result (Fig. 2). Free grafts may be used for coverage and distortion of features is avoided. Widely placed stay sutures are not used on the face, since it is impossible to ever eradicate their marks which are known as a "ladder effect." If necessary sutures at a distance from the edges of the wound can be tied over a fold of gauze to prevent permanent suture marks.

Lacerations resulting from a blunt force are usually accompanied by contusion, undermining and, in general, damage to all

- Juerg, A. L.: Preservation of Hearing in Surgery for Chronic Ear Diseases. *Laryngoscope* 64:235, 1954.
- Lempert, J.: Fenestra Nov-ovalis. *Arch. Otolaryng.* 34:880 1941
- Lindsay J. R. Ménière's Disease. *Histopathologic Observations.* *Arch. Otolaryng.* 39 313, 1944
- McGuckin, F. The Management of Chronic Otitis Media. *Proc. Roy. Soc. Med.* 46:371 1953.
- Paltee, G. L. An Operation to Restore Hearing in Cases of Congenital Atresia of the External Auditory Meatus. *Arch. Otolaryng.* 43:568, 1947
- Reading, P. Management of Soft Tissues in Mastoidectomy. *J. Laryng. & Otol.* 67 457 1953.
- Rosenwasser H. Glomus Jugularis Tumor of the Middle Ear. *Laryngoscope* 62:693 1952.
- Shambaugh, G. E. Jr. Surgical Treatment of Otosclerosis. *Ann. Otol., Rhin. & Laryng.* 51 817 1942
- Shambaugh, G. E. Jr. Developmental Anomalies of the Sound Conducting Apparatus and Their Surgical Correction. *Ann. Otol. Rhin. & Laryng.* 61 873, 1952.
- Shambaugh, G. E. Jr. *Surgery of the Ear*. Philadelphia, W. B. Saunders Co., 1959
- Sullivan, J. A. Recent Advances in Surgical Treatment of Facial Paralysis and Bell's Palsy. *Laryngoscope* 62:449 1952.
- Tracy S. (Mrs.) Listening Eyes. *Tr. Am. Acad. Ophth.* 53:583 1949
- Turner L. *Nose, Throat and Ear*. Baltimore, Wm. Wood & Company 1938.
- Walsh, T. E. Speech Audiometry. *J. Laryng. & Otol.* 67 119 1953.
- Walsh, T. E. Why I Remove the Mairrh. *Tr. Am. Acad. Ophth.* 57:687 1953
- Walsh, T. E. The Effect of Pregnancy on the Deafness Due to Otosclerosis. *J.A.M.A.* 154 1407 1954
- Walsh, T. E. The Diagnosis and Treatment of Ménière's Disease. *Arch. Otolaryng.* 64 118, 1956
- Walsh, T. E., Covell, W. P. and Ogura, J. H.: The Effect of Cholesteatosis on Bone. *Ann. Otol. Rhin. & Laryng.* 60:1100 1951
- Walsh, T. E., and Silverman, S. R.: Diagnosis and Evaluation of Fenestration. *Laryngoscope* 56 596, 1946.

The Mouth, Tongue, Jaws and Salivary Glands

By JAMES BARRETT BROWN M.D.
and MINOT P. FRYER, M.D.

JAMES BARRETT BROWN is a Mark Twain Missourian, who became a pioneer disciple of the young specialty of plastic surgery. He was educated at Washington University and has devoted his professional activities to that institution and Barnes Hospital. His surgical imagination has opened this field of surgery in many directions and his exacting, meticulous ideals have provided results of the highest standards. Consultant in plastic surgery to the Chief Surgeon of the European Theatre of Operations, it was mainly through his efforts that emphasis was placed upon the care of American wounded with loss of tissue in hospital centers in this country.

MINOT PACKER FRYER, a pupil and colleague of Doctor Brown is a product of Brown University and The Johns Hopkins Medical School. He is a disciple of the training in the school of plastic surgery begun by Wilray Blair and developed by Brown at Washington University.

MOUTH

Soft Tissue Injury Injury in the region of the mouth does not respect arbitrary anatomic sites in its effect. Compound facial injury is used to describe the result of trauma of any severity since there is injury to the

soft parts skin subcutaneous tissue and fat and to the teeth and underlying bony support of the face and mouth. Division into the affected parts is made only for the purposes of description.

Contusions or bruises, can result from

Irradiation should be included as an agent of trauma to the lips and mouth. Large individual doses can cause acute inflammation of the part irradiated with progress to ulceration. Small repeated doses after a lapse of years can cause atrophy, telangiectasis, keratosis and ulceration with progression to carcinoma. Treatment is resection of the involved area and repair as indicated. If carcinoma has developed, the area of potential metastatic spread may require resection.

Infection of Soft Tissue. *Pustules* are the simplest form of infection of the soft tissues and may appear in crops. Cleanliness will usually control their spread. *Abscess* of the lips in the mouth or on the face is a localized collection of pus. There may be a mixture of organisms, but staphylococcus is usually present. Drainage is usually spontaneo-

us, but conservative opening may hasten subsidence.

Cellulitis may follow abscess formation in the upper lip when a localized inflammatory process becomes diffuse and spreading. Drainage from here along the angular vein to the cavernous sinus with thrombosis is a potential danger. It was particularly so before the advent of antibiotics. Treatment consists in local atraumatic management and large doses of antibiotics as the danger of intracranial extension and death is to be avoided.

Jaw abscess or abscess in the soft tissue around the jaws is usually related to the teeth, as an extension or penetration of a gumboil or root abscess or due to manipulation of an acutely ulcerated tooth. *Acute pericoronitis* (localized inflammation around

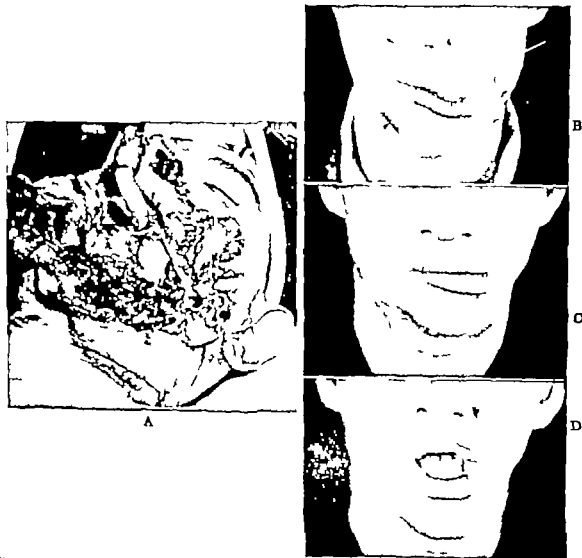


Figure 2. Soft tissue and bone damage resulting from shotgun wound. A, Soft tissue loss less than appears, as often occurs. Careful closure of lining of mouth and face done after minimal débridement around comminuted fracture of the mandible. Features restored as well as possible. B Segment of jaw blown out by close-range shotgun injury. Remains of comminuted mandible fixed and collapse of soft tissue prevented by internal wire in jaw. C and D Watertight mouth, good opening and closing action.

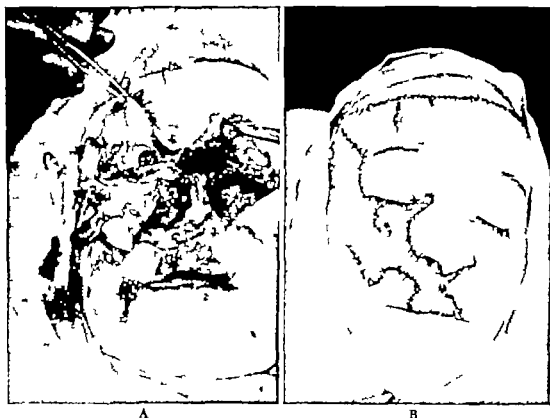


Figure 1 A, Extensive soft tissue laceration and tearing exposing eye, extending into nose and mouth, with partial avulsion of scalp. B, Minimal débridement. Repair in one operation by careful closure with fine sutures placed close to edge of wound and approximating known points. Features saved, eye protected, secondary revision of resultant scars unnecessary

layers of soft tissue. Skin edges are ragged and may be of questionable blood supply but the same principles of treatment are employed in the repair. All areas which might not survive are left for secondary excision rather than sacrificing a portion that would have lived. Subcutaneous fat may be actually exploded from the tissues and droplets will be seen scattered around the wound and over the clothing. This may occur when an individual is thrown forcefully from a car or suffers a similar accident. Deep cleansing of undermined areas by irrigation with normal saline solution is time consuming but invites early healing. Additional fixation may be secured by adhesive strapped across the wound. Steady even pressure is maintained, particularly over undermined areas, by fine-mesh gauze placed over the wound upon which surgical waste is held with a circumferentially wound soft gauze roll and the dressing is fixed in place with adhesive. This type of dressing is desirable over any area of repair. It is necessary over the site of the more complicated injuries, but may not be practicable over simple lacerations, as in the lip. Sutures can be re-

moved from most lacerations about the fifth day. Those in the mouth can be left longer. The wound may require support for an additional week.

Tetanus protection is routinely given. Prophylaxis against gas infection is given as indicated, but primary gas gangrene is extremely rare in the face. Antibiotics are administered as required.

Lacerations inside the mouth can be approximated to hasten healing but should not be tightly closed. Dependent drainage may be necessary. Repair of salivary ducts should be done primarily if possible, using a lacrimal probe or small plastic tubing for a splint. When repair is not possible, the proximal end of the duct may be brought into the mouth as a separate opening and sutured to the lining of the mouth or a local flap of mucosa approximated to this end.

Laceration of the tongue may not require suture, unless it extends through the edge of the tongue. The same is true of the soft palate especially for puncture wounds. A general anesthetic is usually required in children and the choice may be to do the suturing as a secondary procedure.

caused by lack of riboflavin and is manifest by superficial transverse fissures at the angle of the lips with very little inflammatory reaction. Usually 3 to 5 mg. of riboflavin a day is sufficient for cure but with difficulty of absorption 10 to 20 mg. of the crystalline riboflavin should be given by mouth.

Vitamin C deficiency is recognized in the mouth by the red swollen appearance of the

gums and interdental papillae; the manifestations may progress to actual scurvy, bleeding, ulceration and necrosis. Treatment is the administration of ascorbic acid by mouth or intravenously. Fresh citrus fruits and tomatoes are added to the diet, if tolerated.

Congenital Deformities and Anomalies. Cleft lip and cleft palate occur in some combination in one out of 600 to 1500 infants

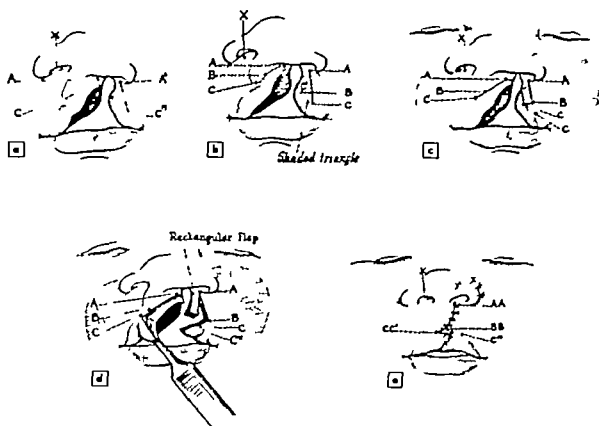


Figure 4 Design for repair of single complete cleft lip.

a. The V-excision operation. While the columella is held over straight, A is marked at the junction of the skin and vermillion border at the level of the base of the columella. X is in the same relation to the columella on the sound side. A bears the same relation to the ala on the cleft side that X bears to the ala on the normal side. C is on the mucocutaneous junction at the point where the vermillion border first begins to thin out. C' is on the mucocutaneous junction, the same distance from A that C is from A. To perform the V-excision operation, A is brought over to A and C' to C, after excision of the edges of the cleft.

b. The flap operation. The V-excision operation is marked out first. C' is on the mucocutaneous junction at the most medial point of good full vermillion border. B is on the line A-C' equidistant from C' and C. The incision is A-B-C' saving the amount of lip indicated by the shaded isosceles triangle. B is on the mucocutaneous junction, the same distance from C that B is from C.

c. The lines A-B-C and A-B-C' are lightly incised with a knife. The incision is carried upward from C' on the mucocutaneous junction to separate the vermillion border from the skin. This is also done on the other side at A, to keep any vermillion border out of the nostril floor. The triangle (see b) is to be undermined at the next step.

d. The lightly incised lines A-B-C and A-B-C' are cut completely through the lip with a stab blade, with care to keep the knife exactly perpendicular to lip. All angles should be completely opened. The vermillion border is inspected and any attached skin removed with a stab blade. The rectangular flap freed from A-B-C' must be loose enough to be rotated up 180 degrees into the nostril floor. Dotted stippling indicates areas of soft parts undermined.

e. C and C' are united and the vermillion flaps are interdigitated in a zigzag fashion, fitting them so that they be naturally together without any pull or stretching. Suturing is then continued on around the vermillion border and up the inside to the fornix. The little flap in the nostril is trimmed to fit with the one from the opposite side and they are sutured together to form the floor. A few key mattress sutures are placed through the ala to unite the lining and covering (which were separated during the undermining). The mucosa inside the lip is then closed.

a partially erupted third molar) is another common cause as are improperly drained fractures. Bone necrosis and sequestrum formation from a variety of causes may be responsible for abscess formation, e.g. irradiation, heavy metals, specific infections. Extension from contiguous structures, such as obstructed salivary glands, or metastasis in the blood stream from an abscess elsewhere in the body is unusual. The inflammatory process from any of these causes may extend into the neck and cause respiratory difficulty. Treatment consists of large doses of antibiotics and, whenever localization has occurred, drainage of the abscess. Localized collections, such as a gumboil, may be gently drained into the mouth. Otherwise, drainage is external through a small incision made below the jaw in a dependent position. Careful atraumatic exploration of the masseter and internal pterygoid regions is done and a drainage tract is established by leaving a loose pack in the wound for twenty four hours.

Ludwig's angina is a vague term applied to diffuse swelling of the neck which may compromise the airway and is the result of an undrained abscess. It has become practically nonexistent since antibiotics have been employed. This is a somewhat famous infection of earlier days because the Queen of Luxembourg was treated for it by Ludwig. However Gensoul antedated Ludwig by six years and designated the brawny edematous cellulitis as septic phlegmon of the floor of the mouth.

Canker sore or *ulcerative stomatitis* is commonly recognized and may be due to a virus alone or in combination with mouth organisms. Treatment consists in attention to mouth cleanliness, the use of mercurial or methylene blue applications and improvement of the general health. Pain may be severe, but not enough to require blocking

or section of the lingual nerve, as has been suggested.

Stomatitis in many forms may occur and may be a manifestation of a general debility such as anemia or vitamin deficiency. If Vincent's organism is the causative agent, control is achieved mainly by cleanliness and a soapy toothpaste. The fundamental deficiency in general health should be corrected.

Noma is a rapidly progressive gangrenous stomatitis, which is rarely seen, and is an expression of general debilitation or of some blood or metabolic dyscrasia (Fig. 3). Treatment is obviously aimed at improving the general condition and large doses of antibiotics are given. Local treatment is of little value. The former use of caustery excision and of x-ray treatment is of little help in comparison with antibiotics.

Specific infections due to tuberculosis and syphilis are only rarely seen. *Tuberculosis* can cause deep dirty ragged ulcers in the mouth, most commonly on the central portion of the tongue and treatment is that of the underlying disease combined with the use of the newer antibiotics. *Syphilis* or a gumma may be the primary lesion. This produces a foul, discharging, dirty ulcer without the surrounding characteristic hardness of carcinoma. Systemic antisyphilitic therapy is indicated, but healing may be marked after the initial dose of the medication employed. *Granuloma inguinale* with involvement of the lips and mouth has been reported.

Vitamin Deficiencies. *Vitamin A deficiency* may be manifest as keratinization and hyperplasia of the gums and is treated by administering large amounts of the vitamin, 25,000 units daily. However absorption of the vitamin, particularly in the debilitated, may be limited.

Deficiency in vitamin B complex can cause angular stomatitis and glossitis. *Cheilosis* is



Figure 8. Noma. Progressive gangrenous loss of entire cheek.

tion and/or development of faulty speech habits. All inclusive rules cannot be made as to time or type of closure but decision must be made for each individual patient by a competent surgeon with consultation as needed with the general dentist, speech trainer otologist orthodontist and prosthodontist.

Reconstruction of single cleft lip and nose deformity can be followed in Figure 4. In general a V-excision operation is first marked out and then a small triangular flap is designed just above the vermillion border on the cleft side to save lip at this point, which is necessary for fullness and a normal relationship to the lower lip. A summary of the objectives of any operation for the repair of a cleft lip would be a full upper lip in advance of the lower lip with a flexion crease above

the vermillion border (Fig. 5). The lip itself should be full without notching, the floor of the nose should be closed at proper level, the ala should be symmetrical and of same level as normal and its direction should be with the curve toward a straight columella. The operation outlined in Figure 4 provides a method for the routine accomplishment of these objectives. The simplicity of the fundamental markings readily allows the repair to be fitted to the deformity. Variations found in each lip can be taken care of on an individual basis.

Repair of double cleft lips (Figs 6 and 7) is usually not done until the infant weighs about 10 pounds, because of the technical difficulties of the repair and for the safety of the child. Repair of a double cleft lip is about twice as difficult as that of a single

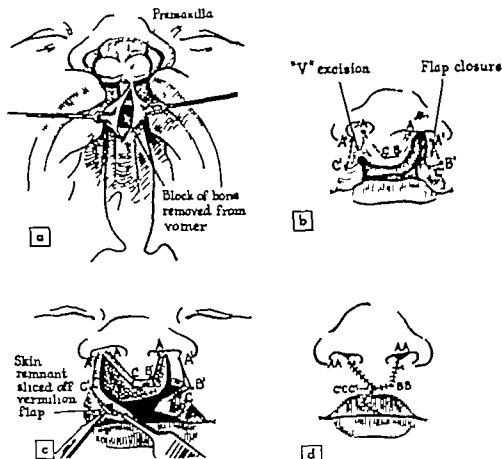


Figure 6 Design for closure of double cleft lip.

a. When the præmaxilla is too far forward to permit closure of the lip, it may be set back by submucous excision of a block of bone from the vomer. This removal of a block, rather than a wedge, permits the pushing of the bone directly back like closing a drawer rather than tilting it back. This factor is of some advantage, as the finished lip should slant forward in the profile view from above downward.

b. Flap closure is done on the total side and a V-excision operation on the partial side.

c. Both sides of the lip are opened up, going completely through the lip with a stab blade knife and using a perpendicular sawing motion. Any skin remnants are sliced off the vermillion flaps. The vermillion border of the prolabium is cut loose from the skin and turned back all around.

d. The closure is done with many fine silk sutures, put in not more than 1 to 2 mm. from the wound edges and about as far apart. Any stay sutures are put in from the inside and are not visible. The lip is closed by interdigitating small flaps.

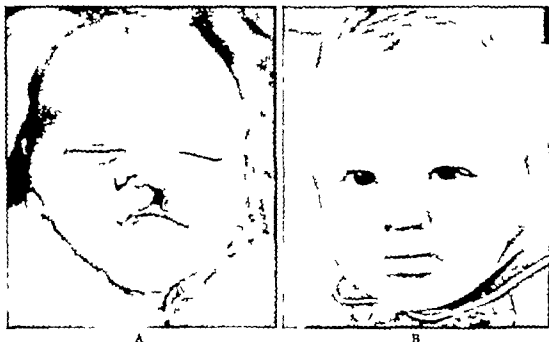


Figure 5. Complete single cleft lip. A, Wide defect of lip and usually associated marked deformity of nose. B Repair of lip and nose done in one operation, as shown in Figure 4. Palate to be closed later

born alive. They may be accompanied by some other deformity. The cause is unknown, and though often present, there should be no parental feelings of guilt. In families, a tendency may follow recessive mendelian progression. Mental capacity of children with this deformity is the same as in the population as a whole. Actual performance in school is usually superior, probably owing to a compensating drive because of the physical deformity. The best possible surgical repair with the steadying influence of constant patience and help in the home usually produces the best of citizens. Over sympathetic parents, because of their own false feelings of guilt, hinder the child's progress to social adjustment more than does any single factor except social agencies which brand him as "cleft palate" and thereby single him out from any other children. As in any child, a lifetime of productivity, happiness and acceptance by others more often stems from a home where there is love and a feeling of being wanted.

Embryologically the lip and upper alveolus are a product of the fusion of two lateral maxillary processes and a central globular segment; this fusion occurs between the ninth and thirteenth week in the embryo. All varieties of clefts of the lip and palate—single or double, complete or incomplete cleft lip and palate or any combination thereof—can result from failure or incomplete fusions of these three elements. Single complete cleft lip and cleft palate is the

most common type. Double cleft lip and palate is more common in males, but incomplete cleft of the palate alone, usually accompanied by an insufficiency or shortness of palate and a somewhat retruded lower jaw is more common in girls.

Repair of the single cleft lip can be done any time after birth if the infant is in other wise good condition, and it helps to initiate a normal up-bringing if the child can leave the hospital with the mother. Double cleft lips are repaired when the child is a month old or has shown a real tendency to gain weight. Reconstruction of the nose at this initial operation is as important as repair of the lip. The excellence of the ultimate surgical result is dependent on the primary repair. No number of secondary procedures can compensate for the failure to construct the best possible lip and floor of the nose at this time. This initial repair may set the pattern for the child's whole life. It is not the time or the place for the occasional surgeon to try out his skill.

The palate is usually closed in the latter part of the second year. If done before, tooth buds located toward the midline of the palate are disrupted before they can migrate to the alveolus. The advantages of one's own teeth over a dental plate do not need to be explained to anyone. There may be orthodontic reasons for postponing palate repair until the child is four years old, but these should be weighed against the possibility of hearing loss from constant middle ear infec-

childhood dental care with the preservation of every available tooth. Orthodontia is necessary in some degree in most children with a cleft palate. Teeth in the line of the cleft are usually crooked and require straightening. Maintenance of the dental arch is im-

portant in some children. Speech training can best be carried out in the understanding privacy of the home after parent and child have had a few lessons or even a single lesson, from a good speech trainer. Hearing measurements should be made if there have

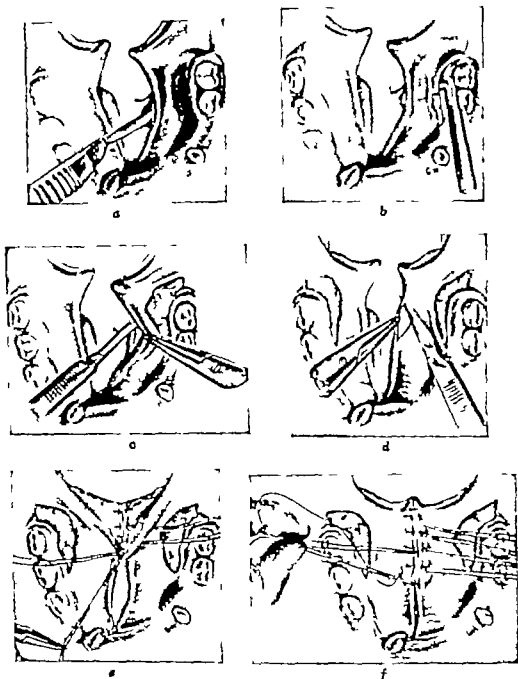


Figure 8. *a*, Mobilization by Warren's approach. *b*, Mobilization by elevation of the mucoperiosteal flaps. The point of the angle elevator enters the lateral incision just in front of the postpalatine foramen, and then by working forward, the mucoperiosteum and with it the palatine artery can be freed from the underlying bone as far forward as the incisive foramen. *c*, Mobilization by division of the palate aponeurosis. *d*, Denudation of the cleft border by paring. *e*, Suturing the uvula and upper surface of the velum. *f*, Suturing the mucoperiosteal flaps. The mucoperiosteal palate flaps are united with vertical mattress sutures, bringing several millimeters of the raw upper surfaces into contact. It is at the junction of the velum with the mucoperiosteal flaps that one is most likely to have failure of union, either because of insufficient relaxation or a deficient blood supply and it is to be remembered that tight suturing interferes with the blood supply. If there is any doubt of the integrity of the major palatine arteries, it is safer to suture as far forward as the original relaxation will permit without tension and to do the remainder at a subsequent stage, as failure of union is less catastrophic than a slough or a distorted flap.

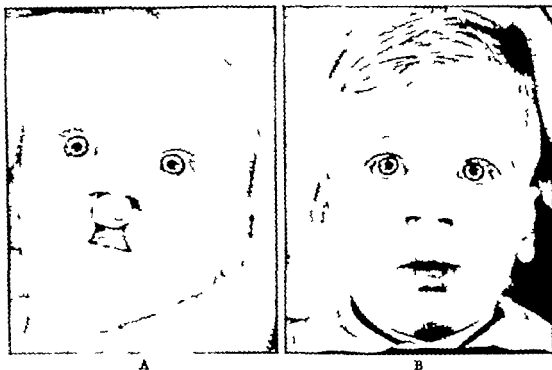


Figure 7 A, Double complete cleft lip and palate. B, Lip and nose repaired on both sides in one operation as shown in Figure 6. Palate subsequently repaired.

cleft lip and the result is, unfortunately about one-half as good. The baby is held up at a 45-degree angle and fed by means of a syringe to which a short piece of rubber tubing has been attached. Gavage is rarely necessary if nursing personnel or the family has the patience to use the syringe method.

Embryologically the deformity is due to failure of fusion of the median globular and the two lateral processes of the maxilla. The degree of cleft on either or both sides is the result of the incompleteness of fusion and so there can be double incomplete clefts, complete clefts on one side, incomplete on the other or double complete clefts. The median globular process becomes the premaxilla prolabium segment which is the central part of the lip and palate. This is an integral part of the lip and palate and is necessary for closure. If possible, the lip is closed without disturbing it unless it is too far out of position to allow for closure or unless after closure the pressure of the closed lip will bend the septum and occlude the nasal airways. In such cases it is preferable to set this segment back in a more optimal position, as shown in Figure 6 a.

Closure of the cleft palate is outlined and described in Figure 8. Adequate surgical repair is the only means by which normal physiologic relationship in the mouth and pharynx can be achieved. Dental prosthetics are an expensive supplement but are no sub-

stitute for direct muscle-controlled components of the palate.

Usually the child with a cleft palate tries to talk at a later age than is considered normal. In addition to the cleft, there may be some unknown insufficiencies of development which might explain the inability of some children with a cleft palate to talk as well as others.

The time to close the cleft palate, as previously stated, is usually during the latter part of the second year before definitive speech habits have developed and after the tooth buds have migrated from the palate. Closure may be postponed until the fourth year because of the often cited observation that facial growth centers may be injured by operating before that time. This recommendation has been made on the basis of poorly executed surgical results and those operations which depend upon molding of the palate bone for closure.

Repair of the incomplete cleft palate which includes closure and elongation is illustrated and described in Figure 9. The incomplete cleft palate is usually shorter than the complete cleft and, unless adequate length is provided, speech may be poorer than expected in the complete cleft.

Ancillary requirements of the child with a cleft palate include general dentistry which seems to be the most neglected. A child with a cleft palate needs the usual

accumulation of food in crevices and pock-ets. Dental prosthesis may be needed if there is no tissue available for closure. Repeated operations on the soft palate add scar with each procedure and this soft structure eventually approaches the character of an inert prosthesis.

Benign Tumors of the Lips and Mouth
Benign tumors can arise from any of the tissue elements in this location, as is true elsewhere in the body. That is *papillomas*, *fibromas*, *lipomas*, *myomas* and *nevi* occur, as do benign tumors arising from any mixture of the basic histologic parts. They are usually easily recognized (Figs. 12 and 13). Treatment is usually surgical excision for cure and microscopic confirmation of the clinical diagnosis. This is not complicated except in the removal of large tumors located where features may be included or the airway is a problem. Closure of the defect may be possible following removal of large benign tumors, but coverage with a graft or substitution with a direct flap or numerous secondary procedures may be

required for restitution of function and the best possible final appearance.

Nevi are divided into several different types, mostly on the basis of microscopic appearance. *Intradermal nevi* are often raised from the surface may contain hair and microscopically show the majority of the nevus cells in the derma. *Junctional nevi* are usually flatter do not contain hair and show nevus cells concentrated at the junction of the dermis and epidermis. *Combined nevi* show elements of both of the above. *Intradermal nevi* may never become malignant but *junctional nevi* do and possibly the combined variety may. *Blue nevus* contains characteristic cells and although it looks grossly very dangerous it probably never becomes malignant. *Sclerosing hemangioma* and *subepidermal nodular fibrosis* may grossly be mistaken for nevi. Treatment is surgical excision with adequate margin of any nevus which is being irritated, bleeding, growing or otherwise changing in character. It is practically impossible to remove all nevi on everyone, but

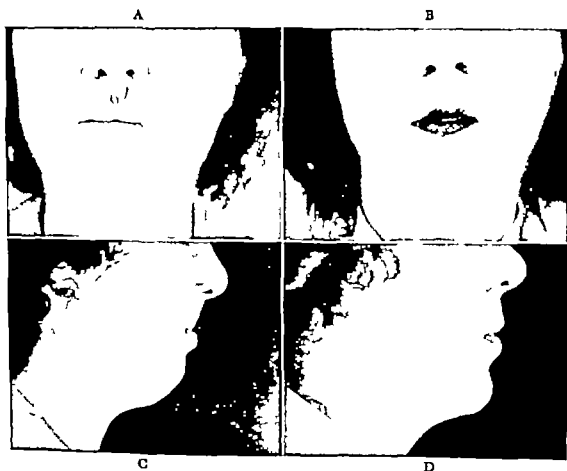


Figure 10. Cross-lip flap establishing proper balance between upper and lower lips, as diagrammed in Figure 11. A, Tight scarred upper lip previously repaired, with redundant lower lip. B, Balance created between lips by the addition of lower lip into upper lip. C, Profile emphasizes marked imbalance. D After cross-lip flap which added bulk into upper lip from lower lip.

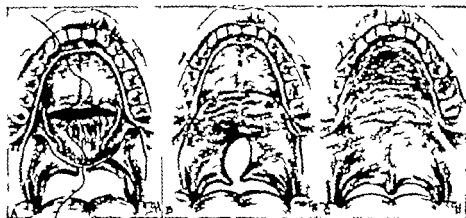


Figure 9 A, The whole palate has been loosened from the bony palate the nasal mucosa opened, the major palatine arteries preserved and the suturing started to carry the palate back. B, The palate is held back with three to five sutures and the exposed bone in front is allowed to cover over. Healing is usually complete in one month. C, The elongated palate is shown, with the posterior opened space much smaller than it was originally.

been repeated middle ear infections and decision may be made for earliest possible palate closure. Prosthetic work is usually confined to tooth replacement and, rarely it may be required when surgical closure has been improperly done. Most dentists can usually add a plug of some plastic material in any position on a partial or complete upper dental plate.

Pharyngeal flaps may be constructed and attached to the palate when there is insufficient length. Pharyngoplasty attempts surgically to duplicate the ridge in the posterior pharyngeal wall, called a Passavant's pad, seen in some patients with good speech.

Efforts to improve any residual deformity in the lip and nose should be carried out until the best possible result is obtained with the tissue available, so that the patient may be as close to normal as is surgically possible. The best possible initial repair of a cleft lip may require some secondary work and, although the change may be comparatively slight, it is worth while if it makes the patient look more normal. However one operation each on the lip and on the palate if properly done, is all that is usually required for the best possible result.

Irregularities in the lip or vermillion border may require smoothing out, or advancement of the upper lip on itself, or disproportion in the size or shape of the nostril may be improved. A cross-lip flap from the lower lip to the upper lip may be required for balance around the mouth because of disproportion of the lips, particularly in the profile (Figs. 10 and 11). The protruded lower lip and retracted upper one give the

most noticeable deformity more often in the double cleft lip than the single.

Orthodontia often is required to expand and maintain the upper dental arch and to bring the anterior forward. But if there is not sufficient success from orthodontia, ramisection, to set the lower jaw back into a more favorable occlusion, may be required to avoid the appearance of prognathism. This is especially true if the lower jaw happens to be large and forward—prognathic.

Prognathism is protrusion of the lower jaw in relation to the upper jaw. Repair depends on the occlusion. The lower jaw may be placed in proper relationship with the upper by closed section of the ascending ramus of the mandible. If the occlusion is no problem, the bone in the symphysis region can be cut down surgically.

Micrognathia is a short underdeveloped mandible. This may be related to some disturbance in the growth centers of the jaw. Repair as in prognathism, depends on the occlusion.

A *short columella* is a characteristic deformity of most complete double cleft lips, resulting in a snubbed-nose appearance, the tip of the nose being down on the lip. The columella can be elongated by a flap taken out of the upper lip and advanced upward along with the tip of the nose. The defect in the lip is carefully closed or covered with a full-thickness graft from behind the ear or neck to obtain the best color and character match of the skin.

Defects in the hard palate alveolus and fornix should be closed surgically whenever possible to stop leakage of air and food or

resection of the part and a serious operation for the infant.

Cavernous angiomas are beneath the skin but may involve it. They are composed chiefly of larger vessels (blood spaces), appear bluish red, are easily compressible and slow growing, may be first noticed at any age and markedly distend with dependency of the part. Microscopically they are composed of thin walled vessels or spaces but they can be an extreme example of benignancy in respect to microscopic appearance and malignancy because of location. A large cavernous angioma of the palate, pharynx, tongue or floor of the mouth gradually enlarges with increasing age and unless surgically controlled, can kill any time by rupture and drowning the patient. Sections of the tumor removed at postmortem examination show simple enlarged vessels. Lip involvement with large vessel angioma presents a problem of increasing deformity with growth that necessitates control of the tumor with preservation of a feature.

Treatment of the small cavernous angioma may be by excision of the growth or cautery destruction. As features are involved, usually multiple stages are required for control. "Multiple suture technique" has been of real benefit in those angiomas involving the lip, tongue or elsewhere in the mouth by collapsing many of the vessels, yet preserving the feature or making subsequent excision easier. This consists of placing chromic catgut sutures directly into the growth from the surface and tying them on the surface to obliterate the spaces. The method depends on occlusion by healing of the thin collapsed wall. Interstitial irradiation has been helpful in those angiomas with a small vessel component in which eventual excision is indicated, since the

caliber of the larger vessels is reduced by their collapse and scarring. The larger spaces themselves are not much affected by the irradiation unless a cauterizing dose is used, which should be avoided since the principle of treatment is to control the growth but leave the area intact.

Port wine stains are the third most common types of angiomas. These are not tumors but rather congenital enlargements or excess of the small vessels in the skin. Mucosal involvement usually requires no treatment, but in the skin of the lip treatment may be indicated because of the appearance. Superficial abrasion has been of some limited value if this addition of scar is of no significance. Cosmetics may cover the redness. Excision and replacement with a graft is the alternative, but it should not be done until all possible spontaneous fading has occurred and until the patient is old enough to understand the cumbersome process and the scarring that results.

These anomalies or tumors may all occur in mixed variety and have to be dealt with accordingly.

The cavernous types tend to get worse with age from gradual destruction and form phleboliths that may be painful and become more prominent on the surface and cause increasing deformity. They may occur deep in the face, pharynx, tongue, parotid and pterygoid regions, go into the temporal fossa and cause severe damage and trouble. There may be thrill and bruit from arterial leaks and, when these occur in the face and around the ear, they are very uncomfortable.



Figure 12. Irritated papilloma of tongue.



Figure 13. Inclusion cyst of tongue.

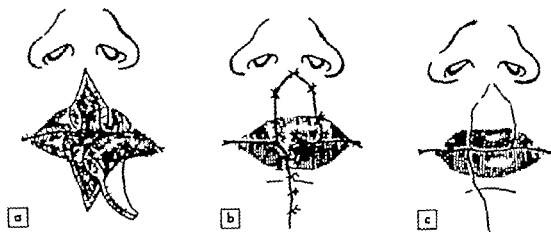


Figure 11 Diagram of lower lip flap adding bulk and creating proper proportion between lips. a, Flap removed from lower lip in triangular shape to allow for closure and, if necessary, it could be designed to fit into the floor of the nostril. b, Lower lip flap in place sustained by inferior labial artery in the pedicle until flap can pick up its own blood supply from the upper lip. Lower lip closed. c, Pedicle has been cut and vermilion set into proper position.

the above criteria can be used in judging the necessity for removal. All junctional nevi should be removed if they could be recognized grossly but final classification is usually dependent on microscopic appearance.

Cysts *ranulas* *angiomas* *lymphangiomas* *keratoses* *leukoplakias* *epulides* and *mixed tumors* are benign tumors but their treatment may present particular problems.

Cysts are usually of the retention variety readily recognized, and are seen in the lips or anywhere inside the mouth. If small, they are completely excised and the defect closed. Larger cysts may require staged removal or simple "unroofing" may suffice. It is apparent that there may be recurrence of swelling or obstruction to any of the many glands in the area of operation and the patient should be made aware of this possibility. *Sebaceous cysts* occur in the lips as elsewhere in the body and require excision for cure. *Dermoid cysts* which are deep inclusion cysts may occur in the lips and the floor of the mouth and require removal.

Ranula is a more complicated type of cyst as far as removal is concerned. It is recognized as a bluish swelling in the floor of the mouth, pushing up the tongue or even extending into the neck. It probably results from some anomaly of the accessory salivary glands and ducts and may develop in a child or not show up until adult life. The fluid is usually clear and may be viscous. *Ranulas* have been controlled by developing permanent drainage with a seton, that is, a heavy silk or wire suture, left in place until the lining of the cyst heals to the oral mucosa. Complete surgical removal may eventually be necessary but this is often of such

magnitude because of the extent of involvement that preliminary control with a seton or partial removal and packing may be safer.

Angiomas of the different types occur in the mouth and may be an actual threat to life because of position. *Growing hemangiomas* called *arterial hemangiomas* because of their bright red appearance, occur about the mouth or lips and are raised from the surrounding surface but are in the skin (Fig. 14). These hemangiomas are seen in the newborn or infants and may at any time cease to grow and eventually control themselves so that there may be some question as to whether they are true neoplasms, vessel anomalies or hypertrophies. Others of this same type however may proceed to ulceration, recurrent bleeding and on occasion lead to death of the infant. They should be treated before destruction of a feature occurs. Microscopically they are composed of growing small blood vessels and endothelial cells, but the microscopic picture has given no clue as to the eventual activity of the tumor. Treatment should not be withheld if there is any question of progression of this type of angioma and usually a minimal dose of interstitial irradiation, i.e., radon seeds implanted directly into the tumor will control the growth. Small growing hemangiomas can be excised or completely destroyed with the fine cautery but if a feature is involved it can be saved with interstitial irradiation. Radon seeds of 0.1 millicurie or less implanted directly into each cubic centimeter of an extensive growing hemangioma in the tongue or spread out in the palate, for example may avoid wide

the mouth or tongue may be excised and the remainder carefully followed in frequent examinations. If a large area requires total excision, it may be done in stages or a skin graft will be required for coverage of the defect. Irradiation seems to be of questionable permanent value in the treatment of leukoplakia.

Epulis is a benign tumor arising from the periodontal membrane of a tooth and its character depends upon the predominance of fibrous tissue, blood vessels or giant cells in its make-up. It can be then white or red, soft or hard, but is usually pedunculated. Treatment is excision of the tumor and usually removal of the associated tooth and its periodontal membrane.

Mixed tumors occur in the mouth, palate, tongue and lips. Biopsy may be required for diagnosis of large mixed tumors, but small tumors can be completely removed for cure and for microscopic confirmation of clinical impression.

Malignant Tumors of the Lips and Mouth (Figs. 15 to 19) *Basal cell carcinoma* is the most common malignant tumor occurring in the upper lip as contrasted to the lower lip where squamous cell carcinoma is the most frequent. Basal cell carcinoma probably does not metastasize as such, but, contrary to the usual general impression and report of microscopic appearance this tumor can kill as surely by local invasion and persistence as can squamous cell carcinoma. Failure to metastasize is no indication of benignancy. Prompt, aggressive treatment is necessary for control. These growths have been reported as metastasizing, but the metastatic lesions probably come from basal squamous growths.

Basal cell carcinomas arise either from sebaceous glands or hair follicles in the skin and do not occur in the mouth. They can

more accurately be divided into three groups on the basis of gross appearance as to expected behavior rather than by the microscopic picture.

Solid raised basal cell carcinomas are slow growing tumors with a tendency to grow up from the surface of the skin and probably are the reason the basal cell carcinoma is viewed with little concern by many. However it can change character at any time grow rapidly and become invasive though previously dormant for years. These tumors are easily excised when small. Radiation may be effective. Later when extension is wide plastic surgery restoration of the area may be necessary following excision or radiation.

"Field fire" basal cell carcinoma has a flat healed central area surrounded by a slightly raised smooth edge of actively growing tumor. Remnants of tumor may remain in the central healed section. "Basal cell carcinoma of multicentric origin" is a term which has been used to describe this tumor. Treatment is excision of the entire area and replacement as necessary. Radiation may be used, but, if a biopsy is necessary time and expense for the patient may be saved by one surgical procedure.

Invasive basal cell carcinoma was well named rodent ulcer by older clinicians because of its dangerous persistence. In the upper lip it commonly arises or seeks the nasolabial fold where it eventually invades the pyriform recess and, unless stopped, proceeds to kill the patient over a long, painful debilitating course. Often only a small portion of the tumor presents above the surface of the skin, while a solid, larger invasive component is located beneath the skin and is growing in all directions. The superficial part may or may not ulcerate. Treatment must be aggressive. Radical ex-

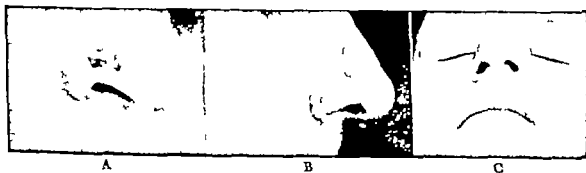


Figure 15 A, Basal cell carcinoma of the ala of the nose with involvement of the cartilage and lining. B, Excision and immediate repair with composite graft cut to pattern from the ear. Composite graft supplies lining for nose, skin for outside of the rim with cartilage contained between these two surfaces. C, View of nose from below showing support to graft by strut of contained cartilage in the composite graft. One operation. Staged flap repair avoided.

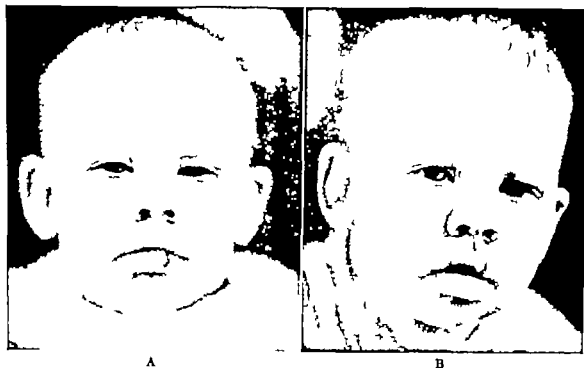


Figure 14 Growing, bright red, arterial hemangioma. A, Raised, red, growing hemangioma started as tiny red spot in lip. If allowed to progress, may destroy entire lip. B, Control of tumor by light dose of interstitial irradiation. One-tenth millicurie gold radon seeds were used. Lip saved. One procedure.

It may be mentioned again that these constitute a typical example of the microscopic pathologic appearance having no relation to the seriousness of the lesion. These angiomas may be truly malignant by position and extent.

Lymphangiomas can involve the lips, tongue or be anywhere in the mouth. Usually they extend into the neck and consist of multiple, flat, endothelial-lined spaces. They may be associated with a hemangioma or the tumor may actually be a mixture of both, i.e. lymphangioma-hemangioma. Sabins' theory of blood vessel development could explain this association. Lymphangiomas usually appear at an early age but may not show up until later on in life. Small confirmed lymph tumors not involving features can best be surgically excised. Extensive involvement usually requires more than one operation for the safety of the patient. Aspiration and obliteration with escharotics are possible. Limited excision, breaking down the multiple septa and packing with an escharotic pack is often the procedure of choice. *Hygroma* is a lymphangioma of the neck and may extend into the mouth, face or parotid region. Acute respiratory obstruction due to encroachment or pressure on the airway requires immediate relief.

Keratosis are rough, dry raised areas which appear on the lips with advancing

age. They are more commonly noticed in persons whose faces have been exposed to the out-of-doors. Certainly most keratoses never become malignant, but likewise most squamous cell carcinomas of the lips have developed on a pre-existing keratosis. Treatment is simple cautery destruction of the keratosis or if there is any question of change to carcinoma, then excision for microscopic examination should be done. This simple surgical procedure is much more important in the over-all control of cancer and will save more patients than any extensive ablative procedures that can be done after cancer has developed.

Leukoplakia is readily recognized as flat or slightly raised white patches occurring anywhere in the mouth. It may be a small solitary spot or involve most of the mouth and tongue and may be a response to some irritation. Progression to carcinoma is possible any time and the patient should be cognizant of the potentiality of this lesion. Initial treatment consists in the removal of any known irritant and mouth cleanliness. Smoking should be stopped, or at least cut to a minimum. The teeth should be cleaned, cavities filled and any sharp teeth or fillings removed or smoothed out. Small patches may be excised for microscopic examination. The more dangerous-appearing or piled-up areas in extensive involvement of

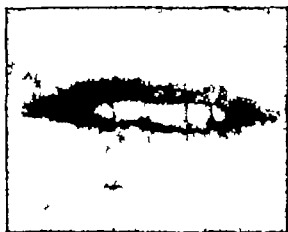


Figure 18. Carcinoma of the lower lip. Flat ulcer type, edges piked up. Oval excisional biopsy should be done which may suffice locally or further excision or irradiation may be required. Decision as to time for bilateral upper neck dissection can be made any time.

for infection dyscrasia or irritation then it should be removed entirely or as a specimen for microscopic diagnosis.

As to treatment of all malignant or potentially malignant tumors, cure depends on staying outside of the tumor field in the treatment, whether surgical or radiation. If the tumor field is entered or some of it left behind in a small area, or if a small area has not been controlled by radiation, then a primary cure is not obtained.

For recurrent tumors the same rule applies of still staying outside the tumor field.

For metastatic areas the same rule still applies. The ideal operation for any advanced malignant lesion is block resection of the growth and its regional lymphatics. Unfortunately, there are few such possibilities, but carcinoma of the buccal mucosa presents one of the best possibilities for this procedure.

Neck dissection, or removal of the lymph-bearing area in the neck which drains from the lip may be the most important factor in the patient's cure. If neck metastases are palpable in a patient with carcinoma of the lip, a neck dissection should be done immediately or as soon as it can be determined that the local lesion can be controlled. No blanket rule can be made as to which patients without palpable metastases should have a neck dissection, except that all patients with a carcinoma of the lip are and remain candidates for a neck dissection as long as they live. This statement is substantiated by numerous long term follow ups. Statistics are available showing that 80 to 90 per cent of patients with carcinoma of the lip do not have neck metastases, but

the physician in charge of the patient with a lip carcinoma should consider this patient as he would a member of his family who might be in the 10 or 20 per cent group in which metastasis to the neck occurs. Decision as to the necessity for neck dissection must be made on an individual basis. Various factors help with this decision, among which are age—the young patient is more likely to have early metastasis than is an older one duration—the longer the carcinoma has been in the lip the more likely it is that metastasis has occurred to the neck nature of the lesion—in the small ingrowing ulcer neck metastasis is more likely than in the more outgrowing type of carcinoma, availability for follow-up—some patients will be less faithful in returning for follow-up than others, for one reason or another and in these it would be safer if a neck dissection were done—experience of the surgeon—the experience and ability of the surgeon probably comprise the most important single factor since the usual bilateral upper neck dissection done for carcinoma of the lip should be associated with no mortality and require around a week's hospitalization.

A bilateral upper neck dissection is usually adequate for control of the area of spread from a carcinoma of the lip. Depending on the location of the primary tumor metastasis can be to one or both sides of the neck. The operation can easily be varied to include all the lymph nodes on one side with the jugular vein, if necessary and the upper part of the neck on the other. Again, selection of operation is made on an individual basis. A bilateral upper neck dissection includes all of the nodes to which carcinoma of the lip usually metastasizes first. This would embrace the region of the omohyoid muscle up to the jaw on both

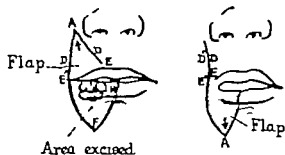


Figure 19. Cross-lip flap for immediate reconstruction of lip following excision of infiltrating, deep carcinoma. Area E' F H in the lower lip is excised and the defect replaced with E A E (shaded area) from the upper lip and D' D closed. Further revision at the corner of the mouth may be necessary.



Figure 16. Carcinoma of lower lip. Outgrowing fungating epidermoid carcinoma of lip.

cision may be combined with irradiation. Interstitial irradiation with the use of 0.5 or 1 millicurie gold radon seeds may stop the deeper extension and avoid some secondary reconstruction. Composite grafts can be used for immediate total reconstruction following removal of tumors involving full thickness of the nose, thereby avoiding complicated flaps and numerous operations (Fig. 15). Thoughts of reconstruction are always secondary however to the most effective means for controlling the tumor.

Basosquamous carcinomas do occur in the upper lip but are more common in the upper part of the face. This classification is on the basis of microscopic appearance in which elements of both basal and squamous growth are present. Grossly they look like solid basal cell carcinomas which have a history of fairly rapid growth. They do metastasize, but do so as squamous cell carcinomas. Treatment is surgical excision of the local tumor and the regional nodes, as indicated. Irradiation may be effective in cancer dosage and wide application, but the tendency to recur is high.

Malignant melanomas occur in the lips

and the possibility of effective control or cure is relatively high. The local lesion can be radically excised and the potential lymph-draining area resected, if necessary en bloc.

Squamous cell carcinoma is more common in the lower lip than on the upper and is easily recognizable. There is no question of its potentialities. It usually occurs as a fissure flat ulcer or chronic leukoplakia that finally becomes malignant or a fungating mass. There need be little discussion as to the treatment of the primary tumor. Biopsy is required and removal of the entire tumor at this time is indicated if the extent is not too great and there is real question about the malignancy of the individual tumor. Complete excision for the biopsy may be the only treatment required, completed in one operation, and availability of the entire tumor may invite a more positive microscopic report from the pathologist. After a positive diagnosis has been made, decision as to the necessity for more radical excision of the lip or some form of irradiation can be made. V-excision of carcinoma of the lip is not planned for cure, but rather as a repair of a deformity.

Interstitial irradiation, as 0.5 millicurie of gold radon seeds and external x ray and radium therapy has repeatedly saved lip and reduced the necessity for secondary substitution operations.

It has been repeatedly stated that all carcinomas of the lip should be cured, and so they should. But they should also be prevented by early removal of suspicious lesions. As the safest procedure for all lesions of the face, mouth, tongue and jaws, the following may be taken as fundamental. If a lesion persists for two weeks and, at the outside, four weeks under usual treatment

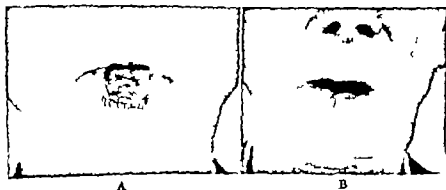


Figure 17. Carcinoma of lower lip. A, Widespread involvement of lip by epidermoid carcinoma. Carcinoma did not show invasive tendency. Oval cautery excision done. B, Defect in lip following excision of carcinoma allowed to heal and spontaneously reconstitute itself. One operation. No secondary reconstruction necessary in this patient.

usual symptom is pain and diffuse enlargement of the circumvallate papillae is seen sometimes localized to one area or the other.

Irritation by strong chemical applications, cauterization or removal is contraindicated as the scar produced may make the process worse. At present there is no known preventive or medical cure. Treatment consists of general mouth cleanliness including no smoking, care of sharp teeth or fillings and, of course, close observation and the removal of any active lesions. Sometimes the lesion may be widespread and thick and approach the process of verrucous carcinoma the whole surface of a cheek or tongue may be involved.

Removal and repair with a free skin graft may be required. These widespread deep involvements require active treatment or removal, since they will frequently become cancerous. This is one example of being able to remove or control a disease but not knowing how to prevent the tendency.

Carcinoma of the Tongue. Carcinoma of the tongue is usually seen as a fissured ulcer with tissue piled up along the edges (Fig. 20). The margins of the ulcer may be overhanging, bleed readily, may be sloughing and may be painful. The lesion may be a solid tumor but usually ulceration has occurred. The most common location on the tongue is along the edge in the molar area, but the lesion may occur as a solid mass in the center of the tongue or anywhere else. The ulcer or solid component may extend to the floor of the mouth, over the alveolus, up the tonsillar pillar or backward to the epiglottis or pharynx. When it is located in the back of the tongue, there may be no pain until there is extensive development and even metastasis. This area may be practically a "silent one" as far as discomfort is concerned and the first symptom may be the appearance of a metastatic lump in the neck.

Lesions of the face, mouth, tongue and neck can be easily seen and palpated, biopsy is easily done and the presence and nature of none of them need be missed.

Any ulcer or area of growth in the mouth should be biopsied if present for three weeks and if it has not responded to usual methods of treatment for inflammation, nonspecific or specific, or for lesions of dyscrasia, allergy or drug reactions. If there is no question as to the diagnosis or real evidence of growth, there is no reason to wait until the end of this arbitrary period. There also should be no further delay if the patient has

waited this maximum period before being seen for the first time. Suspicion by the examining doctor or dentist that carcinoma may be present is as important in the control of cancer as encouraging the lay public to be on the lookout for such occurrence. Likewise wishful thinking that a lesion is not cancer has no place in dealing with lesions that possibly are cancer. Progression to inoperability may be very rapid once the patient has noticed something wrong.

The method of taking the biopsy specimen is important. Small lesions may be totally excised since carcinoma may be present in a small area of leukoplakia or of an inflammatory reaction and if carcinoma is not present total excision may effect a cure. Adequate sections should be taken from the most suspicious parts of larger tumors. Repetition of a biopsy is preferable to overlooking a carcinoma.

The cell type is usually that of the squamous variety with more or less pearl formation. In or around the tonsillar fossae there may be little hornification of the cells. These tumors have been called transitional cell carcinoma or lymphocarcinoma since they contain small round cells. Tumors of this pathologic type, as would be suspected, are particularly sensitive to irradiation but tend to recur and metastasize readily. The more differentiated the individual tumor the better the chances of control. Adenocarcinoma is infrequent but occurs usually in the body of the tongue. Sarcoma of the tongue is rare.

The position on the tongue is mentioned because of prognosis. The further forward



Figure 20 Carcinoma of the tongue. Typical ulcer with a solid component beneath a surrounding visible area. Primary carcinoma could be controlled with 1 millicurie of gold radon seeds implanted directly into the tumor and area of potential invasion. The patient will need a complete neck dissection.

sides and the superior deep cervical chain of nodes with the contents of the submaxillary triangle en bloc. There is minimal scar and no actual disfigurement. The term "suprahyoid dissection" does not describe the operation because in this procedure a serious potential area is left in the center of the neck. The term "upper or supraomohyoid dissection" is preferable.

It is almost needless to state that a cure for carcinoma would be as welcome in this field as elsewhere. But until this is forthcoming, radical surgery neck dissection and sufficient restoration must be included in the procedures that may give the patient a chance of survival.

Sarcoma of the mouth and lips is rare but may occur. Usually it appears as a solid, hard enlargement which does not tend to ulcerate. It has been seen arising from soft tissue elements following irradiation. Diagnosis is usually made on biopsy. Treatment is usually surgical excision because most types of sarcoma are resistant to irradiation.

Carcinomas of the cheek, alveolus and floor of the mouth are considered together as these regions are often involved by one carcinoma because of anatomic proximity and the treatment is often similar. *Buccal carcinoma* occurs more often in the older age group than in the younger and may be slow growing and warty in appearance, even fungating out into the mouth. This latter type is called verrucous carcinoma. Deep invasion into the cheek may be late but there may be extension down over the alveolus into the bone and into the floor of the mouth. Treatment of verrucous carcinoma can be local en bloc excision of the tumor. If bone involvement is suspected, the mandible may be included in the resection as may the floor of the mouth. When metastasis or extension into the neck is present, the en bloc removal can be combined with or considered a part of a complete neck dissection on the involved side.

Carcinoma of the jaw is an extension of carcinoma of the buccal mucosa with early involvement of the bone. When the first lesion is on the gum, the extension is to the alveolus early and the lesion is sometimes called alveolar carcinoma. Alveolar carcinoma itself usually originates from a flat type ulcer but it may arise from leukoplakia as often do buccal carcinomas.

Carcinoma extending from the cheek or floor of the mouth, and secondarily involving the alveolus, invades the bone much later. Attention of the patient may be called to the carcinoma by a tooth socket that fails

to heal. Bone involvement may be considerable when this type of carcinoma is first seen and x ray examination may not give a very good indication of the actual wide extent. The best treatment is a combined operation in which the primary alveolar tumor and involved mandible are removed and a complete neck dissection is executed in one block. This is the same type of complete radical neck dissection advised for carcinoma of the tongue.

Carcinoma of the floor of the mouth may start laterally or anteriorly to the tongue and the bone is involved often very early. Therefore, a combined operation with removal of the primary tumor and involved mandible and complete neck dissection is necessary for control. Metastasis also occurs early in the course. If the mandible is not implicated, the primary tumor may be controlled with radon seeds and complete neck dissection on the involved side may be done. In recent literature, the term "commando operation" has been used to describe the basic operation but has no informative value other than that a radical operation is necessary.

Carcinoma of the hard palate and upper alveolus is similar to alveolar carcinoma. Recognition of possible extension of carcinoma of the upper alveolus to the antrum or pyriform recess or pterygoid region is of particular importance. Early bone involvement by the tumor is usual and care necessitates its removal. The local area is usually left open for constant inspection, but food and saliva leak can be prevented by a dental plate made to fit into the defect. Later repair can be done with a local flap or if this is not available, a distant flap, as from the arm. Neck dissection, as is done for carcinoma of the tongue, will eventually be required.

A low grade carcinoma such as that not infrequently seen covering a fairly extensive area of the hard palate may be locally excised and the lining to the nose and antrum preserved.

TONGUE

The effects of *trauma* and *infections* upon the tongue and the appearance of the tongue with *vitamin deficiencies* and *congenital abnormalities* have been considered. *Benign tumors* found in the tongue are also the same as those described elsewhere in the mouth.

Hypertrophied circumvallate papillae are frequently seen and often overtreated because of failure of proper diagnosis. The

shielding the remainder of the mouth and treatment is often followed by protracted painful dryness and other effects of irradiation directly on uninvolved mucous membrane. Surgical excision of carcinoma of the tongue can be done adequately, but the magnitude of the operation itself the mutilation resulting and the repeatedly superior results obtained with radon usually obviate the necessity for operation. Involvement of the jaw by extension of a carcinoma of the tongue or failure of radiation may require extirpation. Glossectomy or hemiglossectomy has been rarely necessary during the last twenty years.

Neck dissection is necessary for the cure of carcinoma of the tongue and is as much an integral part of the treatment as is treatment of the primary tumor. Over 60 per cent of carcinomas of the tongue metastasize

to the neck usually to the same side as that in which the primary lesion is located and only occasionally to the opposite side. The time to do the neck dissection is the same as that of treatment of the primary tumor unless it is felt that there is considerable doubt that the tongue lesion can be controlled. Postponement should not be longer than a month, particularly if metastases are already present in the neck. The technique of complete neck dissection for metastatic carcinoma has been described in detail. It should be common knowledge that removal of all the lymph bearing area from the clavicle to the base of the skull, yet with preservation of vital structures, is necessary for cure. Adequate removal of the deep cervical chain is impossible without removing the internal jugular vein. Figure 21 shows the extent of removal and identifies

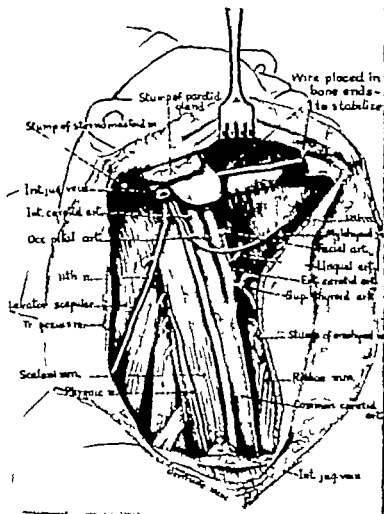


Figure 22. Combined operation for removal of carcinoma of the jaw and complete unilateral neck dissection. Complete removal of the potential metastatic nodes from the clavicle to the base of skull including the sternomastoid muscle, jugular vein and contents of the subaxillary triangle, with transection at the base of the skull and ligation of the jugular bulb. Primary tumor in the tongue, floor of the mouth or alveolus and involved bone removed in one block with the mass from the neck. The opening into the mouth is closed and jaw is stabilized with wire placed in the ends of the bone to prevent collapse. Tracheotomy is usually avoided. The structures remaining in neck are identified in Figure 21. The neck flaps are carefully closed and a firm pressure dressing is applied.

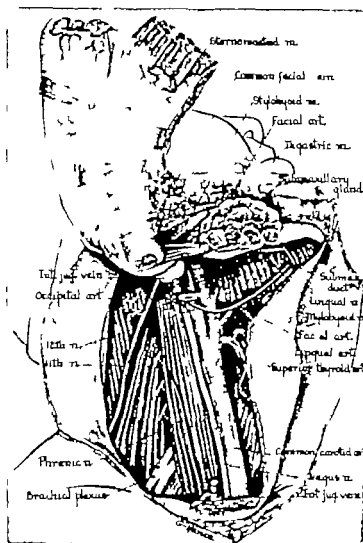


Figure 21 Complete unilateral neck dissection. Internal jugular bulb ligated and divided near the base of the skull. The submanillary duct and chorda tympani branch of the lingual nerve are divided and the freed anterior segment is rotated out and up onto the external surface of the mandible. The mass containing the sternomastoid muscles, the contents of submanillary triangle and the potential metastatic nodes from the clavicle to the base of the skull are ready for division at the jugular bulb, through the parotid gland and above the mandible. The remaining structures are indicated, any of which could be included with the block removed if necessary for cure of the cancer yet compatible with life. Firm pressure dressing is applied with mechanics waste held by gauze rolls after the flaps have been carefully approximated. The scar is minimal and the patient is usually discharged from hospital on seventh to tenth postoperative day.

on the tongue, other things being equal, the carcinoma is located, except for involvement of the floor of the mouth, the better the chances of cure. This may be due in no small measure to the inescapable fact that the nearer the tip of the tongue the lesion is located, the sooner it will be noticed. Epidermoid carcinoma primary in the base of the tongue or pharynx may first be found after metastasis has occurred in the neck. This also is of particular importance as far as prognosis is concerned with carcinoma primary in this location.

Treatment of the primary tumor is usually most successfully done with interstitial irradiation in the form of radon seeds. A

calculated dosage is given to the tumor usually by direct implantation of 1 millicurie of gold radon seeds, using one seed per cubic centimeter of tumor and tissue to be irradiated, 1.5 millicurie seeds are also used. Use of radium needles in the treatment of the primary tumors has been given up to a large extent because of the difficulty of maintaining accurate position in and around the tumor and the extreme discomfort to the patient during their period of application in this sensitive mobile structure. Use of external irradiation remains an alternative if radon is not available. In the employment of x ray therapy on the primary tumor there is apparently considerable difficulty in

but they are usually retained to give aid in the fixation of the fracture. They may be removed later.

Reduction of fractures in the body of the mandible is accomplished when the teeth have been replaced in normal occlusion for the patient. Reduction of the edentulous jaw or positioning of the teeth may be done by palpation under direct vision.

Fixation after reduction, if teeth are present in all fragments is most simply done by interdental wiring. Steel wires no. 24 or no. 26 gauge are twisted around the necks of apposing teeth and the lower jaw is wired to the upper jaw using the upper as a splint for the lower. The bicuspid or premolar teeth, if available are the most satisfactory teeth for wiring. Loops may be made of the wires and rubber band traction can be used for fixation or for gradual reduction. There are many forms of wiring and of bar appliances, but the basic procedure is to reduce the fracture so that the teeth will come up into proper occlusion and be held there. Other means of fixation must be used if usable teeth are not present, even to wiring dentures in place with circumferential wires around the jaw and denture.

Internal wire-pin fixation has been found the most satisfactory supplement in fractures near the angle of the jaw or posterior to molar teeth in fractures at the symphysis where nonunion can frequently occur without solid fixation. If there is extreme comminution of the mandible or widespread opening into the mouth, if there has been necessary delay in reduction and fixation, or in the edentulous jaw. Use of the wire pin for fixation is not difficult but does take some practice. After the fracture has been reduced, a 0.05- to 0.08-inch steel wire is driven with a power drill through both fragments across the fracture site below the nerve canal. Multiple wire pins may be needed in complicated fractures. Open mouth and some chewing are possible during the healing period. Circumferential wires have been used in conjunction with interdental and internal wire-pin fixation.

Direct wiring or using complicated, expensive external fixation or traction apparatus is usually unnecessary. Metal plates with screws have not proved consistently successful in fractures of the human jaw as they tend to become loose and infected.

External drainage is considered in all mandible fractures. It is almost impossible to break the lower jaw and not have an

opening into the mouth particularly around the teeth. Drainage can be instituted simply by making a small incision beneath the jaw and carrying the opening up to the fracture site by blunt dissection and putting a small drain to this spot. Protracted healing and osteomyelitis can be avoided by this simple procedure. Obviously with the aid and protection of antibiotics, this external drainage is omitted in many patients but this is a deviation from the rule rather than that drainage is the deviation.

Condyle fractures are suspected if there is a history of trauma to the jaw followed by pain in the side of the face or near the temporomandibular joint and change of occlusion or bite. This is especially true in subjects who have received blows to the chin or to the "button" in fights. Spontaneous fracture is rare. Condyle fractures are often associated with other fractures of the jaw and their presence must be ruled out if only one fracture is seen as in the body. The condylar neck is one of the weaker areas of the jaw and consequently is more likely to be broken even though the trauma be indirect. It is usually sufficient to wire the teeth in the patient's normal occlusion for three weeks then to allow normal return of jaw action. Only very occasionally is open operation necessary and, even if thought necessary a trial of wiring in occlusion may be done. Operation usually consists of exposure of the condyle avoiding damaging branches of the facial nerve and removal of the small upper fragment. Fixation, satisfactory for solid union in this location is practically rarely possible and is unnecessary. Fracture of both condyles is an extremely serious situation and fixation in occlusion is especially indicated to try to prevent permanent deformity.

Temporomandibular Joint Disorders. The *auriculotemporal pain syndrome* is probably the result of erosion of the temporomandibular meniscus and joint and irritation of the auriculotemporal nerve. Diagnosis may be difficult, but the patient usually complains of severe pain about the ear and temporal region and radiation down into the tongue. Opposing molar support may be lacking and can be tested by seeing if relief is obtained by opening the mouth and swinging the jaw from side to side or by building up the molar region with rubber mats or plugs. If relief is obtained by either maneuver permanent build-up of the molars can be done by the dentist or if the patient is edentulous, new teeth can be provided.

the remaining structures. Figure 22 shows the combined operation for removal of carcinoma of the jaw and complete unilateral neck dissection. Deformity following this radical operation is minimal. Bilateral complete neck dissections have to be done at times, but it is safer if the jugular veins on both sides of the neck are not removed at the same operation.

JAWS

Fractures of the Mandible. Because of the exposed position of the lower jaw fractures of this structure are frequent. The type of fracture depends on the point of contact, the force of the trauma and the relative strength of various parts of the jaw. Displacement of the fragments also is the result of the type and force of the trauma with the addition of muscular pull.

Diagnosis of a fractured mandible is usually not difficult and can be made on examination. A ray examination, in the postero-anterior right and left lateral positions, is routinely made. Special views of the condyle and temporomandibular joint may be necessary. The mandible is likely to be broken in more than one place and if one fracture is demonstrable, the presence of a second must be ruled out. The inferior alveolar nerve may be severed and if there is numbness of the lower lip, this should be recorded.

The time for reduction and fixation of a fractured jaw is as soon as possible if the general condition permits. Swelling in itself is no contraindication to immediate repair. Postponement may make reduction more difficult and eventually fragments may be permanently fixed out of position or non-union of the bone will occur.

The airway may be of primary concern in patients with a fracture of the mandible. Bilateral fracture of the body of the lower jaw and the resultant loose central segment allow the tongue to drop back and occlude the airway. Immediate temporary fixation, positioning of the patient with the head down, traction on the tongue, or even tracheotomy may be necessary for survival, particularly in an individual with a shattered lower jaw. Blood in the mouth resulting from soft tissue tears, as is often found in persons with displaced mandible fractures and secretions may actually drown a semiconscious patient unless they are sucked out or allowed to drain out by putting the head down, or a tracheotomy opening is made. Accumulation of blood, or even air in the neck, may occlude the airway by

compression and necessitate a tracheotomy. Tracheotomy may be needed at any time, but fortunately this procedure is not usually necessary.

Hemorrhage with a fractured mandible may require immediate control. Displacement of sharp fragments which may cut major vessels in the neck, necessitates adequate exposure and individual ligation. Bleeding into the neck may be more dangerous by compression of the airway as mentioned above, than is bleeding to the outside.

The general condition of the patient is of next consideration when the airway has been provided for and hemorrhage has been controlled. Complete physical examination is done to rule out the presence of intra-abdominal or chest injury and, if either is present, the necessity and the time for repair are decided. Fractures or injuries of the extremities may require treatment before after or during jaw or facial repair. A careful, complete neurologic examination is made in all patients having facial injuries or jaw fractures. If brain damage is suspected the advice of a neurosurgeon is sought. For snap blows in which the head is forcefully flexed, cervical spine x-ray films should be studied, as fractures may occur in the spine and, when present, may be a serious complication.

Anesthesia for reduction and fixation of simple fractures of the jaw may be secured by procaine block of the dental nerves or of the second and third divisions of the fifth cranial nerve as they leave the skull. General anesthesia may be preferable and decision is made as to whether general or local anesthesia would be better for each individual patient. An airway must, of course, be provided by a tube placed in the trachea, either through the nose or mouth, if general anesthesia is used.

Teeth may be involved in any fracture of the jaw or may in themselves be the extent of the injury. Chip fractures of the teeth can often be smoothed off by a dentist. Cracked teeth may be serviceable for years. If a tooth is loose many times it will become solid again and stabilization with wires or an arch should be done. Single anterior teeth actually knocked out may be replaced in their sockets if available following the accident, as they may become serviceable again. However this does not condone the practice of using another individual's teeth for re-implantation. Teeth in the fracture line of the jaw will probably eventually be lost,

other facial fractures, but usually in no way indicate the extent of the actual damage. There may be multiple small chip fractures with fragments no larger than a fingernail and these are not usually seen on the x ray film. In a badly crushed face there may be twenty to forty small fractures like a broken egg or glass. Fracture lines through dense portions of bone may be seen as may irregularities of the orbital borders and antral cloudiness. Reduction may involve pulling the face forward and the upper alveolus up or down, depending on the displacement. No preconceived routine is applicable the reduction involves reversing the action and force of the trauma.

It may be said that in these fractures as in most other trauma, basically the surgeon attempts to do the opposite of what the trauma did.

Fixation particularly in severe facial crushes requires all means of stabilization. The dental arch can be held, using the lower jaw as a splint, with interdental wires. Internal wire-pins anchored in any stable point such as a zygoma, can hold other loose points by cantilever action. Also several loose sections of the face can be stabilized in one plane by internal wire-pins driven horizontally across the face (Fig. 23). Needless to say reduction or elevation to proper position is necessary before the permanent wire fixation is done. External and overhead traction apparatus is used by some surgeons.

Zygomatic arch fractures are mentioned to distinguish them from those displacements of the zygoma itself. Arch fractures usually result from direct trauma, such as that incurred by a fall on a curb or a blow



Figure 23 Extensive crushing of face, nose and frontal sinus, as shown on roentgenogram, of a back seat passenger in a traffic accident. The patient was seen after seventeen days and operation was performed with the subject under endotracheal anesthesia. The nose and face were advanced into normal position. Cross-wires were used for the repair of the nose and two internal pins for repair of pyramidal and palate fractures. This case demonstrates the ease of course for the patient without the use of extensive apparatus. Patient was out of the hospital with both internal and cross-nasal wires in place, and only one operation was necessary.

X-ray examination of the joint may be helpful in making the diagnosis.

The *temporomandibular meniscus syndrome* results from derangement of the meniscus and may be acute following a difficult dental extraction or external trauma or may appear suddenly in chronic progressive derangement. Cracking or popping, pain and blockage with the jaw open or closed are the usual symptoms, appearing often in that order as the disease progresses. Treatment consists in rest of the joint by avoiding wide opening, minimal chewing and wearing a chin vertex bandage. Enforced rest with interdental wiring in occlusion may be necessary. Injection and joint operations have not been very successful.

Anterior dislocation of the jaw may be a part of the temporomandibular meniscus syndrome or may result from trauma on the jaw while the mouth is open or from the pull-scar neck contracture. One or both sides of the jaw may be involved. Diagnosis is usually apparent, since the mouth is held open with the lower jaw forward, or pointing toward the unaffected side in unilateral dislocation. Inability to swallow or talk is usually present at first. X-ray films may be exposed to rule out the possibility of fracture of the neck of the condyle. Treatment, if there have been recurrent dislocations, might be carried out by the patient, since oftentimes the condyle slips back easily. Downward, backward pressure in the molar area may reduce the dislocation or a general anesthesia may be necessary to accomplish this. Protection and rest should be given to the joint as described above. If reduction is impossible, open operation may be necessary and the condyle may be removed. Trauma to the chin with the mouth open may drive the condyle up through the base of the skull into the brain and this dislocation may be called an *anterosuperior one*. Treatment is usually directed toward the brain damage. Later reduction can be done and occasionally repair of the glenoid fossa is necessary.

Posterior dislocation of the jaw usually results from a forceful smash on the chin when the mouth is closed. The condyle is driven back into and crushes the bony portion of the ear canal. Reduction is often spontaneous, but recognition of what has occurred is important so that steps may be taken to prevent permanent occlusion of the ear canal. Treatment usually consists of opening the ear canal and maintaining its patency. Reduction of condyle dislocation

may require surgical exposure and possible removal of the condyle.

Fractures of the Upper Jaw. Fractures of the upper jaw are a part of middle-third facial fractures except for the occasional separate upper alveolar fracture.

The diagnosis of fractures of the upper jaw may be difficult because of swelling, however palpations and suspicion that a fracture may be present following fairly severe trauma are of the most importance in making the diagnosis. X-ray examination may not give as good an indication of the type or severity of the fractures as will simple palpation because of superposition, but it is routinely made. Posteroanterior views of the facial bones showing the orbital borders or a modified Waters film are the most valuable.

Upper alveolar fractures may involve any or all of the alveolus. Extension up into the pyriform recess or medially through the palate is common. The diagnosis is obvious on even cursory examination and palpation. Although minimal débridement in facial injuries is urged, removal of loose alveolar fragments may avoid troublesome drainage and future trouble. Reduction and fixation with interdental wires using the lower jaw as a splint are done if necessary and possible. Internal wire-pin fixation has been used as the only or supplementary means of fixation.

Transverse fractures of the upper jaw and face are frequently seen in persons involved in an automobile accident. These fractures usually result from striking the face on the dashboard, steering wheel or rear of the front seat. The diagnosis can be made on the basis of history of trauma, swelling and examination. Fracture lines may be palpated and movement of separate segments of the face or jaw can be seen. The line of fracture may start in the alveolus on one side, extend up into the pyriform recess and up into the orbit—a pyramidal fracture. If the main force is at a higher level the nose will be crushed down into the face and the face will be completely detached from the skull. This separation can be demonstrated by grasping the teeth and moving them up and down, but this is not appreciated by the unanesthetized patient. The fracture line in this fracture extends across both orbits. In severe facial crushes there can be any combination of these transverse facial fractures and they are often associated with a fracture-dislocation of one or both zygomas. X-rays are routinely taken, as described for

mask the flatness of the cheek but the displacement can usually be felt. In addition to flatness of the cheek which will be obvious when the swelling subsides diplopia due to elongation of the orbital cavity with downward displacement of the zygoma and with the eyeball following on down into the antral region difficulty opening the mouth due to impaction of the zygoma on the coronoid, and infra-orbital nerve paralysis due to crushing by the dislocated zygoma may be present. All of the above signs or symptoms should be tested for and recorded, since only one may be elicited. Disability will be permanent unless the deformity is corrected. X rays, made with the patient in the posteroanterior position or the modified Waters position showing the orbital borders of the facial bones are of the most practical value and are taken routinely. Here again any x ray film many times does not give a very good indication of the extent of the damage.

The time the repair should be made is the same as that for fractures of other facial bones i.e., as soon as the patient's general condition permits.

Minimal debridement is done in facial fractures. Completely detached bits of bone may be removed, but if there is a chance of survival they are left if necessary for removal secondarily. Three weeks is about the limit of time at which the zygoma can be reduced from its displaced position. Earlier than this is preferable.

Reduction should be done by the simplest possible method and will suffice if impaction occurs. A large jaw hook pushed through the soft tissue can elevate the zygoma by grasping the inferior border and probably provides the simplest direct approach to the zygoma. There are numerous other operations for reducing the zygoma, but if impaction in the normal position does not occur a method of fixation is needed. Reduction and fixation are usually possible through entrance into the antrum through the fracture line in the buccal fornx. This requires only a short incision through the buccal mucosa. From inside the antrum the displaced zygoma can be elevated with a palate elevator or Kelly clamp the surgeon using his free hand for guidance as to proper position. The correct position can be maintained, until solid union occurs, with an iodoform pack incorporated with balsam of Peru lightly accordioned into the antrum. The pack acts as an internal splint and makes it possible to "mulch" into proper

position the multiple comminuted fragments in the floor of the orbit and antral walls. This pack should be removed in ten to twenty days. Multiple internal wire pins driven across the face using the opposite zygoma as a point for cantilever support have given sufficient fixation alone or may be used in conjunction with other methods. When both zygomas are loose wire pins have given enough fixation for solid union to occur and in severe facial crushes have even provided an initial point on which to base fixation of other shattered features.

Nasal fractures are probably more often overlooked than they are diagnosed. Bleeding from the nose following trauma to the part is the result of fracture or laceration of the mucosa by one of the bones or cartilages and careful examination may be required to ascertain the cause. Airway obstruction by the bent septum should be corrected and other depressed supports to the nose should be elevated. Maintenance of the reduced position can be provided by intranasal packing and an aluminum splint applied over the nose. In crushes of the nose with extreme comminution the fractured segments are raised up out of the face and often require further support. No. 26 steel wire passed beneath the nose and anchored over lead plates on the outside of the nose may act as a cradle with the nose resting on the wires.

Fractures of the nose are further illustrated in Figures 28 A, B and C. Injury to the soft parts as in Figure 28 A, illustrates the necessity for their accurate reposition as well as that of the underlying supporting framework. The x ray shows marked displacement of the fragments.

If bone fragments comminuted as they usually are in nasal fractures, remain displaced and depressed, secondary build-up is necessary as shown in Figure 28 B. If the dorsum is distorted and enlarged as in Figure 28 C whether it was due to a fracture or a racial characteristic, cut-down and straightening can be done.

Jaw Infections. *Osteomyelitis* (Fig. 27) is most commonly caused by infections dependent on the teeth or by fractures of the jaw but it may be associated with cysts in the jaw or follow injuries to the mucosa or infrequently result from infections elsewhere in the body such as in the paranasal sinuses. Other causes may be heat and irradiation.

The most common infecting organisms are staphylococci or streptococci, but others infrequently seen are the *Mycobacterium tu*



Figure 24. Method of reducing dislocation of zygoma. This is the simplest direct method applicable when fragments will impact in the reduced position. The position of the heavy hook beneath the flange of the zygoma after piercing the skin is shown. Reversing the direction through which the dislocated zygoma has passed by pulling on the hook, the zygoma is guided into impacted reduction with the fingers of the other hand on the upper border of the bone. This is the method of choice for the usual zygomatic arch fracture, avoiding an incision above the hairline. However, extreme care should be taken to keep the upward pull of the hook controlled at all times. Failure of the fragments to impact by this method or any other type of reduction necessitates changing to the approach through the fracture line above the canine fossa, which allows for fixation by the use of an internal splint in the form of a pack in the antrum.



Figure 25. Drawing showing the result of fracture-dislocation of the left zygoma and the method of direct binasal reduction. Heavy zygoma crushing the thin-walled orbital floor and antral wall causes expected comminution, while the main body of the zygoma itself is rarely broken. The possibility of injury to the optic nerve by these sharp fragments can be appreciated as well as damage to the orbit by the upright process of the zygoma. Position of lengthened orbital cavity leaving the globe to drop, and the resultant diplopia are shown. The end of a Kelly clamp engaging the angle of the zygoma to elevate it into position after entrance through the fracture line in the antral wall into the antrum itself is demonstrated. At this point the fingers of the other hand are placed on the inferior orbital floor and the zygoma is placed in reduction. The comminuted floor and wall are "munched" into position by a clamp placed on the inside and a hand placed on the outside, then held, until union has occurred, by an iodoform pack impregnated with balsam of Peru accorioned to the antrum.

with the fist. The deformity can be palpated through the swelling and x ray pictures can be taken to compare the arches. Treatment consists in direct elevation of the depression in the arch with the hook through the skin (Fig. 24) or a large towel clip can be used. There is no scar. Impaction usually occurs and nothing further is needed. A more indirect approach may be made through an incision above the hairline and the depression may be elevated by sliding it down beneath the temporal fascia. Impaction is not dependent on the method of elevation. If impaction does not occur open operation and wire fixation may be necessary.

Fracture-dislocation of the zygoma (Fig. 25) usually follows trauma to the cheek bone because of the prominent position of the zygoma and is often associated with fracture of other bones in the face or jaw. The zygoma itself is usually not fractured but is separated from all of its attachments and crushes the thinner surrounding bones, including the maxilla, with crumpling of the antrum and depression of the orbital border and floor. The effect of dislocating the

zygoma might be compared to that of placing a marble on an egg and striking the marble with a hammer the marble representing the zygoma and the egg the surrounding bones. As could be imagined, the marble would be broken if a strong enough blow were used, but crushing of the egg is inevitable.

Diagnosis of a fracture-dislocation of the zygoma is usually easy to establish by palpation, particularly of the inferior orbital border. Swelling may be marked and may

berculosis Actinomyces and Treponema pallidum.

Periapical abscess is really localized osteomyelitis and usually spreads by the extraction of an acutely abscessed tooth. *Pericoronitis* is soft tissue inflammation around a partially erupted third molar which may progress to osteomyelitis spontaneously or more commonly following extraction performed when the tooth was acutely abscessed.



B



C

Figure 29. A, Severe fracture of nose with soft tissue laceration and confusion as shown in x ray. Accurate replacement of bony fragments and soft tissue parts avoids secondary operative procedures. B Result of permanently displaced old fracture of the nose. Secondary dorsal build-up necessary when immediate elevation not done. C, Cut-down of nose results in proper feature balance.

Fractures are the second most common cause of osteomyelitis. In all fractures of the jaw drainage to the outside should be considered since it is almost impossible for the jaw to break and not lacerate the mucosa if teeth are present. An avenue to the bone for mouth organisms is opened up. Prevention by prophylactic drainage is the best treatment although reliance is often placed on antibiotic protection.

Cysts are a potential pocket for infection and the avenue for mouth organisms may be via a nonvital tooth root.

Metals such as arsenic and mercurial compounds rarely cause necrosis of the jaw.

Thermal necrosis of the jaw may follow cautery destruction of an overlying eardrum and is usually relatively localized.

Irradiation is not an infrequent cause of osteomyelitis and is distinguished by the intense pain. It usually follows irradiation to carcinoma of the mouth near the bone. Involvement may be widespread with the healing slow and promise of secondary breakdown great.

In the acute stage of osteomyelitis pain is the prominent symptom and it may be very severe. There may be a history of a recent extraction of an acutely inflamed tooth. Swelling may be diffuse about the face and extend down into the neck. Tenderness is marked and may be over the jaw or generalized over the swollen area. Trismus may be marked. There are also the general signs and symptoms of infection, such as fever and general malaise. X ray films may show very little in this acute phase other than the cavity left by extraction of the tooth with slight bony erosion. In the chronic stage, there is local reaction to a foreign body and purulent drainage with acute exacerbations accompanied by general reaction. Multiple sinus tracts will be present by now possibly with an oral fistula. X ray examination in the chronic stage will show cavities, sequestra and, later the developing involucrum.

Prevention is the best possible treatment. Though this may be a trite saying, and should be self-evident, prevention is worth while if one patient can be saved from osteomyelitis. Dentists have learned that an acutely inflamed tooth should not be extracted since instead of allowing for drainage, new avenues for spread are opened up by this procedure. External drainage for fractures of the jaw will reduce the incidence of osteomyelitis.

Antibiotics in large doses are used and



areas of necrosis. Identification of tubercle bacilli is necessary for the final diagnosis. Treatment may be more radical than for other forms of osteomyelitis. Sequestrectomy, excision of involved soft tissue and closure of the wound are done while an adequate streptomycin blood level is being maintained.

Infection of the jaw with *Actinomyces* should be strongly suspected from the history. Typically a chronically infected lower molar is extracted and three weeks later a painless mass appears over the jaw which increases in size and becomes painful and inflamed, trismus appears and there is the usual general response to infection. Drainage may be spontaneous and subsidence occurs, but the painful swelling appears recurrently. The typical late picture is that of widespread induration in the face and neck, multiple draining sinuses and scars and the inability to open the mouth. X-ray examination shows osteomyelitis. The diagnosis is established by finding sulfur granules in the drainage but it may be necessary to open a new pocket to demonstrate them. Microscopic confirmation is advisable. Treatment consists in drainage of the multiple abscesses, x-ray therapy and prolonged systemic usage of antibiotics. Excision of the involved soft tissue may be necessary.

Syphilis involving the jaw bones is rarely seen. The usual involvement is of the hard palate and the supports to the nose. Treatment consists of the usual systemic therapy for syphilis, but later reconstruction and closing of the defects may be necessary.

Ankylosis may be true or false. True ankylosis results from bony union at or near the joint and has been seen following mastoiditis, osteomyelitis elsewhere in the jaw and fracture. Patients have been seen who were unable to open their mouths for years. One or both sides may be involved. When it occurs on one side, the body of the jaw appears more normal on the ankylosed side than it does on the uninvolved side. If any opening is possible, the jaw deviates to the ankylosed side, and the preangular notch is deepened on that side. X-rays may show the extent of damage and union at the joint. Also, involvement of the coronoid should be determined since it has been seen solidly fixed by bony union. A series of coronoid ankylosis alone, resulting from war wounds has been reported. Treatment of true ankylosis consists in resection of enough bone for a false joint to develop. Bilateral true ankylosis is a particular problem, since for

ward support of the jaw may be lost and tracheotomy may be necessary. In such instances the worse side should be operated on first and possibly enough opening may result following, unilateral relief. *False ankylosis* is due to scarred bands which prevent the jaw from opening, but it may be difficult to distinguish from bony ankylosis both may be present to some degree. The scar requires excision and some substitution with a graft or flap is required for relief of false ankylosis. The scar of a neck contracture may produce a type of false ankylosis by pulling on the chin and inducing an anterior dislocation of the jaw as in Figure 28.

Tumors of the Jaw Tumors benign and malignant which occur in other bones of the body also are found in the jaws. Their treatment is essentially the same as in other locations and further discussion is unnecessary. In addition there are tumors in the jaw which are peculiar to it owing chiefly to the presence of teeth.

Torus palatinus is an osteoma or exostosis which is more or less symmetrical and is found in the midline of the hard palate. Because of its size, which may affect the speech or prevent the proper fitting of dentures or irritation, it may require removal. Excision is not difficult but opening into the floor of the nose should be avoided. *Torus mandibularis* is a similar occurrence of an exostosis inside the lower jaw which may be in the midline or symmetrical on both sides. Removal is usually not required, but it may be necessary to allow for a lower dental plate.

Dental cysts root cysts or radicular cysts are probably of infectious origin and are seen on x-ray films as a cyst in the bone surrounding a tooth root or a retained root. Cure requires removal of the root and the cyst. The bony defect is allowed to heal from the bottom.

Dentigerous cysts are epithelia lined and contain a tooth bud or unerupted tooth. Bone is eroded as they distend but is not invaded by the cell. They must be completely removed.

Adamantinomas or ameloblastomas are tumors which arise from the preameloblasts, or cells which later form the enamel of a tooth. They may be cystic or solid and usually occur as a mixture of solid and cystic elements. X-ray films may show a multilocular arrangement with erosion of the bone by pressure. The more solid the tumor is, the more apt it is to invade bone. This may proceed so far as to make the tumor malign



Figure 27 Osteomyelitis. The spread of the infection through all the closing muscles and the location of the drainage incisions are shown. The incision over the angle might have been made lower except that the cheek was just about to perforate here. It is practically always best to drain on both the buccal and lingual surfaces of the jaw. The second picture was taken two weeks after the first.

have prevented the local progress of the infection to extensive bone necrosis. Their use has also undoubtedly saved lives.

Upper alveolar or maxillary osteomyelitis is much less common than is osteomyelitis in the mandible and occurs most frequently in the region of the incisors, canines or at the tubercles. Infection may spread from the nose or paranasal sinuses in addition to that associated with the teeth. Often the most prominent symptom is periorbital edema. Treatment follows the same principles outlined for osteomyelitis of the mandible.

Gumboil, or alveolar abscess, should be drained by incising the mucosa and gently retracting the periosteum. The tooth can be extracted when the process has subsided.

Once an abscess has developed, it is usually opened externally otherwise the infection may spread and dissect all the closing muscles of the mouth, including the masseter internal pterygoid and the temporal. A short collar incision is made through the skin, followed by blunt dissection into the abscess and to the bone, care being taken to make the opening into the masseter space on the outside and into the internal pterygoid space on the inside.

The abscess is evacuated and a light iodoform pack is inserted to be left in place for twenty-four hours to establish the drainage tract.

No cutting of bone is done at this stage.

The jaw sequesters rapidly and to cut into it invites further spread of the osteomyelitis. Spontaneous separation of any fragments is awaited for up to twelve weeks and, during this time, secondary drainage may be necessary. Also, during this time the process changes from an acute to a chronic one and becomes less dangerous.

Maintenance of the arch of the jaw is the most important consideration when osteomyelitis has developed. Conservative management is indicated. Though antibiotics may hasten the development of an involucrum and save lives, the dental arch is a mechanical problem and the principles of handling potential sequestra are the same as those outlined twenty years ago. The involucrum must have formed before sequestra are removed and then they can be gently lifted out. If the jaw is allowed to collapse by radical débridement, it is almost impossible to reconstruct the arch.

Block necrosis of the mandible has been followed by regeneration of an essentially normal arch by conservative management of the sequestra.

Tooth buds in children should be conservatively drained.

Tuberculosis of the jaw is rare and is usually a more indolent process than is osteomyelitis. A large fluctuant area may be present over the jaw with multiple draining sinuses. X-ray examination may show spotty

their own splints. As these grafts may bridge defects and do not simply act as splints while union occurs they are thought to be true grafts and to persist as such. Obviously bone-bank nonviable material is not considered as applicable for this permanent bridging of defects as are fresh autografts.

The soft tissue pocket into which the graft is to be placed is most important for persistence of the bone graft and union to occur because success of any deep repair is dependent on primary soft tissue healing over it.

SALIVARY GLANDS AND DUCTS

Trauma. Contusion of a salivary gland, duct or surrounding soft tissue may cause obstruction which is usually of a temporary nature and subsides spontaneously.

Laceration of a salivary gland may be associated with facial lacerations and will be evident on examination. Salivary drainage will be seen and is always looked for in the presence of laceration near the salivary glands. Tears of the parotid or submaxillary salivary glands may result from sharp fragments in jaw fractures. If the laceration is only in the substance of a salivary gland, no special consideration is necessary as healing will take place spontaneously.

Laceration of a duct can result from the same causes and recognition is particularly important as drainage will continue or blockage develop unless it is repaired. All deep lacerations in the region of the salivary ducts should be carefully investigated, as should fractures of the mandible near the submaxillary duct, and immediately repaired. Late scarring may produce obstruction secondarily. Transplantation of the proximal duct opening further back in the mouth may be done instead of anastomosis.

Salivary duct fistula to the outside persists

after injury is very annoying to the patient and requires operative closure if drainage can be established through it. Changing of the fistula to the inside of the mouth may be necessary if the continuity of the duct cannot be re-established. A ray treatment to shut off the salivary flow may result in the closure of some small fistulas but cannot be relied on for large ones.

Obstruction to the Salivary System. Obstruction to the flow of saliva from any salivary gland may be due to compression or involvement by scar infection or tumor or other mass near the duct, infection in the gland or duct, salivary calculus, tumor of the duct or gland and scar or deformity of the duct. Obstruction without any demonstrable cause is not infrequent.

That obstruction is present is usually obvious as the affected salivary gland becomes enlarged and prominent. The swelling is usually worse on salivary stimulation and may subside to some extent between meals. Swelling may appear suddenly or may develop gradually depending on the cause. Pain is usually present and in degree related to the amount of swelling and its speed of development. Examination will reveal a swollen tender gland and lack of salivary flow from the orifice of the duct in the mouth, even on tender pressure over the gland or milking of the duct. Purulent material may be draining from the duct. Fever and malaise may be evident, depending on the individual reaction to the particular obstruction. Careful examination of the duct with a lacrimal duct probe may determine the cause of obstruction. X-rays should be taken but often they are of no or little aid in the diagnosis.

Extrinsic causes of obstruction, such as a constricting scar infection or tumor require relief of the primary condition. Care should

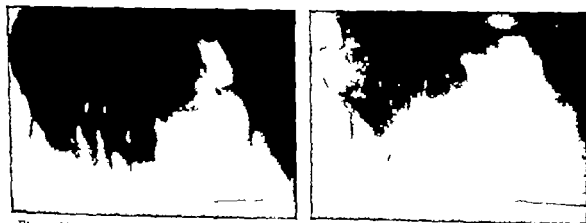


Figure 20 Adamantinoma. X-ray appearance indicates the cystic character and expansion of bone.



Figure 28 Contracture neck from old burn scar dislocating lower jaw. A, Repeated attempts by the body to heal the open flexion area have developed scar strong enough to actually dislocate the mandible. There is continual drainage of saliva onto the chest. B The appearance of the patient following one operation. The deep scar has been excised, opening up the flexion area and relocking the jaw. defect has been covered with a free split-thickness graft. Watertight integrity of mouth has been restored.

nant by position or extension toward the base of the brain. Adamantinomas have been reported in the long bones. The benign character may change any time and invasion of bone and soft tissue occurs. The change is usually to squamous cell carcinoma, though the original solid adamantinoma looks like a basal cell growth microscopically. Treatment as long as the tumor is contained in the bone, may be intraoral as for dentigerous cysts. Invasion of the bone requires block excision, which has also been done intraorally and facial scars avoided. Extensive soft tissue involvement requires block removal through a neck approach.

Odontomas arise from odontoblasts. Small tumors of this type may be removed in the same way as are unerupted teeth, but larger ones may require removal of a segment of jaw.

Osteofibromas and **giant cell tumors** are probably the most common benign tumors found in the jaw which do not arise from dental elements, but they are seen only infrequently. In rare instances, giant cell tumors may occur as part of the picture of a parathyroid tumor.

Osteogenic sarcoma is infrequently seen

but is the most common malignant primary jaw tumor. *Metastatic carcinoma, erosion or invasion by alveolar carcinoma* or that arising in *salivary glands* or by *secondary invasion from metastatic lymph nodes* is of more common occurrence.

Chondrosarcoma usually occurs in the lower jaw about the joint and is perhaps one of the sarcomas most often controlled by operation.

Carcinoma of the jaw does not occur spontaneously but by secondary invasion from buccal or alveolar carcinoma. There is, however, an exception to the above statement, in that adamantinomas may become malignant and form carcinomas in the jaw itself, the originating epithelial cell coming from the paradental debris left in the jaw following formation of the enamel organ.

Bone Grafts to the Jaw Bone grafts are necessary to bridge defects in the jaw following loss from any cause. They are often used to promote union where there previously has been failure and to fill out contours. Ribs are most useful for graft material because of their natural shape and iliac crests may be well shaped to fit defects. Solid grafts may be wired in place to become

vary glands are of the same types as those which are found in soft tissue elsewhere in the body e.g., lymphangioma, angioma and lipoma. Mixed tumors of anlage origin (Fig. 31) are usually thought of as being inherent in salivary glands, but they have been observed elsewhere in the body. Papillary cystadenoma lymphomatosum is found in these salivary glands. One hundred and eighty-seven tumors were removed from the parotid gland over a twelve-year period without evidence of recurrence or paralysis of the facial nerve. One hundred were benign mixed tumors. Papillary cystadenoma lymphomatosum comprised 3.4 per cent, lipoma, 0.6 per cent, angioma, 1.4 per cent and 5.3 per cent proved to be lymphangioma.

Benign mixed tumors occurring in the salivary glands are thought to be of anlage origin as postulated by Halpert, from nests of embryonal ectoderm which do not imitate salivary gland or distort the gland with growth. Similar tumors occur elsewhere in the body and the relationship between tumor and gland is incidental. Occurrence may be multiple. Eighty nine per cent are found in the parotid, 10 per cent in the submaxillary and 1 per cent in the sublingual gland. They may be found in persons of any age, most commonly in those between thirty and fifty years. The characteristic microscopic appearance is that of epithelial cells in cords, lumps or stellate arrangement set in a stroma of collagen which may look like car-

tilage and which may be part of the tumor cells themselves.

The diagnosis is made on the history of a painless mass which appears in a salivary gland and slowly increases in size but unrelated to salivary stimulation. A solid mass can be felt in the gland not attached to the skin. Facial nerve function remains unimpaired regardless of the ultimate size. Stalography usually does not help in making the diagnosis. Removal is recommended as soon as the diagnosis is made. In the parotid gland, the closeness of the facial nerve and its frequent association with the tumor make the problem of complete tumor removal without damaging the nerve worthy of special consideration. Wide exposure to the entire surface of the gland is necessary to remove the tumor safely (Figs. 32 and 33). A direct approach is made to the tumor and the mass is removed completely with careful minute pressure on all surrounding gland elements which might contain nerve filaments, before they are cut. Fortunately the majority of tumors lie over the facial nerve and they can best be removed in the manner just described. If the tumor is beneath the nerve, this will be evident. The major divisions of the facial nerve can be identified and protected from injury by dissecting the tumor from these and smaller fibers. An individual observer of the face is part of the operating team and he reports any movement or twitching of the face as the operation progresses. The skin is carefully closed



Figure 31 Bilateral benign mixed tumors of anlage origin; removed as indicated in Figures 32 and 33 with preservation of the facial nerves.



Figure 30. X-ray appearance of salivary calculi in submandibular duct.

be exercised in removal of tumors near the salivary ducts.

Infection in a salivary gland or duct is one of the most important causes of obstruction. It may be associated with a calculus and this should be established at the onset. Obstruction may result from the edema of infection or persist after removal of a stone. Although a single organism may initiate the infection, the usual mouth organisms can be cultured when symptoms begin. Swelling of both submandibular and parotid glands may be noticed in severely debilitated subjects.

Treatment consists in the administration of systemic antibiotics and salivary stimulants, mouth cleanliness including care of gum disease and carious teeth, and administration of sedatives or analgesics necessary for general comfort. Carefully probing the duct with a small lacrimal duct probe might reveal the presence of a stone. It is a part of the treatment, allowing draining and at least temporary relief from the swelling. Subsidence is usually fairly rapid unless there is some other fundamental reason for persistence, but recurrences are common and enlargement of the duct orifice may be necessary. Persistent infection in the gland may require its removal. Glandular elements by this time are pretty well destroyed. Use of irradiation to stop salivary secretion is considered in those subjects in whom no cause for the obstruction is demonstrable.

Calculus in the duct or in the gland is the second most common cause of obstruction. The signs and symptoms are those of obstruction and infection is usually present. Careful probing of the duct with a small lacrimal probe will establish the fact that obstruction is present or a grating of the duct on the stone may be heard or felt. X-ray examination may help to determine the position of the stone (Fig. 30) but

superposition of the jaw may prevent visualization. More than one stone is often present. Treatment consists in removal of the stone and relief is usually immediate. Enlargement of the duct orifice may prevent stone formation or permit passage of stones already formed. It may be impossible to remove stones in the gland or those which have migrated into the surrounding area and it may be necessary to remove the affected gland and duct. Antibiotics and salivary stimulants are used.

Tumors arising from the salivary ducts are rare and obstruction is slower than that by stone or infection. Tumors in the gland itself are not usually present.

Scar or deformity of the duct from other causes resulting in obstruction is not uncommon. Scars following passage of a stone or resulting from excessive trauma in their removal are often not of sufficient severity to require operative correction. Repaired lacerations of ducts may be responsible for obstruction after healing is complete. Local revision of the scar may relieve the obstruction or a new opening may be made in the mouth proximal to the blocked area. Deformity of the duct may be due to atresia anywhere in the duct, but the commonest site seems to be at the orifice. The opening may be of sufficient size but, in persons of any age, slight inflammation in the duct or around the opening into the mouth may cause obstruction to the salivary flow. Repeated careful dilation with a lacrimal probe may suffice, but the orifice may require enlarging.

Obstruction to any salivary gland may occur without any demonstrable cause being found. This may be recurrent. Three light doses of deep irradiation may be sufficient to shut off the gland, but, if not, actual removal may be necessary.

Benign Tumors of the Salivary Glands. Benign tumors occurring in the major salivary

salivary gland tumors will be the most accurate because of the high incidence of a mixture of various types of carcinoma in the same tumor. Tissue resembling adenocarcinoma and squamous cell carcinoma has been seen on the same slide. In addition to these two types of carcinoma, a third, called malignant mixed tumor is not infrequently seen. Evidence of benign mixed tumor may or may not be found in association with a malignant tumor. Carcinoma may originate as a benign mixed tumor.

Malignant tumors of the parotid gland can be accurately diagnosed by the history of a hard solid mass in the gland which has rapidly increased in size or has paralyzed part or all of the facial nerve, or if there is fixation of the mass to its surroundings or evidence of extension or metastasis. X-ray examination of the chest should be made. Biopsy is only occasionally necessary because of the high degree of accuracy of clinical diagnosis and of the plan of procedure in which the original operation is considered as a biopsy if unsuspected malignant cells are discovered. Frozen sections of those tumors that are difficult to diagnose are not dependable in many instances. Microscopic interpretation is evidently so difficult that if biopsy is necessary removal of adequate material is worth while. Tissue

obtained by punch biopsy may show a benign tumor but a few millimeters distant, there may be carcinoma which would be missed. Biopsy if necessary has the same surgical requirements as does removal of a benign tumor.

Treatment of malignant parotid tumors may be surgical excision, irradiation, or a combination of both. Interstitial irradiation with radon seeds has been used very successfully to control the malignant tumor locally and the facial nerve is saved if it is not already involved by the carcinoma. Cure of metastatic lesions when present, requires surgical excision as do areas of potential spread. A radical operation for cure of carcinoma of the parotid includes removal of the entire gland, including the nerve, as indicated, and occasionally the overlying skin and mandible, combined with a complete neck dissection. En bloc excision of the primary tumor and area of lymph spread is possible; excision of the jaw and skin is carried out as necessary.

Malignant tumors of the submaxillary gland require removal of the entire gland and a neck dissection. The tissues can be removed in one block and as much of the mandible and skin is removed as is necessary. X-ray examination of the chest should be made.



Figure 33. Benign tumor in the parotid gland elevated from its bed, showing the usual position of the facial nerve beneath the tumor. Injury to the nerve during tumor removal is guarded against by observation of the facial movement as is done when the tumor is located beneath the nerve. The wound is carefully closed with fine sutures and a pressure dressing is applied to seal the flaps down and insure a minimal scar.



Figure 32. Removal of benign tumors in the parotid region. The incision necessary for thin forward flap elevation for safe exposure of the parotid area and complete removal of the tumor is made in natural creases as far as possible leaving a scarcely noticeable scar. The lower edge may be elevated if necessary or the incision may be continued into the neck for neck dissection when a malignant parotid tumor is present. An endotracheal tube is used to provide an airway when general anesthesia is employed. The entire face is exposed during the operation.

and a comfortable firm pressure dressing is applied. Healing takes place in five to seven days. The resultant scar is minimal owing to the placement of the original incision.

Papillary cystadenoma lymphomatosum is removed in the same manner as are benign mixed tumors. It recurs less frequently than do the benign mixed tumors.

Lipomas are treated in a similar way as positive diagnosis often cannot be made until the tumor has been exposed.

Growing, solid *hemangiomas* which are found in infants usually do not require a biopsy for diagnosis and are very responsive to irradiation given in small doses. Surgical removal of this type of angioma is unnecessary and invites damage to the facial nerve. Cavemous angiomas, or a mixture of large and small vessel angiomas, are evident on examination and are treated by a combination of irradiation, packing and excision.

Lymphangioma is usually seen in infants but may be first noticed in individuals of any age. It occurs as a soft, compressible, painless tumor which is indefinite in outline. Change in character to an acutely swollen tender mass may be due to blood

leak into one of the spaces. The tumor may extend into the mouth or into the neck where it is called hygroma. The treatment is the same as that described for lymphangioma occurring in the mouth and it is sufficient to say that complete removal is often not possible or necessary. Making an opening into the area through the usual wide incision, partial excision of the mass and packing with gauze impregnated with a mild escharotic agent usually will result in destruction of the thin walled flat endothelia lined spaces and healing of the space may occur.

Other salivary glands may contain benign tumors. The easiest method for removal of these is usually total excision of the gland and the contained tumor.

Malignant Tumors of the Salivary Glands. Malignant tumors may occur in any salivary gland. The parotid is most frequently the seat of tumors, but the submaxillary tumors are most often malignant. Between 20 and 30 per cent of parotid tumors are malignant. Incidence of malignancy in the submaxillary gland is reported as high as 80 to 90 per cent.

The simplest microscopic classification of

THE NECK

Developmental Anomalies, Tumors, Infections and Wounds of the Neck

By WALTER W. CARROLL, M.D.

WALTER WILLIAM CARROLL, Associate Professor of Surgery at Northwestern University, received his education and surgical training at the University's School of Medicine. He is representative of the young surgeon in practice who devotes a large share of his time to teaching and research upon a wholly voluntary basis. His interest in surgery of the neck is manifested in his contributions to the literature.

DEVELOPMENTAL ANOMALIES OF THE NECK

Early in the course of intrauterine life the embryo differentiates the anlage of adult neck structures. The jaws and neck are intimately related to the transient branchial arches which appear in the third and fourth weeks. The thyroglossal duct also appears at this time. During the course of the subsequent two to three weeks, these structures lose their identity as they form the more permanent organs of future life. Within the depths of these tissues the paired jugular lymphatic anlage appears during the six to eight week period and primary lymph glands begin to differentiate shortly thereafter during the third month. Characteristic lateral and midline anomalies may result

from slight alterations in these normal embryologic transitions. Defects in fusion or failure of normal obliterative processes results in formation of cysts, sinuses or even fistulae connecting with the pharynx. In the newborn, these may be of such size as seriously to interfere with vital functions. If respiratory or cardiovascular complications are minimal, they may remain only as cosmetic deformities or become the source of repeated episodes of infection.

LATERAL ANOMALIES

The majority of cysts, sinuses and fistulae located in the lateral portion of the neck are the result of faulty development. These lesions are found at any age, but for the most part in the young. While in patients

READING REFERENCES

- Brown, J B The Utilization of the Temporal Muscle and Fascia in Facial Paralysis. *Ann. Surg.* 109 1016 1938
- Brown, J B Double Elongations of Partially Cleft Palates and Elongations of Palates with Complete Clefts. *Surg. Gynec. & Obst.* 70 815, 1940.
- Brown, J B., and Cannon, B. Composite Free Grafts of Skin and Cartilage from the Ear. *Surg. Gynec. & Obst.* 82 253, 1946.
- Brown, J B., and Fryer M P Inflammatory Lesions of the Jaw. In *The Cyclopedia of Medicine, Surgery Specialties*. Philadelphia, F A. Davis Company 1950 vol. 7 pp. 553-562.
- Brown, J B., and Fryer M P Tumors in the Parotid Region. *Am. Surg.* 18 680, 1952.
- Brown, J B., and Fryer M P Plastic Surgery for Severe Facial Paralysis in Elderly Patients. *J. Am. Geriatrics Soc.* 2 830 1954.
- Brown, J B Fryer M. P., and McDowell, F Internal Wire-Pin Stabilization for Middle Third Facial Fractures. *Surg. Gynec. & Obst.* 93 676, 1951
- Brown, J B and McDowell, F Field-fire and Invasive Basal Cell Carcinoma-Baso-squamous Type. *Surg. Gynec. & Obst.* 74 1128, 1942.
- Brown, J B and McDowell, F Internal Wire Fixation for Fractures of the Jaw. *Surg. Gynec. & Obst.* 74 237 1942.
- Brown, J B and McDowell, F Neck Dissections for Metastatic Carcinoma. *Surg. Gynec. & Obst.* 79 115, 1944.
- Brown, J B., and McDowell, F., Simplified Design for Repair of Single Cleft Lips. *Surg. Gynec. & Obst.* 80 12, 1945.
- Brown, J B., McDowell, F., and Byars L. T., Double Clefts of the Lip. *Surg. Gynec. & Obst.* 85 20, 1947
- Brown, J B., McDowell, F., and Fryer M P Surgical Treatment of Irradiation Burns. *Surg. Gynec. & Obst.* 88 609, 1949
- Brown, J B., McDowell, F., and Fryer M P Direct Operative Removal of Benign Mixed Tumors of Anlage Origin in the Parotid Region. *Surg. Gynec. & Obst.* 90 257 1950
- Brown, J B. and others. Ankylosis of the Coronoid Process of the Mandible. *Plast. & Reconstruct. Surg.* 1 277 1946
- Fryer M P A Simple Direct Method of Reducing a Fracture-Dislocation of the Zygoma. *S. Clin. North America* 30 1861, 1950

in some patients drains to the outside in a spontaneous manner. If this occurs or if the cysts are drained surgically a permanent sinus tract develops. The cysts are then converted into a sinus with cystlike pocketing above the opening.

These anomalous sinuses and fistulae may present themselves in three variations with respect to surface openings. The tract may be complete—that is there may be a well-defined external cutaneous opening as well as an internal pharyngeal opening. In such instances the external opening will be found anywhere along the anterior border of the sternocleidomastoid muscle, but most commonly along the lower third. Coursing upward, it is somewhat superficial but penetrates the platysma muscle and enveloping layer of the deep cervical fascia. It passes over the common carotid artery and, remaining anterior to the hypoglossal nerve, it turns medially to pass through the upper portion of the carotid bifurcation area to enter the pharynx at Rosenmüller's pouch (Fig. 1).

The sinus tract may be incomplete in one of two ways. The more common of this type are those in which there is a well-defined external orifice leading into a sinus tract which extends only a short distance into the neck but follows the general path upward as described for the complete fistula. The less common variety is that in which there will be found only an internal opening into the pharynx which connects with a short sinus or flat cyst in the parapharyngeal region near the tonsil. In reality these latter two types are only sinuses and by definition should not be referred to as fistulae.

Correlation of Embryology and Pathology
It is significant to note that these complete fistulae extend from the tonsillar fossa downward under the angle of the mandible passing under the midportion of the posterior belly of the digastric muscle and anterior to the glossopharyngeal and hypoglossal nerves through the upper area of the carotid bifurcation, eventually making their way to the cutaneous surface at some lower point. In an effort to substantiate the theory that these lesions arise from the branchial apparatus rather than from the thymic anlage Gross points out that the fourth and fifth clefts can be ruled out because the cutaneous openings are always found above the residues of the fourth and fifth clefts. To rule out the third cleft, the site of origin of the thymic duct, it is mentioned that the tract to be derived from this must course

posteroinferiorly to the glossopharyngeal nerve which runs in the third arch. This appears to be most rare. Further if a lateral cervical fistula were to develop from the second branchial cleft it would have to lie between the internal and external carotid arteries and extend into the pharynx only in the area of the base of the tonsil or along the posterior tonsillar pillar. Since this is the common experience it is concluded that the great majority of congenital fistulae of the neck originate from the second branchial pouch and cleft. The existence of this complete fistula presupposes the breaking through of the embryonic membrane separating the pouch from the cleft. This may come about through an embryonic perforation or postnatal infectious process.

Diagnosis. Branchial cysts most commonly are found at the hyoid level. They tend to produce a superficial bulge which may displace the sternocleidomastoid muscle. They often can be moved slightly by the examiner and, on aspiration, they yield a typical fluid which contains cholesterol crystals. The latter finding may be helpful since some of these contain fluid that bears a striking resemblance to tuberculous exudate. Injection of a radiopaque iodine solution into the cyst sometimes will confirm this differentiation. Branchial cysts are quite smooth-walled and thus will produce a sharp outline as compared with the ragged edges of tuberculous abscesses.

Branchial sinuses and fistulae can be recognized by their characteristic external opening along the anterior border of the

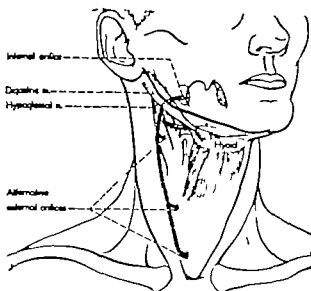


Figure 1 Course and relations of embryonic branchial sinuses. All lateral cysts and sinuses follow this tract.

under fifteen years of age more fistulae and sinuses than cysts will be found, in the age group over fifteen years there will be a predominance of cysts. Of the fistulae which require surgical treatment over half will have been present since birth, the majority of the remainder having appeared before the age of fifteen. On the other hand, cysts are found to arise more commonly as an insidious and painless swelling along the anterior border of the sternocleidomastoid muscle in the second and third decades.

While branchial cleft anomalies are usually thought to be developmental rather than genetic in origin, it is of some interest to mention that recent distribution studies of patients with bilateral defects have demonstrated a "familial tendency" i.e., an inherited tendency which has been transmitted through the male as well as the female.

Embryology Despite a high degree of consistency in the various locations, appearance and clinical courses of these anomalies, there are divergent views regarding their exact manner of origin. These entities have properly become known as branchial cysts and sinuses, because of the general agreement that those lesions found at or above the level of the hyoid bone arise from the second branchial cleft and pouch. Since some fistulae and sinuses are found below this level along the anterior border of the sternocleidomastoid muscle, some credence has been given to the consideration that their embryologic derivation may be the transient thyropharyngeal duct. The first view seems to be the more reasonable.

During the third week of intrauterine life, the human embryo presents rather clearly a series of five rounded branchial arches on either side of the neck. They are demarcated by four branchial clefts which run parallel to one another somewhat obliquely downward and anteromedially. Each external cleft is matched by an internal evagination called a pharyngeal pouch. Normally the intervening membrane between each cleft and pouch does not rupture, so there is no communication between the pharyngeal pouch and the branchial cleft.

From the first arch are developed the upper and lower jaw the malleus and incus from the second, the lesser cornu of the hyoid, the styloid process and part of the stapes from the third, the body and greater cornu of the hyoid, from the fourth, the cuneiform cartilages and most of the thy-

roid cartilage and from the fifth, part of the thyroid cartilage and the corniculate, arytenoid and cricoid cartilages.

The first branchial cleft obliterates except at its dorsal part, which becomes the external auditory meatus. The remaining clefts disappear. Internally the first pharyngeal pouch gives rise to the eustachian tube and the tympanic cavity the second may be retained as the tonsillar and supratonsillar sinus the third by entodermal outgrowth forms the thymus (by virtue of the thyropharyngeal diverticular duct) and the inferior parathyroids, and the fourth, the superior parathyroids. The tympanic membrane represents the dividing membrane between the first branchial cleft and the first pharyngeal pouch.

Each branchial arch originally is supplied by a main aortic branch, the vessels passing in an anteroposterior direction. These vessels in the first and second arches disappear as main trunks, but the third aortic arch remains as the base of the internal carotid artery. The fourth arch on the right forms the base of the subclavian artery and, on the left, it is transformed into the arch of the aorta.

Pathology and Clinical Course. Branchiogenic cysts and sinuses are characteristically located anterior to the sternocleidomastoid muscle. The cysts usually will be found high in the neck, at or above the level of the hyoid, while the sinus openings may occur at any point from the ear to the suprasternal notch. Both the cysts and sinuses are lined with stratified squamous cells but some will present columnar or ciliated epithelium. Transitional epithelium has been reported in some cysts located high in the neck. The walls are made up mostly of fibrous tissue interspersed with considerable amounts of lymphoid tissue in the form of scattered follicles. Squamous epithelium has been found by Ward lying deep in the lymphoid tissue and showing some degree of keratinization. This has been interpreted as a probable precancerous change in the same manner as one might consider leukoplakia in the mouth.

The contents of the cysts vary from clear serum to thick sebaceous material, according to the character of the lining. An opaque watery or milky fluid can be expected with squamous epithelium, whereas columnar epithelium produces a thick, sticky mucoid material. Such cysts will not transilluminate on examination. If they become infected, the fluid content becomes frankly purulent and

which at a later date becomes the hyoid bone. Its distal end corresponds to the region which later becomes the pyramidal lobe of the thyroid. The duct usually atrophies during the sixth week of fetal life, but remnants of an epithelial cord persist for a short time. Rests of cells from this cord may remain anywhere along this tract and later can produce the cysts and sinus tracts found so characteristically in the anterior cervical midline.

Pathology and Clinical Course. Thyroglossal cysts, sinuses and fistulae occur anywhere between the foramen cecum of the tongue and the region just above the suprasternal notch. They are most commonly found about halfway between these extremes, just below the hyoid bone. Some of these cysts may be located superficially while others are much deeper. The majority can be found just beneath the enveloping layer of the cervical fascia, while a few will be deep to the pretracheal fascia. These locations are dependent upon the alternative embryologic levels which the thyroglossal duct may take in relation to the hyoid bone and the fascia which attaches to its anterior and posterior surfaces. An occasional cyst can be found above the hyoid within the substance of the base of the tongue.

These cysts and tracts are lined by stratified squamous, columnar or occasionally transitional epithelium. The wall is composed almost entirely of fibrous tissue, the thickness being related to the amount of previous infection. Contrary to the characteristic finding in branchiogenic cysts, these do not have lymphoid elements in the walls. This correlates to some extent with similar findings in the preauricular cysts and sinuses of the branchial group which arise from the first arch and cleft areas. In about 80 per cent of the patients the original lesion is a cyst, while in the remainder it is a sinus or fistula. The anomaly is discovered early in life, more than half being noted from soon after birth up until the patient is ten years of age. Since the lesions produce so few symptoms, there often is delay before patients in the remaining group report for treatment.

Thyroglossal cysts vary from 1 to 5 cm. in diameter the average being about 2.5 cm. They are smooth, round and well defined. Because of their attachment to the hyoid bone, the cysts move directly with the act of swallowing but cannot be displaced by the examiner. Unless infection occurs

they are not attached to the skin. If drainage occurs they immediately become attached to the skin with formation of a permanent sinus tract. Recurrent infection is the rule in such instances, even though the cyst has been surgically drained. Intermittent drainage then becomes an objectionable problem.

Diagnosis. The salient feature in diagnosis of the average thyroglossal cyst consists in the finding of a smooth, well rounded and well-demarcated mass lying anterior to the hyoid bone which may transmit light and always moves easily with deglutition. In the absence of any sinus formation, three alternative lesions must be differentiated: submental dermoid inclusion cysts, sebaceous cysts and ectopic thyroid tissue.

Treatment. The majority of thyroglossal duct cysts and sinuses eventually will require surgical excision. The smaller cysts may not seem significant, but the larger ones obviously are disfiguring. Both the large and the small cysts can be the site of bothersome local infection. When the cyst contents become suppurative the abscess must be incised and drained as a preliminary procedure. After the induration in the surrounding tissue has resolved, the sinus tract and underlying cyst should be completely excised through a generous transverse elliptical incision. The tract or cyst will be found attached to the hyoid bone. In a few instances, there will be no further upward extension, but a considerable number will penetrate the hyoid. To prevent a recurrence, removal of the central portion of the hyoid should be included in the procedure. The persisting tract, or embryologic residue, above this

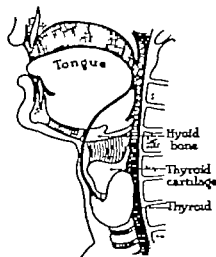


Figure 3. Diagram showing course and relations of the embryonic thyroglossal duct. Fistulae, cysts or thyroid remnants may involve any portion of this tract.

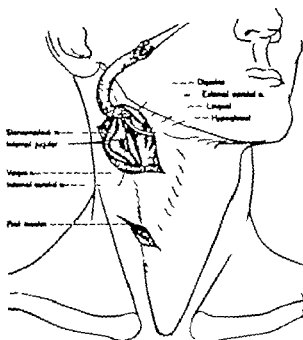


Figure 2. Dissection of branchiogenic sinus tract by means of stepladder incisions along skin creases. Typical relationship to digastric muscle and hypoglossal nerve.

sternocleidomastoid muscle. The only symptoms may be the annoying continuous or intermittent discharge of mucus. Occasionally an episode of cellulitis may occur around the tract. Traction on the external opening may demonstrate the typical upward course of the fibrous walled sinus. In children, x ray visualization of the sinus tract is apt to suggest less extension than actually exists, but in adults the well formed tract can be demonstrated by the injection of a suitable radiopaque solution. Under local anesthesia, a purse-string suture can be placed around the external opening to retain the dye, thus facilitating obtaining x-ray films in various projections. If surgery follows this examination immediately the dye can be permitted to remain in order to add some bulk to the tract for identification during the excision. Demonstration of the internal opening can be accomplished by injection of methylene blue. This should be done about two days before definitive surgery is to be performed, because the dye is apt to diffuse into the tissues about the tract and be the source of confusion. After a couple of days, it is sufficiently fixed to the tract tissues so that their identification can easily be made. In general this dye is needed only to confirm location of the internal orifice and is not necessary to outline the tract for excision.

Treatment. Branchial cysts are treated en-

tirely by surgical excision. The best approach is by means of a generous transverse incision parallel to the lines of skin cleavage and over the most prominent portion of the cyst. This will permit adequate mobilization of all surfaces since these cysts often have numerous points of attachment. Particular care should be exercised to avoid any rupture, assuring complete removal of all epithelial elements. If there is a connection with the pharyngeal wall, careful closure of this should be accomplished to prevent fistula formation.

Branchial sinuses and fistulae are best excised through the "stepladder" method developed by Hamilton Bailey which is simple and practical (Fig. 2). It allows for entire excision, permitting adequate visualization of the critical uppermost parapharyngeal area. When the fistula is complete, the internal opening can be closed by direct suture of the wall or by ligating the tract flush within the pharynx after it has been turned inside out. The skin closure produces a fine cosmetic result, since all incisions are parallel to the skin folds.

MIDLINE ANOMALIES

For the most part midline cervical cysts and sinuses arise from remnants of the thyroglossal duct. They constitute the great majority of developmental anomalies located in the anterior portion of the neck. A few anteriorly located cysts (sublingual and submental) are classified as dermoid inclusion cysts since they stem from displaced epithelium. All these lesions produce some cosmetic deformity and, in addition, frequently produce considerable degrees of distress from repeated episodes of local infection. The rare teratomas of the neck occasionally present formidable pressure symptoms in the newborn and therefore are significant.

THYROGLOSSAL CYSTS

Embryology. The thyroid anlage arises as a midline diverticulum from the first branchial pouch at a point marked in later life by the foramen cecum on the posterior portion of the dorsum of the tongue. This epithelial evagination is transformed into a stalked vesicle by the time the embryo has reached three weeks of age. It projects downward and backward from its point of origin as a tubular duct (Fig. 3). This thyroglossal duct bifurcates at its distal portion to form the thyroid lobes. During its transitory existence, the duct passes through an area

at a surprising rate and with a considerable degree of pressure particularly in the newborn.

Pathology Cystic hygroma consists of a benign multilocular cystic mass which is very thin walled. The contents are serous and straw colored though sometimes they may be blood stained. Often there is free communication of fluid between the many compartments so that the mass is not tense, but rather of a soft consistency. The cyst walls do not contain many vessels and those present are very small. Obvious lymph tissue or nodes can be found adherent to the cystic mass or as a part of the walls. On opening the specimen one will be impressed by the gray white color of the lining endothelium.

Microscopic examination of the hygroma reveals the wall to be made up of simple connective tissue, but of variable thickness and constitution. The youngest cyst will present scanty cellularity and have a myxomatous appearance. The lymphatic infiltrations will be variable. In older cysts the collagen and fibrous components will be greater in proportion to the increased thickness of the wall.

Occasional discovery of atrophied or necrotic muscle, nerve or other connective tissue has been explained by Goetsch as the result of a part of the fundamental growth and extension of the hygromas. It is his view that there are narrow cords of endothelial cells which occur as finger like projections from the sequestered lymph buds and that these cords insinuate themselves between muscle bundles, nerves, blood vessels and other structures. At a later time these cords develop a lumen which is filled with lymph like fluid and which eventually enlarges to surround the previously mentioned tissues and to include them in the cystic mass.

Clinical Course. Two thirds of these cystic masses will be found at birth, and 90 per cent of them will have developed by the end of the second year of life. Distribution between the sexes is equal. A few more are found on the left side than on the right, the ratio being about 3 to 2. They occur chiefly in the posterior triangle behind the sternocleidomastoid muscle, and often occupy the whole supraclavicular area. A smaller number will be found in the submaxillary area of the anterior triangle tending to infiltrate the floor of the mouth.

The size of the mass does not bear any positive relationship to the age of the patient or to the duration of the lesion. Some of the largest specimens have been found in infants only a few weeks old. Since the symptoma-

tology is minimal there are instances in which many months or even a year or two may pass before definite treatment is considered. The cystic mass never is tense as are thyroglossal and branchiogenic cysts. The thin and fragile wall of the early cyst is such that the borders may be poorly defined. The lesion can easily be transilluminated in this phase. With the passage of time some degree of infection may develop and a thicker walled, more fibrotic cystic mass then develops with more extensive involvement through adhesion to the surrounding parts.

Diagnosis. The characteristic presenting complaint is that of a mass in the neck which produces some disfigurement and occasionally some limitation of motion because of its extremely large size. These cysts tend to lie in a superficial plane despite the fact that they are known to infiltrate deeply even into the mediastinum. Since they do not interfere with the great vessels, trachea or esophagus, they do not present bothersome obstructive symptoms as a rule.

Considerable variation of opinion exists with regard to the value of roentgen ray examination in these patients because the full extent of the mass seldom can be determined by this method. Certainly some of the cavities can be outlined by the instillation of an aqueous iodide solution. This may demonstrate more exactly any displacement of surrounding parts, as well as give further information concerning extension of the hygroma into the mediastinum. Such positive findings often are helpful to the surgeon but negative results from such examinations should be recognized in advance as meaningless.

Treatment. A good deal of improvement in the surgical treatment of cystic hygroma has taken place in the last two decades, so that this form of therapy now is the one of choice. An expectant attitude once was advised in the hope that spontaneous regression might occur. Although temporary decrease in the size of such a cyst does occur and even a spontaneous disappearance of the mass may take place, eventually it refills and to such an extent that it may become larger.

Radiation therapy should be mentioned only to point out its ineffectiveness. Both radium as a surface application and roentgenotherapy have been used, but with minimal decrease in the size of the average mass. Suffice it to say that hygromas are radio-resistant.

Injection of sclerosing agents has been

level then can be cored out of the base of the tongue up to the foramen cecum. This type of block dissection, originally suggested by Sistrunk in 1928 reduces recurrence to a minimum. The hyoid bone need not be sutured in the midline, since this defect does not alter subsequent muscle action. The wound is closed primarily for the best healing.

Ectopic thyroid tissue occasionally will be found in the area usually occupied by thyroglossal duct cysts. This should be mentioned only as a warning because such ectopic tissue may constitute the entire supply of thyroid for the whole body. In such cases the thyroid mass will be identified as solid reddish-brown tissue rather than a substance of cystic nature. If no thyroid tissue is found beneath the strap muscles, this solid mass can be split in the middle and gently placed to each side of the trachea under the strap muscles without disrupting the blood supply.

DERMOID INCLUSION CYSTS

Submental dermoid inclusion cysts are the result of incomplete union of the first two branchial arch components. If the failure of union is external, the cyst will be more superficial and will be lined with epidermal elements, thus becoming known as a dermoid containing hair and various types of thick fluid. If the failure of union is internal, the cysts will be deeper in the floor of the mouth (sublingual) and may be lined with mucous membrane. The more common origin is faulty obliteration of the first and second clefts (ectoderm) so that the result is a simple inclusion cyst. The second cleft is the offender much more often than the first, the results being epithelial lined cysts in the aurial, submaxillary, sublingual and submental regions. They need not be attached to the overlying skin as are the usual sebaceous cysts. Since they usually are larger than the latter and contain the mixed product of the sweat and sebaceous glands, the soft cheesy material may present a doughy sensation to the examiner and when found it is characteristic. Treatment is surgical excision.

TERATOMAS OF THE NECK

Teratomas of the neck are of rare occurrence, but when present are quite significant. They usually are noted immediately after birth. They consist of large, cystic, solitary masses in the anterior portion of the neck, often causing various degrees of tracheal obstruction. Their anterior or anterolateral

location often suggests an origin from the thyroid gland, but in most instances this never is confirmed. They tend to be encapsulated and rarely in patients of this early age, are found to be malignant. From the gross as well as the microscopic standpoint, these teratomas are the same as those found elsewhere in that all types of tissue are found, with cysts and solid material being freely interspersed.

Because of their size, they usually require early resection for relief of tracheal compression. In some instances, operation may have to be performed soon after birth in order to prevent further deterioration from respiratory obstruction or difficulties with swallowing. The prognosis in such children always is grave.

CYSTIC HYGROMA COLLI

Cystic hygroma is a well recognized clinical entity which presents as an irregular endothelial lined, fluid-containing tissue mass arising from the embryonic lymphatic buds. This lesion is encountered most often in infants and young children but occasionally is seen in adults. Cystic hygromas have been found in the axilla, chest wall, groin and retroperitoneal tissues, but the cervical location is by far the most common.

Etiology. Prior to the embryologic studies of Sabin, the etiologic explanations for these growths were largely unsubstantiated. It is now appreciated that cystic hygromas occur in those areas of the neck, groin or retroperitoneal spaces in which were previously located two proved embryologic structures, that is, the lymphatic sacs of the neck and the lymphatic sacs of the iliac area. With respect to the cervical area, a capillary plexus is formed along each jugular vein which eventually is cut off from the parent vein. The resultant isolated group of endothelial-lined spaces eventually forms primitive jugular sacs by virtue of dilatation and coalescence of these capillaries. The thoracic duct connects the pelvic and retroperitoneal sacs with the left jugular sac and joins the venous system at the jugular valve. An anomaly of development occurs in a few individuals so that portions of the jugular sacs fail to establish suitable communication but retain the growth factor. In addition it has been demonstrated that hygroma cysts may extend by growth from membranous sprouts derived from the walls of cystic spaces already present. This type of expansion produces a penetrating cystic growth which permeates surrounding tissue.

Since it has been concluded that "making the diagnosis of malignant versus non-malignant carotid body tumors is a dubious venture" by the pathologist especially on a frozen section the clinician can only recall that some of these tumors in the past have proved to be so invasive as to cause death. The remarkably slow rate of growth and the critical nature of the arteries involved may be the factors which confuse the issue. It is best to realize that the actual evidence of malignancy is lower than formerly was thought but the growth potential of this particular tumor is such that, if given enough time it acts much as a malignant lesion in the sense that it has the capacity to kill the host.

Carotid body tumors cause no subjective symptoms until they reach from 5 to 6 cm. in size. Prior to this time only the mass in the neck is noted and the average patient reports that it took about five to seven years for the tumor to reach such size. Mild pain may be reported, but severe discomfort does not develop until other signs of nerve invasion have appeared.

External examination usually reveals a deeply situated tumor mass in the region of the carotid bifurcation, but the tumor never is attached to the skin. While these masses cannot be moved in the vertical plane, some will permit lateral displacement. Bimanual examination with one finger in the patient's mouth may outline the mass with considerable accuracy. When these tumors are of the more vascular type, firm squeezing pressure may temporarily reduce their size slightly by removal of some of the blood. Presence of a thrill and bruit can be elicited in a few. There may be a transmitted but not an expansile pulsation over these tumors.

Diagnosis. Diagnosis is made on the history of a unilateral cervical tumor mass of some years duration. Examination shows it to be in the carotid bifurcation area, as evidenced by its relationship to the two arteries and by the fact that the mass may be moved only in the lateral plane. Carotid angiography may confirm the diagnosis by findings suggesting an intrinsic defect in the carotid wall. Absolute diagnosis is accomplished by histologic study of either the biopsy material or the excised mass. If the study is made of biopsy material, the pathologist should not be required to rule on the question of malignancy since the diagnosis of carotid body tumor itself is difficult enough.

Treatment. In view of the fact that roent-

genotherapy has proved to be of little if any value surgical excision remains the only method by which these tumors can be eradicated. In the past, operative mortality has approximated 30 per cent when the internal carotid artery has been ligated under general anesthesia. In addition among those patients who survived the procedure numerous central nervous system sequelae have been noted. It is obvious that this is too high a price to pay for the eradication of any neoplasm whose incidence of malignancy has been shown to be less than 10 per cent. On the other hand, Munro has reported that 30 per cent of the patients whose treatment was inadequate died as a direct result of their tumors. When the tumor was removed with preservation of the vessels, only 7.5 per cent of the patients died as a result of the tumor.

It must be concluded that early surgical removal is the treatment of choice in order to prevent a mortality of approximately 30 per cent. With care this can be achieved through the simple expedient of obtaining a plane of dissection in the artery wall between the media and the adventitia. Arterial continuity thus can be preserved, but, if this is impossible definite efforts should be made immediately to restore arterial continuity by using a temporary arterial by pass, followed by the insertion of a flexible plastic arterial prosthesis at the end of the procedure.

BRANCHIOGENIC CARCINOMA

The presence of a unilateral enlargement in the side of the neck most frequently is a sign of metastatic cancer. For practical purposes, no other diagnosis in the adult should be considered until this probability has been excluded. In addition to those primary tumors previously mentioned, carcinoma of the lung, breast, kidney gastrointestinal tract and ovary can metastasize to this region. It is of interest to observe that when all of the primary malignant tumors peculiar to the neck are added up the sum does not equal the incidence of involvement of cervical lymph nodes by metastatic disease. The term "branchiogenic carcinoma" has been found in medical literature ever since 1882, at which time it was suggested that malignant lesions found in the lateral neck might arise from the vestigia of branchial clefts. Prominent pathologists have refuted the existence of branchiogenic carcinoma as a real entity pointing out that very few of the reported cases could withstand strict scrutiny. Although the term has been retained,

suggested and occasionally utilized, but this method of treatment is fraught with danger. Even though these thin walled cysts appear to be ideal for this type of therapy the complications of infection may be disastrous. On the other hand, sclerosing agents have been used most successfully by Ward, when followed within three to six weeks by surgical excision.

Surgical excision is the treatment of choice. To be curative it must include removal of all the endothelial-lined tissue. With the use of endotracheal anesthesia and extremely careful technique, the entire mass can be dissected from its multiple points of attachment. Some importance should be attached to early removal of these growths because even though they are thin walled at that time, they are more likely to be better confined to predictable fascial planes than they will later. Older growths tend to be more invasive and adhesive in character. This matter of delay may convert a relatively simple procedure into a long and hazardous operation. These wounds should be closed with suction drainage.

PRIMARY TUMORS OF THE NECK

When one considers the diagnostic possibilities presented by a unilateral cervical mass in the adult patient, on the basis of chance alone the best explanation usually is that it represents metastatic carcinoma from the oral cavity, the laryngopharyngeal area or the thyroid gland. Tuberculosis, sarcoidosis and malignant lymphoma represent other possibilities. If all of these can be excluded, efforts then can more logically be made to identify the mass as one of the less common primary cervical tumors. After branchial cysts and salivary gland tumors are eliminated from consideration, specific soft tissue tumors such as lipoma, neurofibroma and fibrosarcoma may enter into the clinical estimate. In such a differentiation, carotid body tumors and aneurysms should be included for the sake of thoroughness. Because these various lesions present such difficulties regarding diagnosis it is not unusual that the real identity is not established until the mass in question has been surgically exposed and an adequate biopsy study made.

CAROTID BODY TUMORS

Although fewer than 400 carotid body tumors have been reported in the medical literature, this lesion always is a matter of diagnostic and therapeutic importance when

considering lateral neck tumors. Full appreciation of all aspects of these lesions is most desirable for the best therapeutic results because the integrity of the internal carotid artery is directly involved in the surgical excision. Definitive handling of the carotid artery has much to do with the post-operative course and, for this reason this entity presents a real challenge when discovered.

Embryology The carotid body is a small, ovoid, irregular mass of pinkish-tan or gray tissue located within the bifurcation of the common carotid artery. At one time it was thought to be related to the so-called chromaffin system but this does not seem to be the case since it actually is one of a group of discrete tissue masses found near ganglia of the cranial nerves or branchial arch arteries.

These "organs" and the tumors they produce are histologically similar. Though these tumors are recognized as paragangliomas the term chemodectoma has been used more frequently since first introduced in 1950. They do not give a true chromaffin reaction, contain no epinephrine and do not secrete a hormone. The nerve supply of these bodies is multiple, but mostly sensory: the carotid body for example, being supplied by the glossopharyngeal nerve. These bodies have been described near the jugular bulb, the ganglion nodosum of the vagus nerve, the innominate artery, the tympanic membrane and the carotid bifurcation. The carotid body in particular is known to arise within the adventitial layer of the carotid artery early in embryological development. Although it grows outward from this point as a discrete mass, it never loses this intimate anatomic relationship. In order to excise such a mass, dissection between the media and the outer adventitia is necessary.

Pathology and Clinical Features. Carotid body tumors are noted for their slow rate of growth. Estimation of malignancy from the histologic appearance has been difficult since mitotic figures are exceedingly rare in these tumors. They show a remarkable tendency to reproduce the normal architecture of the carotid body. Although they may vary from an epithelium-like appearance to that of an angioma-like type, the fundamental pattern of tissue origin can best be discovered by the use of reticulum silver impregnation stains. The majority of them are somewhat adherent to their surroundings, especially to the arterial wall, but the latter point is of embryologic and not neoplastic significance.

For example, a peritonsillar infection can spread directly into the pharyngomaxillary space or more commonly the submaxillary space will present a complication of dental infection without any break in mandibular continuity. In addition to these there is a small group of infections which are secondary to a great variety of open and closed injuries in or about the mouth and neck.

Applied Anatomy Cervical fascia. The cervical fascia commonly is described as consisting of a superficial and a deep layer. Such a division does exist but the deep layer is much more complicated and important than such a simple classification would suggest.

The superficial cervical fascia consists of a layer of loose connective tissue located immediately superficial to the platysma muscle over the anterolateral aspects of the neck. It is the *tela subcutanea* which gives support to the skin and which carries a generous supply of nerves, vessels, fat and some muscle. The platysma is intimately attached to it. The superficial fascia encircles the neck as a single layer maintaining the same relationship to the skin throughout its distribution.

Immediately beneath the platysma, the deep layer of cervical fascia is clearly defined (Fig. 4). The outermost (anterior) layer is an enveloping layer surrounding the large neck muscles so as to invest and hold them together. In the posterior half of the neck the deep fascia functions entirely to support the heavy musculature whereas in the anterior half (musculovisceral compartment) it consists of a number of layers, some ensheathing the long muscles, some covering the viscera and the deepest muscles lying over the vertebrae.

As the enveloping layer of the deep cervical fascia extends upward, it attaches anteriorly to the hyoid bone and, after splitting, it attaches to and extends upward over both surfaces of the mandible. It reaches inferiorly to the clavicle and sternum, attaching to the anterior and posterior surfaces to form in the midline the space of Burns. As it extends laterally it divides to enclose the sternocleidomastoid muscle, crosses the posterior triangle and invests the trapezius. It continues around dorsalward to attach to the spines of the cervical vertebrae.

The middle layer of the deep cervical fascia is also known as the pretracheal fascia.

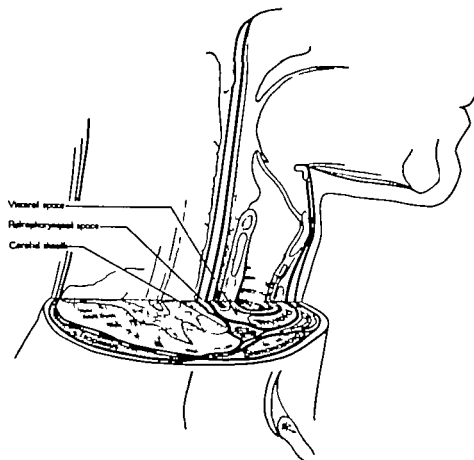


Figure 4. Deep layers of cervical fascia. Direct access to mediastinal area from upper visceral space is evident.

it seems likely that few examples will be substantiated in the future.

Diagnosis. In order to make the diagnosis of branchiogenic carcinoma the following criteria are necessary (1) the tumor must be located along a line drawn anterior to the ear which passes downward along the anterior border of the sternocleidomastoid muscle (2) the histologic appearance of the tumor must suggest origin from epidermal branchial vestigia (3) the tumor should demonstrate some relationship to a cyst or sinus tract which might be considered branchial in origin (4) after removal of the tumor the patient must survive five years without the development of any other primary malignant lesion which could possibly have produced, by metastasis, the previously excised lesion.

Treatment. If no other primary lesion can be found and the unilateral neck mass is reported as "epidermoid carcinoma," it might be well to defer surgery in the interests of prudence. Radiation can be used for temporary control of the suspect metastatic lesion while sufficient time passes to permit further exploration for the primary lesion. In a few patients, a complete neck dissection may be indicated if the presenting mass is quite large or if the mass proves to be radioresistant.

DEEP CERVICAL INFECTIONS

While not so common in the present practice of surgery deep cervical infections once were the cause of considerable morbidity and in the neglected state carried an unusually high mortality. Abscesses are particularly prone to occur in the deeper layers of the neck because the deeply situated lymphatics receive drainage of infected material from the oropharynx and upper respiratory passages. Such infections are not only deep in location but also in origin, since the source of infection may be the pharynx, dental structures, temporal bone, salivary glands, the esophagus and respiratory passages. The majority of the abscesses arise from dental infection or from infected tonsils. Continued improvement in the care of the teeth and over-all oral hygiene combined with earlier correction of throat infections have done much to diminish these very troublesome and serious infections.

While, for many reasons, deep cervical abscesses previously were accompanied by a high mortality rate, one of the most significant reasons was the difficulty in accurate localization of the infection because of fail-

ure to appreciate the important relationships of the deep cervical fascia. Even though today we possess a chemotherapeutic and antibiotic armamentarium of considerable potency early drainage of retained purulent exudate still is required for prompt recovery. The application of the principles of early surgical drainage occasionally is necessary in deep cervical infections and adequacy of such drainage often is dependent upon knowledge of these deep anatomic relationships.

The universal recognition of the importance of fascial planes in the diagnosis and treatment of neck infection has been a relatively recent development. The fascial planes of the neck form well-defined compartments and potential spaces. Infection confined within these compartments may be so hidden that the diagnosis of the presence of an abscess may often be seriously delayed. Extension into the mediastinum may take place while the patient is being observed. Moreover cardiorespiratory complications may intervene before appropriate drainage has been instituted. Early diagnosis and appropriate treatment often are delayed because the signs usually associated with the accumulation of pus elsewhere in the body may not be manifest when there is a purulent pocket in the deeper structures of the neck.

Etiology. Almost all known organisms have at some time or other been cultured from deep cervical abscesses. The great majority of these infections are due to some strain of streptococci, the latter frequently being obtained in a pure culture. It is not unusual to find a mixed infection, especially with both aerobic and anaerobic variants. While the fusospirochetal organisms of Vincent may be found in both the oral source and the abscess cavity the blood stream invader may be only the hemolytic streptococcus. Accurate identification of these organisms is important from the therapeutic standpoint because specific sensitivities can be determined to assure the correct choice of antibiotic or chemotherapeutic agent.

Not only is the organism significant, but so are the portal of entrance of the bacterial invaders and the manner in which the infection has developed. Two mechanisms may be at work in the pathogenesis of these infections that is, there is the possibility of contiguous spread of the infection by direct extension or embolic spread may occur through the lymphatics to the collecting lymph nodes within certain compartments.

over the lower portion of the body of the mandible just posterior to the facial vessels as they lie in relationship to the anterior border of the masseter muscles. The muscle fibers can be divided by blunt dissection and an appropriate soft rubber drain inserted into the pocket. The superior extension of this cavity should be identified since the temporal extension of this compartment may also require drainage near the zygoma.

PAROTID SPACE. This space contains only the parotid salivary gland, a few significant lymph nodes, a major portion of the facial nerve and numerous vessels. The fascia which invests the masseter muscle thickens considerably at its posterior border and splits to surround the parotid gland. This thick parotid masseteric fascia is firmly attached to the investing layer of the deep cervical fascia which covers the sternocleidomastoid muscle and, as a result, the gland is completely surrounded by heavy fascia except medially where the parotid tends to become retromandibular. At this point, the deeper layer of the fascia extends toward the pharynx in the region between the styloid process and the internal pterygoid muscle. This lack of covering permits free connection with the retropharyngeal and the pharyngomaxillary spaces at this point.

Infection originating within this space usually is directly related to the salivary gland itself. If it becomes secondarily involved from masticator or pharyngomaxillary space infection, the abscess can be drained by a small vertical incision made over the most prominent dependent portion of the swollen gland. The thick capsule must be opened transversely to avoid injury to the facial nerve. More than one area may require incision. The gland then can be spread by blunt dissection to obtain drainage.

THE RETROPHARYNGEAL SPACE. The retropharyngeal space is that area found between the prevertebral fascia and the posterior wall of the pharynx. It contains rather loose connective tissue and a few lymph nodes. Inferiorly this space is in direct contact with the mediastinum.

Retropharyngeal abscess most commonly is found in early childhood. At least two well-defined lymph nodes are located in this space at about the level of the second cervical vertebra. Infection in the nose, nasal sinuses or nasopharynx may spread to these nodes or infection may arise within the adenoid region of the posterior nasopharynx and thus extend directly into this area. This may easily become a serious infection since

it lies upon the prevertebral fascia and thus it may extend downward into the mediastinum or laterally into the pharyngomaxillary space and eventually may involve the sheath of the great vessels. In neglected patients, this retropharyngeal swelling may protrude so far down the posterior pharyngeal wall as to involve eventually the larynx and complicate respiration.

The symptoms may begin with fever and a cough and examination may not reveal the indistinct boggy swelling of the posterior pharyngeal wall. Dysphagia and dyspnea develop later. Lateral roentgenograms of the cervical spine area may be helpful in making the diagnosis inasmuch as the soft-tissue swelling displacing the pharynx forward may thus be identified. This abscess should be incised and drained orally before it extends into the lateral cervical region. It is most important in doing this to keep the patient's head low and to use suction to facilitate the immediate drainage.

THE PHARYNGOMAXILLARY SPACE. This is a cone-shaped potential space whose base is in relation to the skull around the jugular foramen and whose apex is at the greater cornu of the hyoid bone. Medially it borders upon the superior constrictor of the pharynx and the tonsillar fossa. The posterior portion is in direct relationship with the retropharyngeal space, the prevertebral fascia actually forming its posterior border. The lateral surface anteriorly is in relation to the internal pterygoid muscle while more posteriorly it faces the retromandibular portion of the parotid gland which is not covered by fascia. At this level, as well as below the parotid, the space is in direct contact with the carotid sheath and its contained vessels.

There was a time when infections in this space accounted for over half of the deep infections of the neck, but this number has been greatly reduced in the last decade owing to better control of infection in the usual portals of entrance. Because of its position, this space may be infected from the tonsillar fossa and peritonsillar region, over half of these infections coming from this source. It has been said that every peritonsillar infection is a potential pharyngomaxillary space infection. Less common sources are mastoid infection, infected retropharyngeal nodes, molar teeth and invasion from the parotid and submaxillary spaces. The intimate relationship of the vessels which traverse this compartment is of extreme importance.

Purulent exudate in the pharyngomaxillary

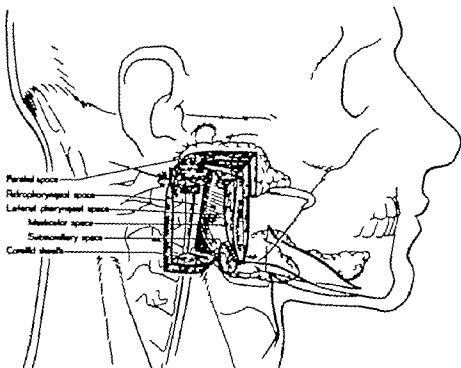


Figure 5 Cut-away diagram to show intimate relationships of fascial spaces in parapharyngeal regions.

It invests the sternohyoid, sternothyroid, thyrohyoid and omohyoid muscles. These split layers fuse laterally and attach to the carotid sheath for firm anchorage. This middle layer attaches superiorly to the hyoid and inferiorly to the posterior surface of the sternum and clavicle, fusing with the enveloping layer of the deep fascia.

The visceral layer of the deep cervical fascia encloses the hypopharynx, esophagus, trachea and thyroid gland, known as the buccopharyngeal fascia. It covers and supports the outer wall of the pharyngoesophageal region, especially the constrictor musculature. The thyroid is covered by this layer by virtue of its embryologic descent through it, as it moved from the midline region to take up a position lateral to the trachea. This visceral fascial layer forms a definite compartment between the middle layer and the firm prevertebral fascia. It extends from the base of the skull to the thoracic mediastinum.

The prevertebral fascia is the deepest (posterior) layer of the cervical fascia and as such covers the bodies of the cervical vertebrae, the longus colli and the scalene muscles in the posterolateral neck area.

Fascial spaces (Fig 5) THE MASTICATOR SPACE. The masticator or submasseteric space includes the area which contains the masseter temporal and both pterygoid muscles and all the associated vessels and nerves. This compartment is the result of the

upward splitting of the outer layer of the deep fascia as the latter comes into relation with the mandible. While this space is not primarily cervical in location, it has a direct bearing upon the differential considerations of a deep cervical abscess.

Infection may reach the masticator space by direct extension from the second and, especially the third lower molar teeth. Occasionally this region may become indirectly involved by infection from the buccal area anterior to the body of the mandible, from the floor of the mouth or from the parapharyngeal region. Pain in the region of the masseter muscle with some swelling and tenderness accompanied by evident trismus indicates infection in the masticator space. An appropriate dental evaluation is necessary to confirm this diagnosis. Owing to the severe trismus the patient may be unable to cooperate so that examination may be limited to a roentgenogram of the jaw. A typical area of radiotranslucency will confirm the source of the infection as dental. If left untreated, this type of infection will often spread to the submaxillary compartment or posteromedially into the pharyngomaxillary space. Although accompanying cellulitis will be noted over the angle of the jaw area, a direct extension of the infection posteriorly into the parotid compartment is quite unlikely owing to the thick fascial demarcation. Drainage of this space is accomplished by a small transverse incision

one or both sides of the midline. This differential characteristic is very important since only this latter type of infection carries with it any significant morbidity. When the objective findings of inflammatory involvement are such as to make the diagnosis of Ludwig's angina, early surgical drainage is required, while with the other infections some degree of temporization may be permitted.

The portal of entry of this infection may be a lesion anywhere about the tongue floor of the mouth and teeth of the lower jaw. The role of dental infection is increasing and is reported to be as high as 90 per cent in some series. The infection may arise either by lymphatic spread or by contiguous extension from infection in the floor of the mouth. In either manner the sublingual and submaxillary compartments eventually both become involved. The end result is the development of diffuse, edematous and rapidly spreading cellulitis beneath the outer layer of the deep cervical fascia.

Owing to the restraining capacity of the deep cervical fascia, and to a lesser extent of the mylohyoid muscle, inflammatory swelling within the submaxillary and sublingual compartments pushes the tongue upward and backward within the confines of the oral cavity. The patient will have difficulty with speech as well as swallowing. The infection eventually may cause obstruction of the respiratory passages. In addition, a certain number of patients will die from pulmonary infection because of extension if appropriate antibiotics are not used.

The usual clinical history of typical Ludwig's angina is that, following a dental extraction or known dental infection, a painful swelling develops in the floor of the mouth, over a period of two to four days accompanied by gradual appearance of trismus. Examination of the floor of the mouth, when possible, reveals considerable edema, induration and tenderness. The tongue may be so markedly swollen as to protrude between the teeth and press upon the palate. The submaxillary compartment involvement is manifest by the tense, brawny tender swelling in the suprahyoid region. This swelling may be either unilateral or involve the entire suprahyoid region on both sides of the midline.

Since the development of chemotherapy and antibiotic therapy not only has the incidence of this infection been greatly reduced, but also its mortality. In the fully developed case respiratory obstruction still

remains the most feared complication despite these advances.

One should not wait for fluctuation as a sign of localization or it may be too late. The phlegmonous exudate always is quite deep, tends to expand and hardly ever is purulent. It is possible in some subjects to abort the process by adequate drainage of a primary intraoral source either through the mouth or through the submaxillary fossa as indicated. In the neglected individual the need for maintenance of a satisfactory airway may take priority over plans for definitive drainage. Early tracheotomy under such circumstances will assure improved respiratory exchange so that the necessary drainage of the infection then may be accomplished with greater safety. Drainage is accomplished through a generous transverse, curving incision below and parallel to the outline of the under surface of the body of the mandible. The deep cervical fascia and the mylohyoid muscle are cleanly divided in order to open widely the submaxillary and sublingual compartments.

The essentials of successful treatment are the recognition of the need for tracheotomy and the value of early drainage of both the sublingual and submaxillary compartments. General anesthesia is not to be feared if

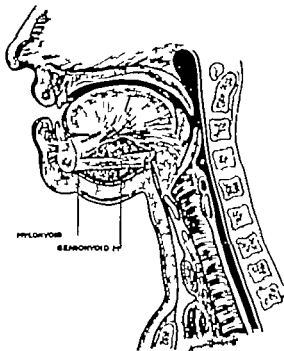


Figure 7 Semidiagrammatic sketch showing the location of infection in Ludwig's angina above or below the geniohyoid muscle. Note the bottle-like compartment filled by the tongue and adjacent structures and the ease with which inflammation may extend to the region of the larynx.

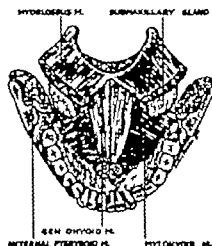


Figure 6 Semidiagrammatic sketch showing the diaphragm formed by the structures in the floor of the mouth.

fossa may cause the lateral pharyngeal wall to bulge inward. Such change in contour may be mistaken for a simple peritonsillar abscess but careful inspection will reveal that there is very little swelling of the tonsils themselves. The intimate relationship of the infection to the internal pterygoid muscle and the pterygomandibular ligament frequently results in trismus as an early sign. There also may be some swelling of the parotid gland because of involvement of the exposed retromandibular portion which is not covered by fascia and lies in direct relationship with the lateral aspect of the fossa. These three findings, internal pharyngeal swelling, trismus and parotid swelling, are indicative of deep infection within the pharyngomaxillary space. Early diagnosis is of great importance because, if undetected, these infections may lead to serious deep jugular phlebitis.

Drainage of this space can be accomplished by a generous transverse incision in the lower submaxillary region anterior to the border of the sternocleidomastoid muscle. After the lower portion of the facial vein is divided, the submaxillary gland is elevated. To gain access to the latter the outer layer of the deep fascia must be cleanly incised just above the level of the hyoid. Blunt dissection in the direction of the angle of the jaw and toward the styloid process deep to the mastoid tip will result in a free flow of pus as the pharyngomaxillary space is reached. Insertion of a soft rubber drain completes the procedure.

THE SUBMANDIBULAR SPACE. This is rather a large space located immediately beneath the body of the mandible. It is outlined by the peripheral borders of the submental and

submaxillary triangles and its depth consists of all the tissues lying between the mucous membrane of the floor of the mouth and the outer layer of the deep cervical fascia (Fig. 6). The submandibular space contains the submaxillary and sublingual salivary glands, numerous related lymph nodes and the hypoglossal and lingual nerves and vessels. The anterior belly of the digastric, the mylohyoid, the geniohyoid and the geniohyoid muscles add structure to the space.

It should be appreciated that there are three salient compartments within the submandibular space owing to the diaphragm-like arrangement produced by the mylohyoid muscle. The deepest compartment and actual floor of the mouth is the sublingual space. It contains those structures which lie superior to the mylohyoid and extends from the hyoid bone to the mandible. It connects at the midline with the contralateral sublingual space and with the ipsilateral submaxillary compartment by virtue of the course of the submaxillary salivary gland at the lateral edge of the mylohyoid. The submaxillary compartment contains the submaxillary gland and intimately related lymph nodes. It communicates posteromedially with the pharyngomaxillary and retropharyngeal spaces and thus indirectly with the mediastinum. The third, or submental, compartment consists of the superficial area between the median raphe and the submaxillary border deep to the outer layer of the deep cervical fascia but superficial to the mylohyoid muscle.

Infections in the submandibular spaces may vary from mild cellulitis to fatal phlegmon (Ludwig's angina). Since the different compartments of this space may participate in these infectious processes in varying degree, the anatomic relationships of the area must always be kept in mind (Fig. 7). While the right and left halves of this area may become confluent in the midline when serious infection is present, it is the deep and superficial sections of each half that must be separately evaluated. This is of specific importance in order that a correct diagnosis of Ludwig's infection be made in the event that it should occur. A submental abscess, submaxillary adenitis, cellulitis of the submaxillary gland or an infection limited to the floor of the mouth (sublingual) can be identified rather easily when this anatomic subdivision is kept in mind. On the other hand, a typical Ludwig's infection involves all three compartments, the sublingual, submaxillary and submental, on either

Superficial Wounds. Except for hemorrhage, superficial lacerations of the neck present no problems which differ from those in wounds of similar depth and extent found in other parts of the body. The external and anterior jugular veins which are located immediately deep to the platysma muscle layer may produce a startling amount of bleeding when divided. If the neck wound is uncomplicated by other injuries appropriate first-aid care should be a simple but effective compression dressing until the patient can be transported to a point at which definitive surgical debridement and repair can be accomplished. The struggling behavior of an uncooperative or apprehensive patient will serve to increase the venous back pressure in the jugular system sufficiently to reactivate the venous bleeding to a distressing degree. Since some of this blood comes from the central side of the lacerated vein a portion of this hemorrhage can be prevented by elevating the head 45 degrees during transportation.

Deep Wounds. Wounds which penetrate the outer or enveloping layer of the deep cervical fascia usually are of considerable significance. The deep fascia surrounds the vascular and visceral compartments of the anterior neck in such a manner as to retain any extravasated blood or exudate. The major complications therefore from such injuries are hemorrhage, both internal and external and respiratory obstruction of varying degree and infection. Each and all of these complications can result from severe blunt injuries as well as from wounds by sharp knives and penetrating bullets. A few significant nerve injuries also may occur with penetrating wounds.

A severe contusion on the side of the neck, such as that sustained in boxing when the chin is not flexed to protect the soft parts, may be very harmful. Varying degrees of damage to the wall of the larynx and trachea, as well as deep hematoma formation from ruptured veins, may result. Respiratory difficulties secondary to submucosal hemorrhage, and later edema, may first present as expectoration of blood tinged sputum, hoarseness, dyspnea or even labored breathing with cyanosis. Severe respiratory difficulty may result from the pressure of a large expanding hematoma before it has been decompressed.

The most common incised wound of the neck often is reported to be that inflicted in attempted suicide. This wound usually is made by the right hand and so will extend

more into the left than the right side of the neck. It may be located above the hyoid bone since the chin probably was extended upward. A deep wound at this level may reach the pharynx and even injure the epiglottis. At a lower level, the trachea may be severed by a similar transverse wound producing profuse bleeding from the anterior jugular veins as well as from the thyroid gland. It is quite possible that the large vessels of the carotid sheath will be protected by the sternocleidomastoid muscle or by virtue of their posterior position as compared with those of the injured trachea and thyroid gland.

Anterior and anterolateral knife wounds from an assailant, commonly described as "cutthroat" present the immediate dangers of severe hemorrhage and asphyxia. Deep penetrating or puncture wounds of the neck by knife or missile often are rapidly fatal, either as a result of immediate arterial hemorrhage or from extravasation of blood causing early respiratory obstruction by pressure. Hemorrhage may stop when the blood pressure falls to shock level, only to begin again when the patient strains or struggles or when the wound is manipulated. Sudden asphyxia may result from tracheal division, through either retraction of the cut edges or infolding of a part, or by inspiration of a clot of blood into the larynx or trachea. Division of the vagus or recurrent laryngeal nerve in the depths of such wounds causes further respiratory embarrassment through paralysis of the corresponding vocal cord.

Stab wounds in the supraclavicular region or injuries accompanied by forcible depression of the shoulder may result in severe and sometimes permanent damage to the brachial plexus by severance or by stretching. These lateral incised wounds of the neck occur chiefly in fights or in attempted homicide. They often are followed by severe hemorrhage and, occasionally by the late development of arteriovenous fistulae. When the supraclavicular space is involved, the subclavian veins and their branches and the thoracic duct and the lung may be injured. The possibility of a complicating pneumothorax or a delayed chylothorax must be kept in mind.

Bullet wounds, as well as the lacerations of war and civil life are prone to infection. The neck is filled with numerous lymphatics, blood vessels, nerves, viscera and muscles. Some of the neck viscera are hollow and, if perforated, predispose to infection of the

Pentothal sodium is utilized, provided the trachea is first exposed for purposes of tracheotomy if needed, or provided a preliminary tracheotomy already has been accomplished.

THE CAROTID SHEATH. This space contains the great vessels of the neck and, in its more superior area, some of the emerging cranial nerves. It gives off fascial connections to the parotid, submandibular and pharyngomaxillary spaces. This relationship has been likened to a trifolium, the stem of which is the carotid sheath. The arterial and venous branches leave the sheath to enter these compartments invested with the fascia that composes the sheath and which is in direct continuity with the retentive and supportive layers of the deep cervical fascia. Inflammatory interrelationships between these spaces and the carotid sheath thus are quite real and often produce serious complications by virtue of such extension.

Because of its anatomic location, the carotid sheath can readily be infected from the parotid, submaxillary retropharyngeal and, most commonly from the pharyngomaxillary space abscesses. It often has been pointed out that local signs are of little value in estimating the possibility of a carotid sheath infection or jugular phlebitis. The swelling and local tenderness in the region of the vessels usually arise from related jugular nodes involved by the prime infection. The only reliable data are those suggestive of severe sepsis, chills and fever, spiking temperature, leukocytosis and positive blood culture. Vigorous antibiotic therapy must be maintained. The importance of penicillin in particular should be stressed. Since an underlying deep abscess is the usual cause of carotid sheath infections, one must not forget the need for surgical drainage if the pocket can be found. Ligation of the deep jugular vein and drainage of the hidden deep abscess may quickly solve the problem.

VISCERAL SPACE INFECTIONS

Trauma from food, from foreign bodies and from instrumentation may result in infection developing within the bounds of the pretracheal fascia, below the level of the superior constrictor muscle. This same space may be involved secondarily by extension from the prevertebral area, or the pharyngomaxillary fossa. Wounds and injuries of the neck also may result in infection along this enveloping fascia. Compound lacerations of the larynx, trachea or esophagus or tears

and lacerations of the cartilage from blunt blows in the neck can be followed by this type of infection.

Visceral space infections always are serious, especially those following esophageal perforation. Mediastinitis is an almost immediate development, occurring within one to four days before the usual protective barriers can be established by nature. Subcutaneous emphysema is diagnostic of the perforation and can be established either by the characteristic crepitus on physical examination or by roentgenograms. Soft tissue films may demonstrate air under the skin, in the anterior mediastinum or in the retropharyngeal space, under these circumstances. When appropriate early treatment has been omitted, the more fully developed infection will present signs and symptoms characteristic of the viscera involved. Dysphagia may occur when the infection borders upon the constrictors, while alterations in voice tone follow laryngeal edema. Dyspnea may result from either laryngeal obstruction or bronchopneumonia. A sustained febrile course will indicate the severity of the mediastinitis.

Once the presence of subcutaneous emphysema has been established, the treatment of choice for perforative infection of the visceral space is immediate surgical decompression. The deep fascia should be opened widely and the area drained to prevent further extension downward into the mediastinum. If visceral space infection is of the secondary type treatment is aimed at correction of the source. This may mean simple intraoral drainage of a peritonsillar abscess or exploration and drainage of the pharyngomaxillary space. If obstructive dyspnea is present, a temporary tracheotomy may be required.

WOUNDS OF THE NECK

Injuries to the soft parts of the neck occur somewhat less often than might be expected in view of the extent of their surface in relation to other parts of the body. A portion of the anterior neck often is protected by a flexed jaw or raised shoulder. While such maneuvers often prevent or diminish the force of a blunt injury they may compound or multiply the number of structures injured in instances of penetrating injuries. For example, fracture of the jaw is a common accompaniment of the deep neck wound. A stab wound at the base of the neck often involves the great vessels of the upper chest and even the heart.

stage if respiratory obstruction is present. Atropine will help to diminish the bronchial secretions, but suction through the tracheotomy opening may be necessary to accomplish an adequate bronchial toilet.

After the general condition of the patient has improved, endoscopic and roentgen ray examination can be carried out with more care in order to determine more fully the extent of the injury and to detect the presence of any hidden foreign bodies. If multiple injuries are present, all must be properly assessed and therapeutic priority established. With this information at hand, the wounds then can be repaired.

A general anesthetic should be administered, either by an endotracheal tube or in conjunction with the tracheotomy. Selection of the anesthetic for the patient with a serious neck injury is very important because a high oxygen content is absolutely essential. Although local anesthesia may be used for many elective surgical procedures in the neck, it is not suitable for these patients.

Following appropriate skin cleansing, the packing is removed from the depths of the wound. This is replaced by more specific pressure and each vessel is carefully ligated as fresh bleeding recurs. In knife or bullet wounds, a generous extension of the wound ordinarily will be necessary to obtain satisfactory exposure and control of all bleeding points. Bleeding from large veins may be more troublesome to control than that from arteries because vein walls are so fragile that they tear easily when handled. Ligation of a large vein requires good exposure and gentle technique. Débridement of devitalized tissue and removal of all foreign bodies are done as indicated. Clots are removed and the wound generously irrigated with saline solution.

While major veins in the neck can be interrupted without fear of central complications, every effort should be made to restore vascular continuity when the carotid artery has been injured. Ligation of the common carotid artery involves the risk of hemiplegia or fatality when it is performed under general anesthesia and in the presence of shock. Suture of lacerations of the carotid wall or end-to-end anastomosis should be performed whenever possible. When large defects are present, arterial homographs or flexible plastic substitutes have proved to be successful on a few occasions.

A complete anatomic survey then is necessary in order to identify lacerations in the viscera. Small defects in the trachea can be

sutured and covered with near by thyroid or muscle. Sometimes a two-layered closure of the pharynx can be obtained if the surrounding tissues are properly mobilized. Major defects in the larger muscles can be closed by loose suture. When viscera have been perforated, no attempt should be made to close the deep cervical fascia. A small thin, rubber drain can be permitted to come to the outside from the visceral space, but it never should be left in direct contact with the sutured wall. These maneuvers serve to decompress the retropharyngeal and visceral spaces and therefore diminish the potential of mediastinal infection. Gentle suction drainage will facilitate both wound healing and postoperative nursing care.

In the course of immediate after-care all patients with deep neck wounds should receive penicillin or other appropriate antibiotics in order to reduce the likelihood of infection. Nutrition and fluid balance can be maintained by the early and continuous use of small nasogastric tubes. The late complications of persistent orocutaneous fistula, carotid aneurysm and various arteriovenous fistulae are entities which can be avoided by appropriate early surgical care.

READING REFERENCES

- Arey, L. B. *Developmental Anatomy* 8th ed. Philadelphia, W. B. Saunders Company 1954.
- Bailey, H. *Clinical Aspects of Branchial Fistulae*. Brit. J. Surg. 21 173, 1933.
- Beck, A. L. Deep Neck Infections. Ann. Otol., Rhin. & Laryng. 56:499, 722, 1947.
- Bill, A. H., Jr. Cysts and Sinuses of the Neck of Thyroglossal and Branchial Origin. S. Clin. North America, 36:1590 1950.
- Buckner, F., Lyons, C., and Perkins, R. Management of Lacerations of the Great Vessels of the Upper Thorax and Base of the Neck. Surg. Gynec. & Obst. 107 135, 1958.
- Byars, L. T., and Anderson, R. Anomalies of the First Branchial Cleft. Surg. Gynec. & Obst. 93:755, 1951.
- Casberg, M. A. The Clinical Significance of the Cervical Fascial Planes. S. Clin. North America 30:1415, 1950.
- Coller, F. A., and Yglesias, L. Infections of the Lip and Face. Surg. Gynec. & Obst. 60:377 1935.
- Davis, R. A., Wetzel, N., and Davis, L. An Analysis of the Results of Treatment of Intracranial Vascular Lesions by Carotid Artery Ligation. Ann. Surg. 143:641 1956.
- Fogelman, M. J., and Stewart, R. O. Penetrating Wounds of the Neck. Am. J. Surg. 91 581 1956.
- Gross, R. R. *The Surgery of Infancy and Childhood*. Philadelphia, W. B. Saunders Company 1953.
- Hollinshead, W. H. *Anatomy for Surgeons: The Head and Neck*. New York, Paul B. Hoeber Inc., 1954.
- Jainke, E. J., Hughes, C. W., and Howard, J. M.

surrounding structures. Marked contamination of the deep tissue, involving directly the deep spaces may occur. Foreign bodies may be driven into the area and near by tissue damaged by the traumatic force. Anaerobic infection may be likely especially when significant foreign bodies are not removed. Tetanus and gas-forming infection therefore should be feared.

Delayed or secondary effects within these deep wounds are edema of the larynx, varying degrees of tracheobronchitis, pneumonia, infection along the prevertebral fascia with possible mediastinitis and secondary hemorrhage. If a suppurative process develops not only mediastinitis, but also jugular septic phlebitis, must be feared. In all of these complications glottic edema frequently may be the key to a chain of events leading to fatality. Maintenance of an adequate airway is essential and, therefore, early tracheotomy frequently will be necessary as a prophylactic lifesaving maneuver either at the time of primary repair or later when dyspnea develops.

Vascular Injuries. Fortunately injuries to the great vessels in the neck and upper thorax occur rather infrequently. The significant vessels involved are the carotids, thyrocervical axis, vertebral, subclavian and innominate arteries, the jugular subclavian and innominate veins and the arch of the aorta. Hemorrhage from these vessels can be so rapid and severe as to exsanguinate the patient before definite care can be attempted. In addition, strangulation by compression of the trachea may result from the effects of a massive, rapidly expanding, subfascial hematoma. Fatal cerebral thrombosis or varying degrees of the hemiplegic state may be late central complications of carotid injury in those who survive thrombosis. Late local effects consist in the formation of carotid aneurysms and arteriovenous fistulae.

First aid for both arterial and venous hemorrhage should consist of direct packing of the wound reinforced by a gentle compressive dressing. While formidable bleeding may suggest the need for a more vigorous and more specific on-the-spot type of surgical "first aid," one should withstand the temptation to perform such heroic attempts because sutures and ligatures applied under adverse and contaminated circumstances add to infection and increase the possibility of intravascular thrombosis. Such a procedure merely complicates the later definitive surgical care. Immediate efforts should be directed toward proper transportation of the

injured patient to a hospital where deliberate surgical repair can be accomplished.

Visceral Injuries. Penetration of the hollow cervical viscera, that is, the oropharynx, esophagus, larynx and trachea, by the wounding mechanism permits bacterial invasion to take place along the fascial planes of the neck. Infection from these mucous membrane-lined structures especially from the pharynx and esophagus, is far more significant than infection into the wound from without. In a bullet or knife wound, the presence of subcutaneous emphysema, noted either by physical examination or by soft tissue roentgenogram, indicates that one or more of these organs have been perforated. Wounds involving the floor of the mouth, pharynx or esophagus can be expected to heal quite well if appropriately repaired with adequate drainage of the visceral space. Small nasogastric tubes should be used for feeding purposes. On the other hand, if infection develops, these wounds are characterized by a foul exudate with considerable leakage of saliva. Edema of the glottis, mediastinitis or abscess formation with severe, secondary venous hemorrhage may result. Although there may be difficulty with nutrition and healing is slow the resultant orocutaneous fistula in some patients eventually may close spontaneously but in others secondary closure will be required.

When the larynx or trachea has been cut across or severed by a bullet, there is a tendency for the parts to separate. A temporary tracheotomy may be required to facilitate good oxygen exchange and to permit safe transportation. If the division is complete, the lower end may retract beneath the sternum and be drawn downward with each inspiratory effort, especially if the distal opening is covered with soft tissue. When early repair is not possible the lower cut end can be brought to the surface and a tracheotomy tube inserted as a temporary measure. Reconstruction can be done at a later time.

Treatment. The general principles of wound treatment in the neck are the same as those for any other part of the body except that the local anatomy produces a few problems peculiar to the neck. Control of bleeding and maintenance of an adequate airway are of prime importance. In serious injuries, blood transfusion may be required to stabilize cardiovascular mechanics before surgical intervention can be attempted. A temporary tracheotomy performed under local anesthesia should be considered at this

stage if respiratory obstruction is present. Atropine will help to diminish the bronchial secretions, but suction through the tracheotomy opening may be necessary to accomplish an adequate bronchial toilet.

After the general condition of the patient has improved, endoscopic and roentgen ray examination can be carried out with more care in order to determine more fully the extent of the injury and to detect the presence of any hidden foreign bodies. If multiple injuries are present, all must be properly assessed and therapeutic priority established. With this information at hand, the wounds then can be repaired.

A general anesthetic should be administered, either by an endotracheal tube or in conjunction with the tracheotomy. Selection of the anesthetic for the patient with a serious neck injury is very important because a high oxygen content is absolutely essential. Although local anesthesia may be used for many elective surgical procedures in the neck, it is not suitable for these patients.

Following appropriate skin cleansing, the packing is removed from the depths of the wound. This is replaced by more specific pressure and each vessel is carefully ligated as fresh bleeding recurs. In knife or bullet wounds, a generous extension of the wound ordinarily will be necessary to obtain satisfactory exposure and control of all bleeding points. Bleeding from large veins may be more troublesome to control than that from arteries because vein walls are so fragile that they tear easily when handled. Ligation of a large vein requires good exposure and gentle technique. Debridement of devitalized tissue and removal of all foreign bodies are done as indicated. Clots are removed and the wound generously irrigated with saline solution.

While major veins in the neck can be interrupted without fear of central complications, every effort should be made to restore vascular continuity when the carotid artery has been injured. Ligation of the common carotid artery involves the risk of hemiplegia or fatality when it is performed under general anesthesia and in the presence of shock. Suture of lacerations of the carotid wall or end-to-end anastomosis should be performed whenever possible. When large defects are present, arterial homografts or flexible plastic substitutes have proved to be successful on a few occasions.

A complete anatomic survey then is necessary in order to identify lacerations in the viscera. Small defects in the trachea can be

sutured and covered with near by thyroid or muscle. Sometimes a two-layered closure of the pharynx can be obtained if the surrounding tissues are properly mobilized. Major defects in the larger muscles can be closed by loose suture. When viscera have been perforated, no attempt should be made to close the deep cervical fascia. A small, thin, rubber drain can be permitted to come to the outside from the visceral space but it never should be left in direct contact with the sutured wall. These maneuvers serve to decompress the retropharyngeal and visceral spaces and therefore diminish the potential of mediastinal infection. Gentle suction drainage will facilitate both wound healing and postoperative nursing care.

In the course of immediate after-care all patients with deep neck wounds should receive penicillin or other appropriate antibiotics in order to reduce the likelihood of infection. Nutrition and fluid balance can be maintained by the early and continuous use of small nasogastric tubes. The late complications of persistent orocutaneous fistula, carotid aneurysm and various arteriovenous fistulae are entities which can be avoided by appropriate early surgical care.

READING REFERENCES

- Arey, L. B. *Developmental Anatomy* 6th ed. Philadelphia, W. B. Saunders Company 1951.
- Bailey, H. *Clinical Aspects of Branchial Fistulae*. *Brit. J. Surg.* 21:173, 1933.
- Beck, A. L. *Deep Neck Infections*. *Ann. Otol., Rhin. & Laryng.* 56:439-722, 1947.
- Bill, A. H., Jr. *Cysts and Sinuses of the Neck of Thyroglossal and Branchial Origin*. *S. Clin. North America*, 36:1599, 1956.
- Buckner, F., Lyons, C., and Perkins, R. *Management of Lacerations of the Great Vessels of the Upper Thorax and Base of the Neck*. *Surg. Gynec. & Obst.* 107:135, 1958.
- Byars, L. T., and Anderson, R. *Anomalies of the First Branchial Cleft*. *Surg. Gynec. & Obst.* 93:755, 1951.
- Casberg, M. A. *The Clinical Significance of the Cervical Fascial Planes*. *S. Clin. North America* 30:1415, 1950.
- Coller, F. A., and Iglesias, L. *Infections of the Lip and Face*. *Surg. Gynec. & Obst.* 60:277, 1935.
- Davis, R. A., Wetzel, N., and Davis, L. *An Analysis of the Results of Treatment of Intracranial Vascular Lesions by Carotid Artery Ligation*. *Ann. Surg.* 143:641, 1956.
- Fogelman, M. J., and Stewart, R. O. *Penetrating Wounds of the Neck*. *Am. J. Surg.* 91:591, 1956.
- Gross, R. R. *The Surgery of Infancy and Childhood*. Philadelphia, W. B. Saunders Company 1953.
- Hollinshead, W. H. *Anatomy for Surgeons. The Head and Neck*. New York, Paul B. Hoeber Inc., 1954.
- Jahnske, E. J., Hughes, C. W., and Howard, J. M.

- The Rationale of Arterial Repair on the Battlefield. *Am. J. Surg.* 87:360, 1954.
- Lahav, F. H.: Tumors of the Neck. *J.A.M.A.* 138 264 1948
- Lenner, K. E.: Primary Tumors of the Neck. *S. Clin. North America*, p 965 1958
- Lichtenstein, M. E.: Acute Injuries Involving the Large Blood Vessels in the Neck. *Surg. Gynec. & Obst.* 85:163, 1947
- Lichtenstein, M. E.: Acute Injuries of the Neck Involving the Food and Air Passages. *Surg. Gynec. & Obst.* 85:734, 1947
- Lyall, D., and Stahl, W. M. Jr: Lateral Cervical Cysts, Sinuses and Fistulas of Congenital Origin. *Internat. Abstr. Surg.* 102 417 1956.
- Martin, H. and Morfit, H. H.: Cervical Lymph Node Metastases as the First Symptom of Cancer. *Surg. Gynec. & Obst.* 78 133, 1944
- Martin, H., Morfit, H. M. and Ehrlich, H.: The Case for Branchiogenic Cancer. *Ann. Surg.* 132 867 1950
- Mayo, C. W., and Lee, M. J.: Significance of Tumors of the Neck. *Journal Lancet* 70 420, 1950
- McGoon, D. C.: Teratomas of the Neck. *S. Clin. North America* 32 1399 1952.
- Monro, R. S.: The Natural History of Carotid Body Tumours and Their Diagnosis and Treatment. *Brit. J. Surg.* 37 445, 1950
- Morfit, H. M., Swan, H. and Taylor, E. R.: Carotid Body Tumors. *Surgery* 67 194, 1953
- Pack, G. T., Ariel, I. M. and Miller, T. R.: Malignant Ganglioneuroma of the Ganglion Nodosum of the Vagus Nerve. *Arch. Surg.* 67-645, 1933.
- Parsons, P. B.: Lateral Cervical Sinus. *Am. J. Roent. Genol.* 67 757 1953.
- Pemberton, J. J., and Livermore, G. R.: Surgical Treatment of Carotid Body Tumors. Value of Anticoagulants in Carotid Ligation. *Ann. Surg.* 133: 837 1951
- Rosenman, L. D., Brown, D. E., and Britzolar, L. G.: Ectasia of Neck Veins. *Ann. Surg.* 145:4, 1957
- Sarason, E. L., and Roberts, F. N.: Hygroma Colli of the Newborn Infant. *Surgery* 67 623, 1953.
- Sawyer, K. C., and Woodruff, R.: Cystic Hygromas of the Neck. *Arch. Surg.* 63:63, 1951
- Shapiro, H. H., Sleeper, E. L., and Guralnick, W. C.: Spread of Infection of Dental Origin. *Oral Surg.* 3 1477 1950.
- Slatrunk, W. E.: Technique of Removal of Cysts and Sinuses of the Thyroglossal Duct. *Surg. Gynec. & Obst.* 46 109 1928
- Stuterville, O. H. and Lanfranchi, R. P.: Surgical Management of Pan-neck Infections. *Quart. Bull. Northwestern University Medical School* 31 319, 1958.
- Surgical Lesions of the Neck and Upper Mediastinum symposium. *S. Clin. North America* 33:619-631 1953.
- Ward, C. E. and Hendrick, S. W.: Tumors of Head and Neck. Baltimore, Williams & Wilkins Company 1950
- Williams, A. C. and Guralnick, W. C.: Diagnosis and Treatment of Ludwig's Angina. *New England J. Med.* 228 443, 1943.

Larynx and Trachea

By F JOHNSON PUTNEY M.D.

F JOHNSON PUTNEY started and matured his professional career at Jefferson Medical College and its hospitals following his college education at Furman University in South Carolina. He based his present specialized interests upon a firm foundation of a two-year rotating internship and residency training in otolaryngology.

DISEASES OF THE LARYNX

The larynx may be examined by inspection by mirror or direct laryngoscopy by palpation or by roentgenographic or planographic study of the neck.

Injuries. Contusions resulting from falls, blows or other injuries are common. When hematoma develops absorption is the rule without incision and drainage. Subperichondrial effusion and hemorrhage into the soft tissues of the interior of the larynx may produce narrowing of the airway with dyspnea.

Pain is almost invariably present and is made worse by swallowing. Tracheotomy may become necessary.

Fractures. The larynx is protected by the lower jaw and its mobility over the cervical spine allows it to roll with a blow so that the force necessary to cause fracture must be extreme and applied after the organ is fixed. The fracture may involve either the thyroid or cricoid cartilage but rarely both, unless the force is a crushing one. Fracture of the cricoid cartilage is serious and disabling.

Healing occurs by fibrous union. Perichondritis may follow infection and increase the difficulty. Unilateral paralysis due to recurrent laryngeal nerve damage is a rare complication following cricoid fracture.

The symptoms are pain on swallowing, hoarseness and cyanosis. Soft tissue edema and redness, with or without an external wound, may make palpation of the fragments difficult but abnormal mobility and crepitus are usually present. Subcutaneous emphysema establishes the diagnosis. Roentgen-ray examination generally discloses the fracture and is helpful in determining the position of the fragments.

It may be necessary to insert a nasal feeding tube to overcome the extreme odynophagia. Tracheotomy must be promptly performed unless the patient can be kept under close observation for subsequent obstructive dyspnea from hemorrhage and edema.

Wounds. Wounds of the larynx are commonly the result of war injury or suicidal attempts by cutting or shooting. If a large neck vessel is injured, death may result from hemorrhage or asphyxiation. Treatment consists in arresting the hemorrhage, controlling infection, performing a tracheotomy if there is dyspnea and closing the wound with drainage. Injury to the laryngeal cartilages, particularly the cricoid, is commonly followed by cicatricial stenosis.

Bilateral Paralysis of the Larynx (Bilateral Recurrent Paralysis, Bilateral Abductor Paralysis) When bilateral paralysis of the muscles of the larynx occurs, both vocal cords are in the midline position of adduction.

Paralysis of both posterior cricoarytenoid muscles is commonly observed in bulbar lesions but may be caused by involvement of the recurrent nerves in carcinoma of the cervical esophagus, carcinoma of the thyroid gland and trachea, malignant disease of the mediastinal lymph nodes and by trauma to the recurrent nerves during thyroidectomy.

In addition to the causative factors, the loss of function of the abductor muscles of the vocal cords interferes with the respiratory function of the larynx.

The voice may not be changed, but the patient will speak as though breathless. This is caused by inspiratory dyspnea due to failure of the vocal cords to separate during inspiration.

By mirror laryngoscopy the vocal cords are found in adduction. On inspiration, there is no separation and only a slight drawing

downward and inward of the edges of the vocal cords. The cause of the paralysis must be determined and the paralysis differentiated from cricoarytenoid ankylosis.

The prognosis is unfavorable because sudden death from asphyxia is always imminent. If paralysis is complete there is no hope of spontaneous improvement. If partial, as in syphilis or viral infections, appropriate treatment may be beneficial.

Treatment of complete paralysis consists of tracheotomy followed by surgical restoration of an adequate laryngeal airway. Removal of the cause rarely is possible. Anastomosis of nerves in patients with paralysis due to injury has not been successful. A one-way tracheal cannula can be employed. Ventriculocordectomy has been unsatisfactory. Submucous resection of a vocal cord has been superseded by either mobilization and lateral fixation of an arytenoid cartilage or arytenoidectomy and lateral fixation of the vocal cord. These procedures are highly successful.

Infection. Infection of the larynx becomes of surgical importance when the inflammatory changes and associated edema produce stenosis and necessitate tracheotomy. Pyogenic abscess may develop in the epiglottis, arytenoids or ventricular bands and must be differentiated from an underlying foreign body or malignant disease. The abscess may rupture and discharge pus or it may have to be incised and drained by direct laryngoscopy.

Acute Inflammatory Edema of the Larynx (Edema of Glottis, Obstructive Laryngeal Dyspnea) Stenosis of the larynx may result from acute laryngitis, acute infectious diseases, acute inflammatory processes involving surrounding structures, abscess of the larynx, acute septic laryngotracheobronchitis, injuries or wounds of the larynx or neck, burns or scalds of the larynx, inhalation of irritating vapors, chondritis and perichondritis, chronic diseases or foreign bodies.

The pathologic changes are those of the underlying cause together with inflammation and edema involving the submucous tissues above and below the vocal cords.

The symptoms produced are those of obstruction to the airway with disturbances of the voice and general systemic reactions. Obstruction to the laryngeal airway is characterized by inspiratory indrawing of the soft tissues of the thoracic cage at the suprasternal notch, epigastrium and intercostal, supraclavicular and infraclavicular spaces. Restlessness, ashy gray pallor and increase

ing pulse and respiratory rates are observed. Cyanosis occurs late and is a danger signal.

The signs of obstructive laryngeal dyspnea are diagnostic. Inspection of the larynx, palpation of the neck, roentgen-ray examinations, general systemic studies and the history are usually required in order to arrive at an etiologic diagnosis.

The prognosis depends on the cause. The dyspnea can be relieved by tracheotomy.

In simple cases, inhalation of detergent and medicated vapors, the use of properly humidified oxygen and general medical measures may suffice. Sedatives must be avoided. If dyspnea is marked, tracheotomy becomes necessary. This should not be delayed until an etiologic diagnosis is made but must be promptly executed.

Noninflammatory edema of the larynx, as observed in patients with nephritis, angioneurotic edema or passive congestion due to venous obstruction, becomes of surgical importance only when tracheotomy is required to relieve the obstruction.

Chronic Stenosis of the Larynx (Cicatricial Stenosis of the Larynx) The laryngeal airway may become narrowed by cicatricial changes. The most common cause is improperly performed tracheotomy or faulty after care. Other causes may be syphilis, tuberculosis injuries, chondritis, perichondritis, foreign body and abscess.

The normal structures are replaced by cicatricial tissue with narrowing of the airway fixation of the vocal cords and often loss of a part of the normal supporting cartilaginous framework of the larynx. If tracheotomy has been improperly performed, a portion of the cricoid or thyroid cartilage may be lacking.

The chief symptom is an inability to secure any or sufficient air through the normal airway. Voice disturbances vary from hoarseness to aphonia. There is an inability to decannulate after tracheotomy.

Examination of the larynx reveals a narrowing or obliteration of the airway the extent and degree being determined by direct laryngoscopy, palpation of the neck, roentgen-ray and planographic examination. Retrograde laryngoscopy and tracheoscopy may be necessary.

The prognosis is good if there is little or no loss of cartilage, but months or years of treatment may be necessary to secure a good result.

In moderate degrees of stenosis laryngoscopic dilation may be adequate. Anterior web stenosis is readily repaired by insertion of a metal (tantalum) or plastic stent. In

marked cases, surgery must be resorted to and consists of laryngostomy removal of sufficient cicatricial tissue to re-establish the airway and the use of some form of mold to maintain the airway during the process of healing with or without the aid of a skin graft. In the open method, a trough corresponding to the stenosed airway is maintained by the use of a dilating apparatus. When a satisfactory airway has been secured, the fistula is closed by plastic operation. In the closed method, after the airway is re-established a rubber or plastic tube is transfixed in the larynx with a silver wire and the neck closed. The indwelling stent is removed perorally after a number of weeks or months when epithelization is complete.

Benign Tumors of the Larynx. The common benign tumors of the larynx are papillomas and inflammatory lesions which are not true neoplasms. These inflammatory tumors consist of polyps, postintubational granulomas, localized edematous membrane and areas of thickened epithelial change. The true tumors consist of fibromas, angiomias, lipomas, cysts and papillomas. Two types of papillomas are recognized those occurring in adults which are either single or multiple, villous-like, pedunculated or flat and those occurring in children which tend to recur after surgical treatment, are usually numerous and resemble warts.

The principal occurrence of benign tumors is in adults, more often in males than in females. These neoplasms are commonly produced by vocal abuse or submucosal hemorrhage in the presence of acute infection.

The lesions vary in color from pale to dark red, in contour from smooth to irregular and in shape from pedunculated to sessile. They are commonly found on the vocal cords but occasionally involve the ventricular bands. They generally are single but occasionally are multiple. Cysts of the aryepiglottic fold and vallecula are frequently observed in routine laryngeal examinations, but they very seldom produce symptoms.

Benign tumors of the larynx neither disappear spontaneously nor recur after surgical treatment. The chief danger consists in dyspnea when they enlarge sufficiently to obstruct the airway.

Early rapidly increasing hoarseness along with an unstable voice and easy voice fatigue are common symptoms. No symptoms are produced from the lesions involving the epiglottis, aryepiglottic folds and arytenoids unless the tumor is large when a fullness in

the throat or difficulty in swallowing develops.

Mirror laryngoscopy generally uncovers the lesion. It must be differentiated from carcinoma by histologic study.

Removal is accomplished by direct laryngoscopy or suspension laryngoscopy with suitable forceps.

Malignant Tumors of the Larynx. The majority of the malignant tumors of the larynx are squamous cell carcinomas but sarcoma, rhabdomyosarcoma, plasmacytoma and chondroma also occur. Almost all of the malignant lesions develop in the mucous membrane, but a few arise in the deeper soft tissues or in the cartilages of the larynx.

Carcinoma. In a consecutive series of 1493 cases at Jefferson Hospital the average age of the patients was 58.9 years, but the disease was occasionally encountered in individuals of both extreme age periods, the youngest patient being 18 years of age and the oldest 88. In this group the males predominated in a ratio of 13:1. A family history of cancer was obtained in 19 per cent of the patients.

In this series, 88 per cent of the patients smoked and excessive smoking was present in 52 per cent. In a certain definite percentage of the subjects keratosis preceded the development of carcinoma. Squamous cell carcinoma predominated with an occasional papillary carcinoma.

Carcinoma most often affects the anterior half of the larynx and usually originates on the vocal cords so that hoarseness occurs early and becomes persistent and progressively worse. The lesion should be suspected in any patient with hoarseness of longer than three weeks duration. In lesions involving the epiglottis and posterior portion of the larynx, the voice may be normal, but the patient complains of vague discomfort in the throat, a sensation of fullness or lump in the throat. In laryngeal carcinoma, aphonia and pain are late symptoms. When located on the vocal cord, even the smallest lesion produces hoarseness. Occasionally the appearance of enlarged cervical lymph nodes first directs attention to the upper respiratory tract and a malignant lesion of the larynx may be uncovered. In extensive lesions, dyspnea becomes marked and tracheotomy may be necessary before definitive treatment to the larynx can be undertaken.

Mirror examination reveals an uneven vocal cord with a rough nodular surface or an infiltrating lesion with fixation of the involved vocal cord. Ulcerating and proliferat-

ing lesions of the epiglottis, arytenoids, aryepiglottic folds and posterior portion of the larynx may be observed. Vocal cord cancer may extend above the surface or beneath the mucosa, spread to the opposite cord and ultimately extend outside the larynx eroding the cartilages and obstructing both the air and food passageways. Metastasis to the cervical lymph nodes is often the first indication of spread outside of the larynx. Direct laryngoscopy with removal of all or a portion of the lesion, is necessary for histologic confirmation of the clinical diagnosis.

A high portion of cures is obtained in laryngeal carcinoma as compared with malignant lesions elsewhere in the body, chiefly because of the anatomic construction of the larynx (enclosure in a cartilaginous box) and poor lymphatic drainage, both of which contribute to relatively late metastasis. Surgery yields a greater number of five-year cures than do other methods of treatment. In early malignant lesions of small extent and limited to a freely movable vocal cord, removal of the growth by laryngofissure is the prevailing operative procedure. Early diagnosis enables these lesions to be discovered while the growth can still be adequately removed and the voice box retained. In early cases, endolaryngeal removal by cauterization through suspension laryngoscopy or irradiation therapy is also successful in a large number of cases. In more advanced disease, total laryngectomy is needed and when palpable lymph nodes are present in the cervical area block dissection of the neck should always be included in one stage. In carcinoma of certain areas such as the epiglottis, subglottic region, arytenoids and aryepiglottic folds, without enlarged cervical nodes, laryngectomy combined with radical neck dissection offers the best chance of survival. When total laryngectomy is employed, the resulting loss of the laryngeal voice need not be disabling for most of these patients develop satisfactory buccal voices.

DISEASES OF THE TRACHEA

Injuries. The trachea is injured commonly following falls, blows or wounds of the neck. The tracheal rings may be torn or fractured, or the interannular membrane may be lacerated. Dyspnea is present, depending on the degree of narrowing of the airway. If vessels have been injured, there is hemorrhage. Subcutaneous emphysema with extension into the mediastinum may result

from laceration of the trachea. Pain on swallowing may be severe. The history with the local evidences of injury is usually diagnostic. Tracheoscopy may be necessary. The prognosis depends on the extent of the injury and the involvement of the surrounding structures, notably blood vessels and esophagus.

Hemorrhage should be controlled first. Tracheotomy is indicated for dyspnea and to decompress a sucking wound. Incised wounds should be repaired with drainage. Cicatricial stenosis is treated by dilation or by plastic repair through a tracheostomy performed as in laryngostomy.

Infection. Simple catarrhal infections of the trachea present no surgical problems. Chronic infections such as tuberculosis and syphilis are of surgical importance if stenosis results.

Fractures. Crushing injuries or penetrating wounds may produce collapse of the trachea, a longitudinal tear or rupture from the bronchus at the bifurcation. Pain beneath the sternum is a common accompaniment of fracture and dyspnea may become marked. Subcutaneous and mediastinal emphysema develops rapidly. Tracheotomy is necessary if a tension pneumothorax occurs or the emphysema progresses.

Tracheoesophageal Fistula. Congenital fistulae produce symptoms soon after birth when the infant begins to take liquids. Prompt recognition of the significance of symptoms followed by iodized oil instillation into the esophagus is necessary so that surgical repair can be accomplished to prevent aspiration and pneumonitis. In adults, tracheoesophageal fistula is usually a complication of carcinoma of the esophagus and the prognosis is poor.

Tumors of the Trachea. Benign tumors of the trachea are rare but may occur in any portion of the trachea, generally in the upper third or near the bifurcation. Papillomas, fibromas, lipomas, lymphomas, chondromas and osteomas have been reported. Adenoma not infrequently develops in the trachea and gives rise to obstructive dyspnea and a marked wheezing. When small or pedunculated, benign tumors can be removed bronchoscopically. Otherwise, surgical exploration with removal of the tumor and repair of the tracheal defect is necessary.

Squamous cell carcinoma is the most common malignant tumor of the trachea. Obstruction to the airway with hemoptysis and the presence of a wheeze is frequently encountered. Marked dyspnea and pneumoni-

tis develop later. Endoscopic visualization and biopsy establish the diagnosis. Unless the carcinoma is an extension downward from the larynx, the treatment is unsatisfactory. Radiotherapy has been disappointing. The best treatment is surgical excision with immediate repair of the trachea by a plastic operation utilizing a prosthetic tube until epithelization becomes complete.

TRACHEOTOMY

While tracheotomy is essential in the treatment of obstructive laryngeal lesions producing dyspnea, it is also employed to relieve secretional anoxia of the lower respiratory tract. The patient may not be able to remove accumulated secretions and aspiration through the tracheotomy tube may be lifesaving. It has become increasingly useful in sustained postoperative atelectasis, poliomyelitis, tetanus, botulism, eclampsia, neurologic depression from head injuries, comatose states and following intrathoracic surgical procedures and operations on debilitated patients.

The chief cause of complications is improper surgical technique either from placing the tube too high, causing pressure necrosis of the cricoid cartilage, or tight closure of the wound so that infection with subsequent tracheal stenosis prevents decannulation. Mediastinal emphysema is frequent but not serious unless tension pneumothorax develops when prompt decompression of the pleural cavity is necessary. When there is injury to the cricoid cartilage, laryngeal stenosis may follow. If the tracheotomy tube is changed prior to forty-eight hours and stabilization of the tract has not occurred, the cannula may be passed alongside the trachea into the mediastinum and introduce infection. After the second day the tube should be changed daily and a clean tube inserted.

The opening into the trachea is made in the midline below the first tracheal ring, preferably below the second ring. General anesthesia is contraindicated and the operation is performed under a local anesthetic or no anesthesia is employed. Tracheotomy should be performed early so that it can be carried out in an orderly manner and before there is imminent danger of asphyxiation. Delay may lead to an emergency procedure which commonly results in a poorly performed operation. The skin incision extends from the thyroid notch to the suprasternal notch (Fig. 8). Division and ligature of the thyroid isthmus are generally needed to

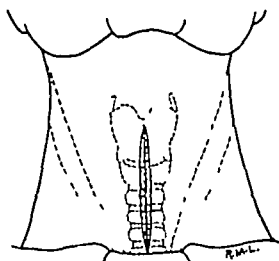


Fig. 8.

Figure 8. Vertical incision in the midline of the neck from the thyroid notch to the suprasternal notch is made for wide exposure of the trachea.

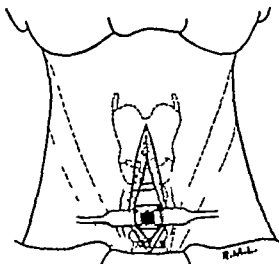


Fig. 9.

Figure 9. Segment of the second or third cartilaginous ring is removed from the trachea before insertion of tracheotomy tube.

expose the tracheal rings. A segment of the second or third tracheal ring should be removed except in young infants when the second or third ring is incised in the midline and the opening dilated to allow introduction of the tracheotomy tube (Fig 9). The tracheotomy cannula should be of proper size and shape for the individual. The wound should not be sutured but left open.

The after-care consists of keeping the air way open by wiping away coughed up secretions, cleaning the inner cannula and aspirating secretions from the trachea through the cannula. With crusting of the secretions bronchoscopy through the tracheotomy fistula may become necessary in order to keep the airway open.

FOREIGN BODIES IN THE AIR PASSAGES

The lodgment of a foreign body in the larynx or trachea is not as common an accident as lodgment in the lower air passages. The most common cause of aspiration of a foreign body is carelessness in holding objects other than food in the mouth. These are generally drawn into the respiratory passages with a deep inspiration when the patient is laughing, coughing, sneezing, singing or talking. The pathologic changes depend on the character and location of the object, the length of sojourn and whether or not there is obstruction. Recent nonobstructive foreign bodies may produce no changes other than slight inflammation at the point of lodgment. Obstructing foreign bodies will ultimately cause suppuration. The initial or immediate symptoms are chok-

ing, gagging and coughing. The subsequent symptoms depend on the size, shape and character of the foreign body and the point of lodgment. Peroral endoscopic removal is the only means of treatment worthy of consideration.

The common symptoms of a foreign body in the larynx are dyspnea and disturbances in the voice. There may be a cough and wheezing. Large, obstructive foreign bodies may produce asphyxiation.

Migratory foreign bodies in the trachea produce paroxysmal coughing. An audible slap and palpatory thud may be elicited. Dyspnea and wheezing respiration may be present. If the foreign body causes obstruction there is commonly bilateral obstructive emphysema. Bronchoscopic removal is indicated.

PERORAL ENDOSCOPY

Direct visualization of the interior of the trachea and larger bronchi is possible by the use of specially illuminated rigid hollow tubes. These are introduced through the mouth and serve as specula to draw aside the structures that hinder the view and bring inaccessible areas into view. The direct laryngoscope is a laryngeal speculum, the bronchoscope is a tracheal or bronchial speculum.

A special instrumentarium is necessary. Certain instruments are regularly used in the common procedures while other instruments are required only in special cases. The normal air passages cannot be dilated beyond their normal caliber. It is therefore neces-

nary to have special sized instruments for infants, children and adults.

Acquired skill in the use of endoscopic instruments may be secured only by educating the eyes and fingers. As this cannot be secured on the living patient, the fundamental principles are best taught on the cadaver. When skill and proficiency have been attained, experience should be secured on narcotized dogs so that the normal physiologic movements may be observed and studied.

General anesthesia is not necessary and is often undesirable, but preliminary sedation is employed. Immediately preceding operation the larynx and trachea of the adult patient are anesthetized, preferably with a 0.5 per cent solution of Pontocaine or a 4 per cent solution of cocaine, by fractional instillation. In children the use of local anesthesia is unsafe.

The patient should be in the dorsal recumbent position with the head and shoulders extending beyond the end of the table. The head is supported by the left hand of

the assistant who is seated at the patient's right. With his right hand the assistant holds the mouth gag in the left angle of the patient's mouth. The shoulders are held flat on the table by a second assistant. For laryngoscopy the operator exposes the larynx with the direct laryngoscope held in the left hand, leaving the right hand free for manipulation and introduction of instruments, or the laryngoscope is suspended or fixed in position by mechanical means. To examine the trachea or bronchi, the bronchoscope is introduced directly through the larynx into the trachea. The skilled technique of the various procedures involved requires study and training.

READING REFERENCES

- Kelly, J. D. Surgical Treatment of Bilateral Paralysis of the Abductor Muscles. *Arch. Otolaryng.* 33, 293, 1941.
King, B. T. A New and Functioning Restoring Operation for Bilateral Abductor Cord Paralysis. *J.A.M.A.* 112:814, 1939.
McNaught, R. C. Surgical Correction of Anterior Web of Larynx. *Laryngoscope* 60:264, 1950.

THE THYROID AND PARATHYROID GLANDS

By GEORGE CRILE JR. M D
and ROBERT W SCHNEIDER, M D

GEORGE CRILE, JR., a Clevelander and son of a distinguished surgeon, was educated at Yale University and the medical school of Harvard University. He has a continuing interest in thyroid disease which was extensively studied in pioneer clinical research at the Cleveland Clinic. He has a wide range of interests in natural history.

ROBERT WOODROW SCHNEIDER was born and raised in Wisconsin and received his education at the University of Wisconsin. He received his training in internal medicine at the Cleveland Clinic, became a staff member in the Department of Endocrinology and now is in practice in Salem, Oregon.

THE RELATIONSHIP BETWEEN THE STRUCTURE AND THE FUNCTION OF THE THYROID GLAND

Many of the changes in the structure of the thyroid gland are caused by changes in its function. Anything which depresses the output of thyroid hormone stimulates a compensatory mechanism in which the pituitary increases its output of thyroid-stimulating hormone (TSH). This increase in TSH causes hypertrophy, hyperplasia and hyperfunction of the thyroid. If stimulation is continued over long periods, many complicated changes in the structure of the thyroid may result.

For many years in accordance with the studies of Marine, it was believed that a deficiency of iodine was the primary factor in the production of endemic goiter. Iodine is an essential part of the thyroid hormone. Without some trace of iodine it would be

impossible for the thyroid to manufacture any hormone at all. But it appears that the role of iodine deficiency is not so important in the production of goiter as was once believed. Particularly in these days when much of our salt is iodized and when foods are shipped from the coasts to the interior the intake of iodine is probably adequate to supply the needs of the thyroid. Other factors besides deficiency of iodine appear to play a major part in the production of goiter. Among these are the goitrogens.

Substances like thiourea, thiouracil, vegetables of the rutabaga family, soybeans, cabbage, thiocyanate, sulfonamides, and other compounds which block the enzymatic system of the thyroid, depress the output of thyroid hormone. Secondly through stimulation of the pituitary this block results in hypertrophy and hyperplasia of the thyroid gland. The administration of iodine often

diminishes the hyperplasia, just as it inhibits the compensatory hyperplasia that follows removal of a large part of the thyroid gland, but it does not altogether prevent it. In short, there are factors other than deficiency of iodine which cause hypertrophy and hyperplasia of the thyroid and iodine does not necessarily give full protection against goiter induced by feeding these substances.

Heredity as well as exogenous factors plays a part in the development of goiter. There may be a strong familial tendency toward goiter and mother and daughters often are affected. Although the cause of this hereditary tendency has not been determined, it is possible that it lies in some intrinsic failure of the enzymatic system of the thyroid to produce an adequate amount of thyroid hormone. This failure results in an attempt at compensation, in the stimulation of the thyroid by the pituitary and in a prolonged state of thyroid strain. It is from this thyroid strain that many of the diseases of the thyroid may arise.

ENDEMIC GOITER

Prolonged stimulation of the thyroid gland by thyrotropic hormone results at first in hypertrophy and hyperplasia. Finally with long-continued stimulation areas of degenerating or regenerating epithelium form nodules in the thyroid. The incidence of nodules in the thyroid is extremely low in babies and children but, gradually, throughout life, it rises until in areas of endemic goiter practically everyone in the older age groups has a nodular goiter. This is one of the end results of prolonged thyroid strain.

In contrast to the multinodular endemic goiters which are the end result of a physiologic process of attempted compensation, there are true tumors of the thyroid, solitary tumors just like adenomas and carcinomas that occur elsewhere. These do not often appear to be a part of the process of multinodular change. They may be either benign or malignant. Usually they are single. These tumors are significant because they represent true neoplasia. They are much more apt to be malignant than are the involutionary nodules in a multinodular goiter.

TYPES OF HYPERTHYROIDISM

There are two types of hyperthyroidism: Graves disease and nodular goiter with hyperthyroidism. Each of these has a different cause. In Graves disease, there appears to be stimulation of the pituitary as well as

of the thyroid. The exophthalmos of hyperthyroidism is probably the result of an increased output of pituitary hormone rather than the result of the hyperthyroidism itself. Many believe that the hyperthyroidism of Graves disease is the result of stimulation by an excess of TSH, but this relationship has not been proved.

In nodular goiter with hyperthyroidism, the hyperthyroidism appears to be the result of the autonomous hyperfunctioning of thyroid nodules which developed as a part of an endemic goiter. Eye signs do not occur in patients with nodular goiters unless the nodules are an incidental finding in the hyperplastic gland of Graves disease.

The symptoms of hyperthyroidism are similar in both Graves disease and in nodular goiter with hyperthyroidism. But in Graves disease, occurring as it does about a decade earlier than nodular goiter with hyperthyroidism, there appears to be a greater restlessness, a greater brightness of the eyes, more sweating, more warmth of the skin and more tremor. In short, the signs of stimulation of the sympathetic nervous system are more obvious in Graves disease than in nodular goiter with hyperthyroidism. In nodular goiter with hyperthyroidism cardiac symptoms are apt to predominate probably because of the older age and the increased incidence of intrinsic heart disease in the patients affected.

DIAGNOSIS OF HYPERTHYROIDISM

Most of the symptoms and signs of hyperthyroidism can be explained on the basis of increased oxidation. The increased pulse rate and the increased pulse pressure are necessary because of the increased demand for oxygen and for blood to bear it. The loss of weight, in spite of a good appetite is evidence of the hypermetabolism. Increased body temperature, warmth of the hands and sweating are a part of the same overactivity.

The diagnosis of hyperthyroidism should not be made on the basis of symptoms alone. Hyperthyroidism is an organic disease. It gives objective evidence of its presence. Nervousness is much more apt to be the result of causes other than hyperthyroidism. Choking sensations and globus hystericus are rarely associated with thyroid disease.

Most of the mistakes in the diagnosis of hyperthyroidism are made because dependence is placed on the patient's symptoms, instead of upon the objective signs of increased metabolism. If a reliable laboratory

is available, the basal metabolic rate is an extraordinarily accurate index of hyperthyroidism. It is of little or no value unless done by a well trained technician under basal conditions. The determination of protein bound iodine is valuable in those patients who have not had iodine. When done accurately it is of about the same order of reliability as the basal metabolic rate. Just as the basal metabolic rate may be elevated in conditions other than hyperthyroidism, such as essential hypertension cardiac decompensation and leukemia, so may the protein bound iodine be elevated in certain of the hypofunctional hyperplasias of the thyroid, as well as when the patient has been taking iodine in large doses.

If the patient has not been taking iodine, study of the uptake of radioactive iodine by the thyroid is a valuable diagnostic test. But this test, too has its limitations. It may be elevated in nonfunctional hyperplasias when no hyperthyroidism is present.

There is no laboratory test which is 100 per cent accurate in the diagnosis of hyperthyroidism. Reliance should not be placed on the laboratory alone nor should a diagnosis of hyperthyroidism be made unless there is objective evidence of this disease.

TREATMENT OF HYPERTHYROIDISM

There are three kinds of effective treatment for hyperthyroidism the antithyroid drugs, thyroidectomy and radioactive iodine.

The antithyroid drugs are of limited value in the definitive treatment of hyperthyroidism. The recurrence rate after withdrawal of treatment is high, amounting to about 50 per cent in patients with Graves disease, and to a much higher figure in those with nodular goiter with hyperthyroidism. Since treatment with antithyroid drugs must be prolonged, requires medical supervision and is costly these drugs are gradually falling into disuse. At the present time they are being used more to prepare patients for surgery or as therapeutic trials than as definitive means of treatment.

Thyroidectomy is an effective way of controlling hyperthyroidism. In nodular goiter with hyperthyroidism, thyroidectomy is nearly ideal. Following operations for nodular goiter with hyperthyroidism, the recurrence rate of hyperthyroidism is less than 1 per cent and, unless total thyroidectomy is performed, the incidence of postoperative hypothyroidism is practically nil. In the hands of competent surgeons, the morbidity and mortality of operation are low. Unless

some better way of treating this disease develops thyroidectomy will continue to be employed as the preferred treatment for the majority of patients with nodular goiter with hyperthyroidism.

In Graves disease the situation is quite different. After thyroidectomy the rate of recurrence is high amounting to as much as 15 per cent at the end of five years if conservative subtotal thyroidectomies are done and to 3 or 4 per cent even when almost all of the gland is removed. On the opposite side of the ledger hypothyroidism or myxedema, occurs in as high as 15 or 20 per cent of the subjects if a very radical thyroidectomy is done. Only if a great deal of the thyroid tissue is left thereby running a high probability of recurrence can the incidence of myxedema be kept as low as 3 or 4 per cent. Since there is no way of predicting which patient is going to develop hypothyroidism after operation which is going to remain euthyroid and which will develop a recurrence of hyperthyroidism, it is obvious that thyroidectomy is not an ideal treatment for Graves disease.

Radioactive iodine is a quick, safe, economical and simple method of treating the hyperthyroidism of Graves disease. It is less effective in nodular goiter with hyperthyroidism because larger doses are required and because the nodular goiter does not disappear as does the diffuse goiter of Graves disease. Usually the nodular goiter gets smaller but almost always it persists. From the standpoint of time, economy and sureness of action radioactive iodine is less effective than surgery in the treatment of nodular goiter with hyperthyroidism.

In recent years, the trend has been toward using radioactive iodine in the treatment of patients with Graves disease. At first, treatment was reserved for those patients in the oldest age groups or for those with recurrent hyperthyroidism. But as experience with this method of treatment increased, the age limits were gradually lowered and now some physicians are treating even children by this means. If children are to be treated, however it is imperative that they should not be allowed to become hypothyroid after treatment. There is evidence that, in prepubertal children, radiation tends to induce cancer of the thyroid, and that the feeding of thyroid tends to prevent the development of cancer in irradiated glands. The amount of radiation that the thyroid receives is high and that which the rest of the body receives minimal, in fact, it is comparable to that received in

the course of an ordinary gastrointestinal series of x ray studies. The thyroid's ability to concentrate iodine makes it possible for the thyroid itself to receive almost all the radiation while it spares the rest of the body. The fact that the radiation is mainly in the form of beta rays which do not penetrate the tissue more than 1 or 2 mm. means that the surrounding tissues are not disturbed by the radiation. Finally since the half life of radioactive iodine is only eight days, there is no danger of prolonged radiation causing late changes.

So far no more than the normally anticipated incidence of malignant disease has been reported following the use of radioactive iodine. If at some future date malignant lesions should begin to develop in those patients who were treated ten years or more ago it would still be possible to compensate for this danger by removing the thyroids of those patients who more recently have been treated with radioactive iodine. Unless in the future some unforeseen contraindication develops radioactive iodine will replace thyroidectomy in the treatment of all patients with Graves disease. Thyroidectomy probably will remain the treatment of choice in those patients with nodular goiters and hyperthyroidism whose physical conditions are compatible with a low risk of operation.

THYROIDITIS

There are three types of thyroiditis: struma lymphomatosa (Hashimoto's thyroiditis), granulomatous (subacute) thyroiditis, and Riedel's struma.

With the diminishing incidence of nodular goiter Riedel's struma has become so rare that it has almost disappeared. For all practical purposes, it can be disregarded. It is a nonspecific invasive fibrosis of not only the thyroid but also of all the tissues of the neck. It is quite impossible to remove a Riedel's struma. Usually all that can be done is to cut into it and shell out the adenoma which commonly is encapsulated in the center of the fibrous mass. This often gives sufficient decompression to allow the patient to go on in comfort and for some unknown reason the fibrosis does not progress and, in fact, may appear to resolve somewhat after the nodule is removed.

Struma lymphomatosa is characterized by infiltration of the thyroid with lymphocytes and formation of germinal centers. The epithelium is often oxyphilic and functions poorly; varying degrees of fibrosis are present.

It appears that one of the primary defects in Hashimoto's thyroiditis is the failure in function of the thyroid epithelium. Recently Doniach and Roitt have shown that this failure may be the result of an autoimmune process directed against thyroglobulin, the thyroid cell itself or against both thyroglobulin and the cell. Failure of thyroid function probably stimulates the pituitary. Perhaps, as a result of prolonged thyroid strain, further decompensation occurs and may lead to either myxedema or to a compensatory hypertrophy of the thyroid gland. Thus, the clinical spectrum of struma lymphomatosa varies all the way from spontaneous myxedema in the adult, with a small fibrous gland which cannot even be felt, right up to the rapidly enlarging, large nodular feeling goiter which is so frequently considered to be composed of multiple adenomas and is often removed by surgeons. There is also a milder phase of this same disease, in which there are no oxyphilic changes in the epithelium, but lymphocytosis is present.

All of these lymphocytic types of thyroiditis respond specifically to the administration of desiccated thyroid. They do not require surgical removal. The diagnosis can be established by needle biopsy if necessary but it is usually obvious as the result of the enlargement and firmness of the entire thyroid gland, the tendency toward hypometabolism and the fact that in these patients the administration of thyroid-stimulating hormone fails to cause any increase in the uptake of radioactive iodine.

Subacute thyroiditis, the third distinct type, bears no relationship to the other two. It appears to be infectious in origin. Perhaps it is the result of a virus infection which destroys part of the thyroid epithelium and allows colloid to escape into the tissues where it is treated as a foreign body and causes a foreign body reaction. Histologically it is characterized by fibrosis and by a foreign-body type of reaction with many giant cells present. It is a self limited disease which eventually ends in resolution without impairment of thyroid function.

The clinical spectrum in subacute thyroiditis varies from an acute disease associated with a high temperature, severe pain on swallowing, pain radiating to the ears and exquisite tenderness in the thyroid to a very low-grade chronic process in which there is a very hard tumor which may not be tender at all. Again, the diagnosis of thyroiditis is made by being able to outline the entire lobe of the thyroid, by the fact

that the lobe retains its normal shape and because the consistency is always hard. Sometimes the thyroiditis will start in one lobe and gradually migrate to the other. Usually both lobes are involved simultaneously.

Subacute thyroiditis responds specifically to the administration of cortisone in doses of 25 mg., given four times a day. The pain is relieved at once and in a few days the thyroid itself has lost its hard consistency, is normal in size and is no longer tender. Relapses are common after withdrawal of therapy. X-ray therapy in doses of about 800 r is also of value and appears to be more permanent in its effect than is cortisone.

Except for the almost nonexistent Riedel's struma, there are no indications for operating on thyroid glands which are the seat of thyroiditis. If the surgeon asks himself each time he examines a goiter "Could this be thyroiditis?" the diagnosis can be made in the majority of cases with no difficulty.

NODULAR GOITER WITHOUT HYPERTHYROIDISM

Although goiters are not as large as they once were, nodular goiter is still a fairly common disease. In approximately 10 per cent of all adult women, small nodules can be felt in the thyroid gland. Nodules of 2 cm. or more in diameter can be felt in nearly 5 per cent of all older women. Since nodules in the thyroid are so common, removal of all nodular goiters would be impracticable and would certainly cause much more damage in the form of complications and operative deaths than would occur if most of the nodules were left alone.

A nodular goiter should be removed if the goiter is large enough to be of cosmetic importance; if the goiter is toxic; if there are symptoms of pressure in a young individual if the goiter is extending into the thorax and threatens, if it continues to enlarge, to cause trouble at a later date, and if the goiter is suspected of being malignant.

Many surgeons argue that all nodules in the thyroid are possibly cancer and that it is quite impossible to tell a cancer of the thyroid from a benign goiter. It is true that in many cases a definite diagnosis cannot be made, yet an experienced examiner can recognize between 80 and 90 per cent of all cancers of the thyroid. In many subjects, the involved lymph nodes are palpable. In many others, the consistency of the tumor, its occurrence in a young individual or its rapid

enlargement in an older individual arouses suspicion. Operation on thyroid nodules should not be conducted indiscriminately on all nodules in persons of all age groups but should be reserved for those patients in whom there are definite indications.

There is no evidence that adenomas of the thyroid commonly become malignant. In fact, all evidence is to the contrary. Most carcinomas of the thyroid are cancers from the beginning and never did originate in a benign tumor. It is true that nodules are present in the majority of thyroid glands in which cancer is present but it is also true that nodules are present in the majority of thyroid glands of patients of the age of those who develop cancer. There is no clear-cut pathologic evidence that cancer often originates in benign adenomas.

CLASSIFICATION OF CANCER OF THE THYROID

Cancer of the thyroid can be classified in two broad categories, the papillary and the nonpapillary carcinomas (Table 1).

Papillary carcinoma is a special variety of cancer. It occurs most often in patients under the age of forty years. It may not be a true cancer at all. Often it fails to grow or to cause any difficulty over periods as long as twenty or thirty years. Sometimes, even when it has metastasized, the metastatic tumor will regress if the patient is given 3 grains or more of desiccated thyroid daily. This form of treatment is probably effective through suppression of the thyrotropic hormone of the pituitary.

Microscopic cancers of the thyroid, most of them of the papillary variety, can be found in 3 per cent of all thyroids removed at autopsy when serial sections are made. In 0.5 per cent, gross tumors are present.

Table 1 Classification and Incidence of Types of Malignant Tumors of the Thyroid (182 Cases)

	PER CENT
I. Papillary carcinoma	58.8
II. Nonpapillary carcinoma	
1. Encapsulated angioinvasive carcinoma	4.4
2. Medullary carcinoma	3.9
3. Adenocarcinoma	8.3
4. Undifferentiated carcinoma	15.4
III. Sarcoma	
1. Lymphosarcoma	0.5
2. Reticulum cell sarcoma	3.3
3. Fibrosarcoma	1.1
IV. Secondary neoplasm and rare types	4.3
	100.0

In recent years the incidence of encapsulated angioinvasive cancer appears to be increasing.

Fortunately most of these tumors never grow or cause any trouble.

Many clinically recognized papillary cancers of the thyroid grow so slowly that they would not be fatal even if no treatment were given. But if papillary cancers of the thyroid are cut into and disseminated, they may implant themselves in the wound, recur locally in an invasive and unremovable manner or they may gain access to the blood stream and metastasize to lungs or bone.

Nonpapillary carcinomas of the thyroid are like other carcinomas elsewhere in the body. They occur mainly in individuals beyond the age of forty years. They vary in their degree of malignancy from the encapsulated angioinvasive carcinoma, which, except for its tendency to invade blood vessels, is indistinguishable from a benign adenoma, right up to the undifferentiated cancer which is one of the most devastating types of cancer there is. Between these two extremes lie two tumors of intermediate malignancy both of them rare. The medullary carcinoma metastasizes to the regional lymph nodes much like papillary carcinoma, but also may metastasize by the blood stream. The adenocarcinoma, also of intermediate malignancy metastasizes primarily by the blood stream. All of the nonpapillary carcinomas of the thyroid have a tendency to metastasize early by the blood stream and the prognosis in all of them is therefore poor. The prognosis is best in the angioinvasive carcinoma because this is the slowest growing of the nonpapillary malignant tumors.

TREATMENT OF CANCER OF THE THYROID

Sarcomas, highly malignant adenocarcinomas and undifferentiated carcinomas of the thyroid usually can be recognized with out difficulty. In four fifths of them, there is no history of a pre-existing goiter. The growth is rapid, the patient usually sees the surgeon within two or three months of the onset and symptoms of pressure pain, hoarseness from invasion of a recurrent nerve or signs of distant metastasis often are present. Rarely is there any question of diagnosis. Treatment almost never succeeds in arresting the disease.

Attempts to remove these cancers usually result in rapid dissemination of the tumor through the blood stream and in prompt recurrence in the wound. In the patients in whom it is suspected that the cancer is highly malignant, it is better to confirm the diagnosis by needle biopsy and treat the

cancer by x ray. Occasionally a satisfactory regression will occur. Almost never will a cure be effected.

Undifferentiated cancers do not take up enough radioactive iodine to make treatment effective. As a rule, there is no use trying treatment with radioactive iodine unless the cancer is sufficiently well differentiated to make colloid.

Adenocarcinomas of moderate malignancy and encapsulated angioinvasive carcinomas do not tend to metastasize to the regional lymph nodes, except in those advanced cases in which the capsule of the thyroid has been penetrated by the tumor. In these tumors, metastasis to regional nodes is almost always an indication of incurability for when nodes are involved, distant metastasis usually has taken place. For this reason, the treatment of adenocarcinomas and encapsulated angioinvasive carcinomas is by total removal of the affected lobe and of the isthmus. Neck dissection adds nothing to the curability of these cancers. The main thing is to remove them completely and not to break their capsules. If the tumor is cut into or if the capsule is ruptured, tumor tissue may be seeded in the incision. Although many patients with this kind of cancer may live for many years with local recurrences or distant metastases to lung or bone about 70 per cent of them eventually die of their disease. Only occasionally is x-ray therapy effective, but in those tumors which make colloid, radioactive iodine gives promise of control.

Since medullary carcinomas metastasize both to the lymph nodes and through the blood stream, even the most radical of neck dissections is rarely curative. Patients may survive five to ten years with the disease but eventually most of them die of it. Usually these tumors are resistant to radiation and they do not take up I¹³¹.

Papillary carcinomas are the commonest cancers of the thyroid and the most favorable from the standpoint of cure. They are the kind of cancer in which treatment makes the most difference in the rate of cure. In papillary cancer the outcome depends largely on the completeness with which the primary tumor and the lymph nodes to which it has metastasized are removed.

The manner in which the operation for papillary carcinoma of the thyroid should be done is a controversial issue. Some advocate use of the conventional neck dissection which is commonly applied to cancers metastatic from the oral cavity. Others believe, with good reason, that sacrifice of the stern-

ocleidomastoid muscle, which is almost never invaded by tumor is a quite unnecessary mutilation and contributes nothing to the curability of the cancer. All agree that the operation should consist of complete removal of the affected lobe and isthmus. In patients in whom there is bilateral involvement, complete thyroidectomy should be performed, the surgeon should not grasp the tumor roughly, put clamps on it or cut into it. The neck should be completely explored on the affected side especially behind the carotid vessels in the posterior triangle, in the nodes in the midline above the isthmus, in the superior mediastinum on the contralateral side and especially in the chain of nodes which lies behind the thyroid along the trachea and around the recurrent laryngeal nerve. Usually this exploration can be done and all involved lymph node groups can be removed through a high, wide thyroidec-tomy incision with division of the strap muscles. Sometimes an anterior sternocleidomastoid incision must be employed. Since most of the patients with this disease are young women in their teens or twenties, the cosmetic aspects of the operation are important.

Following conservative operations over 90 per cent of all patients with papillary carcinoma can be classified in the group of five year cures. No better results have been reported following the more radical procedures. In children under ten years of age, and in men over fifty the disease seems to be more malignant than in persons in the intervening years. Sometimes in these age groups, the cancer metastasizes to lung or to bone. When it does radioactive iodine can be tried, but, before the tumor will take up I¹³¹ all of the normal thyroid tissue must be removed or destroyed. When this is done, myxedema may ensue and, this in turn, by increasing output of thyroid-stimulating hormone may result in rapid progress of the metastases and early death. If the cancer does not take up radioactive iodine, it is important to keep the period of thyroid deficiency to a minimum, and to start full replacement therapy with desiccated thyroid as early as possible. Often, in response to suppression of TSH by feeding thyroid, the tumor will regress or at least fail to grow

USES OF DESICCATED THYROID

The development of multinodular goiter, struma lymphomatosa and perhaps of papillary carcinoma of the thyroid appears to be wholly or in part dependent upon the thy-

roid stimulating hormone of the pituitary. If large doses of desiccated thyroid are given, the thyroid stimulating hormone is suppressed, the lymphoid types of thyroiditis shrink or disappear, thyroid nodules often get smaller and some papillary cancers of the thyroid regress.

Thyroid feeding is tolerated well. In doses up to 3 grains daily it does not cause a permanent increase in metabolism and it gives no symptoms. All that happens is that the thyroid stops making thyroid hormone. The gland is put at rest.

If thyroid strain causes many of the pathologic changes of the thyroid gland, there is no reason why these changes should not be treated by giving desiccated thyroid and relieving the strain. Many small goiters can be treated effectively in this way. The danger of cancer of the thyroid in these goiters is slight. If thyroid strain is relieved, the chances of preventing the development of cancer are good. In the future the thyroid will challenge the surgeon as a problem in endocrine physiology much more often than as a subject for surgical technique.

HYPERPARATHYROIDISM

Hyperparathyroidism is a disorder characterized by an overproduction of parathyroid hormone. It may be either primary or secondary in type. The term "secondary hyperparathyroidism" is reserved for those unusual clinical disorders associated with an increased demand for parathyroid hormone. Its treatment is not surgical but rather requires a recognition of the underlying cause for the increased demand.

Primary hyperparathyroidism is a surgically curable disease. The increased production of parathyroid hormone arises either from an adenoma in one or more of the parathyroid glands, from diffuse hyper-trophy or hyperplasia of all the parathyroid glands or from carcinoma of one or more glands. The majority of cases are produced by a single adenoma located in one of the two lower parathyroids, only 16 per cent arising in the upper two glands. Multiple parathyroid adenomas occur in about 6 per cent of the subjects. Aberrant tumors occur in about 10 per cent. Among these the mediastinum is the most frequent site (63 per cent); other aberrant tumors may be located behind the esophagus or trachea or within the thyroid gland.

The parathyroid glands play an important role in the regulation of calcium and phosphorus metabolism. One of the principal

actions of parathyroid hormone is the regulation of phosphorus excretion in the urine. Following the administration of parathyroid hormone, prompt increase in phosphate excretion occurs and is of sufficient intensity to lower the serum phosphorus concentration. Continued administration is followed by a rise in serum calcium to levels above normal, with an eventual increased excretion of calcium in the urine. Prolonged parathyroid hormone intoxication, as seen in primary hyperparathyroidism, is thus characterized by hyperphosphaturia, hypophosphatemia, hypercalcemia and hypercalciuria. It is important to realize that the calcium fraction which is altered in the blood by parathyroid hormone is the ionized fraction and not the calcium attached to protein and largely to albumin. In making a critical evaluation of serum calcium levels an estimate of serum proteins is therefore necessary.

The alterations in calcium and phosphorus metabolism enumerated may have profound effects. The high levels of serum calcium may lead to impairment in renal function by the production either of renal calculi or of nephrocalcinosis. An increased excretion of water to aid in the elimination of the excessive calcium load leads to polyuria and polydipsia, which at times are sufficiently intense to simulate diabetes insipidus. The urine excreted is often of low pH and low specific gravity. The hypercalciuria leads to a negative calcium balance, which in turn leads to skeletal decalcification, but it is minimized in those individuals who are milk drinkers.

The symptoms and signs in hyperparathyroidism are variable but may be divided into three categories: those due to associated bone disease, those due to associated urinary tract disease and those due to the high serum calcium level.

Bone disease occurs in only about 35 per cent of all patients. The bone changes consist of generalized decalcification with superimposed tumors and cysts. These changes are called *osteitis fibrosa cystica generalisata* of von Recklinghausen. The bone tumors consist of solid masses of soft tissue without bone composed of osteoblasts and osteoclasts, and the supporting cells of the bone marrow. The cysts are filled with fluid and lined with fibrous tissue.

The early symptoms of the bone disease vary from indefinite pain to bone tenderness which often simulates arthritis or neuritis. Bowing of the long bones deformities of

the pelvis, decrease in stature from deformed vertebrae and multiple fractures can occur. The bone tumors are often diagnosed as benign giant cell tumors and occasionally are incorrectly regarded as malignant. Areas of predilection for such tumors are the jaws, metacarpals, metatarsals and the ends of long bones.

Polyuria and polydipsia commonly accompany the hypercalciuria and the phosphaturia. Renal calculi occur with far greater frequency than does bone disease and either renal calculi or reduced kidney function is present in about 75 per cent of all subjects of hyperparathyroidism. The kidney stones are composed of either calcium phosphate or if superimposed infection is present, a mixture of magnesium ammonium phosphate and calcium phosphate.

A serious renal complication is nephrocalcinosis, a deposition of calcium within the collecting tubules of the kidneys. In this condition, the renal tubules become plugged with calcium casts. When the condition is advanced, some degree of urea retention may occur and both the low serum phosphorus and the high urine calcium characteristic of hyperparathyroidism are obscured by the renal failure.

Hypercalcemia can be associated with other symptoms besides those already mentioned. Anorexia, nausea and vomiting may dominate the clinical picture sufficiently to simulate the symptoms of duodenal ulcer. Calcium deposition may occur in the deep conjunctiva of the palpebral fissure and may be seen under the slit lamp. Marked hypotonicity of skeletal and visceral muscles may occur with profound generalized muscular weakness, hypotonicity, absence of deep tendon reflexes and intractable constipation. The electrocardiogram may show shortening of the Q-T interval. The low serum phosphorus levels of hyperparathyroidism do not produce symptoms. They are however the most important single finding of diagnostic importance. The diagnosis of primary hyperparathyroidism offers considerable difficulty because attention usually is attracted by the complications produced by the disease rather than by the true underlying cause. The diagnosis depends chiefly upon indirect evidence and upon the physician's index of suspicion. Integration among history and clinical, laboratory and x-ray findings is necessary for final diagnosis.

The hypercalciuria can be confirmed by the quantitative determination of calcium excretion in the urine on a twenty-four hour

basis and after three days on a low calcium (Aub) diet. Calcium loss per twenty four hours in excess of 200 mg. is indicative of hypercalciuria.

The alkaline phosphatase value is of no aid in the diagnosis of hyperparathyroidism and is never elevated except in those instances where the skeleton is involved. It is a good index of the degree of skeletal involvement. Patients with an elevated alkaline phosphatase value and extensive bone disease are particularly prone to develop postoperative tetany which may require intensive medical therapy until the bones are healed. The treatment of such tetany is identical to the treatment of postoperative tetany following thyroidectomy and consists of the oral administration of calcium lactate powder 3 to 10 drachms per day, and at times the addition of vitamin D 50 to 300 000 units per day or Hytakerol 5 to 30 drops per day. Parathormone 1 cc. once or twice daily may be given for acute attacks but antihormones form quickly and limit its usefulness. The use of intravenous calcium is more practicable and it can be given as a 10 per cent solution of calcium gluconate in doses of 10 cc., repeated as often as is necessary for symptomatic relief.

X-ray study may be of value in contributing to the diagnosis of hyperparathyroidism by the demonstration either of kidney stones or one of the bone defects previously mentioned. One additional x-ray finding consists of the demonstration of the disappearance of the lamina dura (peridental membrane) around the teeth. When the lamina dura is decalcified wholly or in part, it is highly indicative of hyperparathyroidism.

The diagnosis of hyperparathyroidism depends upon indirect evidence obtained from a variety of sources. It is to be remembered that mild degrees of hyperparathyroidism

may be most difficult to recognize and require periodic observation. The association of the symptoms, the physical signs and the laboratory and x ray findings leads to a high degree of accuracy in clinical diagnosis. Occasional cases are encountered in which doubt exists and a tumor can be proved to be present or absent only by appropriate surgical exploration. Above all, it is important to remember that it is possible for serious hyperparathyroidism to exist in the presence of normal total serum calcium levels.

When a parathyroid tumor is found, it is imperative that it be removed completely with the capsule intact. Patients have been reported in whom these tumors have been ruptured during their removal, have implanted themselves in the wound and caused fatal recurrences or metastases.

READING REFERENCES

- Dobyns, B. M., and Steelman, S. L.: The Thyroid-Stimulating Hormone of the Anterior Pituitary as Distinct from the Exophthalmos Producing Substance. *Endocrinology* 52:705, 1953.
- Doniach, D., and Roitt, I. M.: Auto-immunity in Hashimoto's Disease and its Implications. *J. Clin. Endocrinol.* 17:1293, 1957.
- Gribetz, D., Talbot, N. B., and Crawford, J. D.: Goiter Due to Lymphocytic Thyroiditis (Hashimoto's Struma) Its Occurrence in Preadolescent and Adolescent Girls. *New England J. Med.* 250:555 1954.
- Marine, D.: Etiology and Prevention of Simple Goiter. *Medicine* 3:453, 479 1924.
- Mortensen, J. D., Bennett, W. A., and Woolner, L. B.: Incidence of Carcinoma in Thyroid Glands Removed at 1000 Consecutive Routine Necropsies. *S. Forum* (1954) 5:659 1955.
- Rienhoff, W. F., Jr.: New Conception of Some Marked Changes in Diseases of Thyroid Gland. *Tr. Am. A. Study Goiter* pp. 187-189, 1930.
- Skiffen, P. G., and others: Struma Lymphomatosa. Primary Thyroid Failure with Compensatory Thyroid Enlargement. *J. Clin. Endocrinol.* 16:35 1956.

THE BREASTS

By IAN MACDONALD M.D.

IAN MACDONALD is a Canadian by birth, was educated at McGill University and has had an interest in the pathology of tumors, particularly cancer throughout his medical career. He has the quality of combining an exacting biostatistical and scientific attitude with the surgeon's desire for utilizing every possible therapeutic approach to the cure of cancer.

INTRODUCTION

Although the human mammary gland is subject to a variety of pathologic processes there is no other anatomic site of comparable importance in which the problems of hyperplasia and neoplasia outrank all others to an almost exclusive degree. This statement acquires justification from the prognostic implications apparent in comparing all other mammary disorders with those of neoplastic nature, for only in this latter group under modern circumstances is the patient's life in jeopardy.

Numerically too the lesions representing states of abnormal hyperplasia and neoplasia are of overwhelming importance for almost 85 of every 100 mammary lesions will be one of the diagnostic "big three."

1. Some phase of the heterogeneous manifestations of abnormality in the hyperplasia involution cycle or so-called *cystic disease*.
2. The most common by far of the benign neoplasms of the breast, *fibroadenoma*.
3. The one group of lesions with lethal potential, generically designated as *cancer*. This term includes malignant neoplasms of epithelial origin, or *carcinoma* as well as unusual instances of *sarcoma*.

The malignant and benign neoplasms both have as their only reliable and earliest

symptom and sign the presence of a three-dimensional or space-occupying tumor in the breast. Unfortunately some non-neoplastic lesions of the breast also produce space-occupying lumps or tumors, in the general sense of the word. On the other hand, a majority of patients with "cystic" disease of the breast present local indurative changes of varying degree and extent, but separable by competent physical examination from those due to genuine or space-occupying tumors.

Thus, the most important single problem to be determined in the woman whose complaint is related to the breast, or even in asymptomatic patients on physical examination, becomes obvious. Is there discoverable a genuine space-occupying tumor? If so the physician must take steps toward securing an exact, which is to say histologic, diagnosis. For this dictum there is but one reason only by the histologic method is it possible to determine whether the true lump in the breast is neoplastic and whether benign or malignant in nature.

No other maneuvers ancillary to careful physical examination are reliable. Thus the entire responsibility of decision for or against diagnostic action must be based on the physician's ability to develop a *tactus*

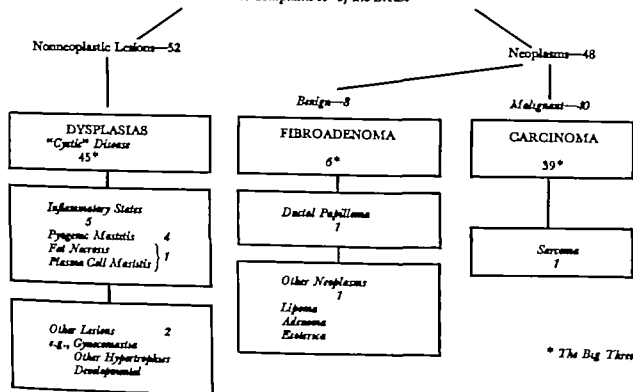
cruditus in examination of the breasts. Development of skill in tactile examination of the breasts should be regarded as an absolute requirement for every clinician who undertakes responsibility in this respect. Failure to achieve such skill and failure to perform a meticulous orderly examination are responsible for the lack of more effective or even lifesaving treatment at a favorable stage of the disease in some patients with cancer of the breast. On the other hand, the inability of the clinician properly to evaluate changes in mammary texture and consistency leads to many unnecessary exploratory procedures for biopsy of areas lacking any evidence of neoplastic change.

Efforts of the American Cancer Society in public education are alerting many more thousands of women each year to the value of periodic examination for the early detection of "a lump or thickening" in the breast. Recommended are self-examination at monthly intervals by a uniform method and examination by a physician at twice-yearly intervals after the woman has reached the age of 35 years. As would be expected, most of the "lumps" detected by self-examination are not three-dimensional tumors the majority represent changes in consistency associated with "cystic" disease areas of premenstrual hyperplasia, cutaneous axillary

structures underlying rib or cartilage or the normal axillary prolongation of the mammary gland. Only one of approximately 1000 adult women doing self-examination regularly for one year will discover a true lump which proves to be a cancer. In forty years a total of forty cancers per thousand would have been discovered.

Balanced against this small yield of malignant tumors, presumably discovered when smaller in size than if accidentally found, is the hazard of unnecessary surgical procedures by inexperienced clinicians. It becomes an urgent necessity for physicians to resist the anxiety engendered by the self-discovered "tumor" and, if the criteria for biopsy are not present, to guide the program of self-examination by careful supervision observation and reassurance. The extremes of conciliation by incision and alienation by indifference are a disservice to the patient and a reflection on the profession. Even though the net improvement in curability achieved by periodic examination may be small in the face of the natural history of cancer of the breast, every physician has an obligation to contribute as best he can to the total effort. The potential impact is that of effective treatment at an earlier stage of the disease in more patients which can be expressed in terms of clinical control for five

Table 1 Approximate Incidence of Lesions of Breast for Each 100 Patients Whose Complaint Is "of the Breast"



* The Big Three

to ten years for several additional thousands of women annually in the United States alone.

In any unselected group of women whose presenting complaint is of the breast, a distinct majority will be found to have some phase of "cystic disease, cancer being next most common, followed by fibroadenoma. The small remainder not included in the "big three" constitute the unusual and esoteric mammary lesions (Table 1). The approximate distribution of lesions in one individual's practice is

	TOTAL	PER CENT
"Cystic" disease	609	45
Cancer	537	40
Fibroadenoma	88	7
All other lesions	111	8

About one-third of patients with "cystic" disease will have developed some phase of the process resulting in a three-dimensional tumor (gross cysts, sclerosing reaction or persistent focal hyperplasia). Some of the "other lesions" are also space-occupying lesions. Thus, about six out of ten women with complaints referable to the breast, at least in a specialized type of practice have presented true, mensurable tumors requiring surgical, diagnostic action. Of the patients requiring such action, not more than 50 per cent should prove to have "cystic" disease. When the diagnosis of cystic disease in biopsy tissue exceeds 60 per cent of all biopsies of the breast, such excess indicates the inability of the clinician to differentiate between true and false lumps and is a reliable measure of the frequency of unnecessary surgical explorations in the experience of the individual surgeon or of the institution.

This introductory emphasis on the problems of clinical and surgical pathologic diagnosis is an essential preface for each individual patient should be considered in relation to established diagnostic probabilities. The problems involved are of paramount importance, for the breast continues to be the most common organ site of cancer in the adult female population of the United States. Efficient clinical performance is in direct ratio to the relative number of biopsy tissues diagnosed as lesions other than "cystic" disease.

To achieve reasonable accuracy in the selection of mammary lesions requiring biopsy the clinician should have a knowledge of anatomic variations physiologic (endocrine) factors, the variegated dys-

plasias ("cystic" disease) and the natural history of certain hyperplastic states.

ANATOMY

In the female, each mammary gland rests upon the fascia of the pectoralis major muscle. The peripheral limits of the gland are variable from one patient to another but ordinarily breast tissue extends almost to the midline over the sternum, to the anterior axillary line laterally and from the level of the second rib above to about the sixth rib inferiorly in a horizontal line drawn through the nipple. The greatest variation in peripheral extension occurs in the axillary area. Most often there is a blunted, narrow extension of the gland which may still lie on the fascia over the converging fibers of the pectoralis major while in other instances this so-called axillary tail of the breast is found below or posterior to the pectoralis major muscle in the lower third of the axilla. In unusual instances the axillary tail may be a prolongation extending into the middle third of the axilla and lying in contact with the pectoralis minor muscle or the intercostal muscles. In such abnormal axillary extensions the indurative changes of "cystic" disease may occur as well as actual neoplasms, and in such instances their origin in mammary tissue may be unrecognized. Still more unusual are the instances of supernumerary foci of mammary glandular tissue most often without corresponding nipples which may occur in the axillary area as well as in the anterior axillary line and also below and medial to the normal breast.

Supernumerary foci of mammary tissue may exhibit the cyclic changes which characterize the physiologic response of the breasts to hormonal stimulation.

The superficial fascia of the breasts is attached to the skin, a fact of extraordinary clinical importance. A layer of fat surrounds the gland except in the area of the nipple and areola. The thickness of the subcutaneous fat is highly variable. In thin women with underdeveloped breasts, the subcutaneous fat layer may be almost nonexistent so that the reflection of skin from the breast during radical mastectomy requires a meticulous peripheral dissection of extremely thin skin flaps. With increasing obesity or with fatty replacement of mammary parenchyma, the thickness of the subcutaneous fatty layer correspondingly increases. The anterior surface of the gland is irregular owing to the lobulations formed by the deep

attachment of fibrous septa, the eponymic designation of which is Cooper's ligaments, which run between the superficial and deep fascia.

The mammary gland is made up of ten or twelve to twenty glandular lobes drained by an equal number of tortuous ducts which dilate close to the nipple to form ampullae, and finally divide into minute ducts ending in small openings in the nipples. Each of the orifices in the nipple corresponds roughly to a truncated segment of breast which it drains, a fact of importance in the investigation of bleeding or other discharge from the nipple.

The parenchyma of the breast is composed of two types of epithelium, acinar or secretory and ductal. Both have a double fibrous covering, the inner periductal or periacinar layer and an outer layer of perilobular connective tissue. The acinar components of the breast are highly variable in quantity during puberty, the reproductive years and even after the climacteric, conditioned upon response to hormonal stimulation. The ductal components are less variable in quantity after maturation except in the instance of pregnancy and lactation. The acinar epithelium is cuboidal in shape while the epithelium of the ducts is columnar.

In men and in prepubertal females, the gland is rudimentary with only a few short ducts and ordinarily without genuinely developed acini.

Cutaneous Lymphatics. There is a continuous intercommunicating network of lymphatics over the entire surface of the chest, neck and abdomen. By this mechanism, the subcutaneous lymphatics over one breast

communicate with those of the opposite gland. There are even some lymphatics originating under the skin which drain into the contralateral axillary nodes. For each breast there is a collecting network of lymphatics under and adjacent to the areola.

Intramammary Lymphatics. The lymphatics originating within the breast drain by the following pathways (Fig. 1)

1. A few will follow the ducts and terminate in the subareolar lymphatic network mentioned above, mainly from the central area.

2. The principal route is the axillary pathway coming from all parts of the gland and draining directly to the central axillary nodes, the nodes around the axillary vein or the subscapular group. There is also drainage to intercostal nodes in the second and third intercostal spaces.

3. A transpectoral pathway penetrating the pectoralis major muscle and ending in the supraclavicular nodes, although some may drain into infraclavicular nodes behind the pectoralis minor muscle.

4. The internal mammary pathway which passes through the pectoralis major and intercostal muscles adjacent to the sternum and drains into the nodes of the internal mammary chain, lying deep to the costal cartilages and surrounding the internal mammary blood vessels.

PHYSIOLOGY

The hormonal control of the parenchyma of the breast has become well established, both by experimental studies in animals and biopsies from human mammary glands during various phases of the menstrual cycle and during pregnancy and lactation. The estrogenic hormones during the first half of each menstrual cycle produce ductal hyperplasia, and at the same time sensitize the acinar epithelium for the action of progesterone during the second half of the cycle, the latter hormone producing acinar hyperplasia. In the absence of preliminary sensitization of the acinar components by estrogens, there is very little hyperplastic response from the administration of progesterone alone, as shown in oophorectomized animals.

These cyclic changes of ductal and acinar hyperplasia, with some hypertrophy occur in irregular focal fashion, although the degree and extent of response each month is highly variable from one woman to another. With the onset of menstruation, there normally occur involutionary changes in the

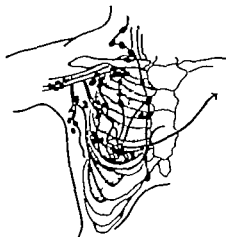


Figure 1 Lymphatics of the breast: 1, subareolar; 2, axillary; 3, transpectoral; 4, internal mammary

areas which have been so stimulated to hyperplasia, only to be succeeded promptly by the same cycle of hyperplasia of ducts and then of acini in other segments of the breast. If these repetitive cycles of response in the parenchymal (target) tissue are unattended by abnormalities throughout the reproductive years there is little in the way of symptomatology except a feeling of premenstrual fullness or at the most, a slight soreness usually limited to small areas in the breast. Distinct abnormalities in this hyperplasia involution cycle are interpreted as the basic mechanism in the production of so-called cystic disease.

During pregnancy the hypertrophic and hyperplastic changes are the same as those during a menstrual cycle except on a grandiose scale. During the first four to five months of pregnancy the changes are largely in the ductal system, while in the latter part of pregnancy particularly in the last three months, the dominant response is that of tremendous acinar overgrowth. The final phase is that of secretory response by the acinar epithelium, with prelacteal exudate draining into the ducts and constituting the material known as colostrum. After delivery and principally under the influence of a hormone from the anterior pituitary prolactin the acinar components rapidly reach a peak of secretory activity and distention to produce the fully lactating breast.

The estrogenic steroids and progesterone concerned in the cyclic changes are of course elaborated by the ovaries, under the control of the corresponding gonadotropic hormones of the anterior pituitary. Following the climacteric, there is evidence that in some women the adrenal cortices take over the function of production of estrogenic steroids and, in occasional instances, such levels of estrogen production may approach those of the woman with an active follicular luteal mechanism. This evidence seems to account satisfactorily for the occasional postmenopausal woman who continues to have symptoms of mammary congestion and soreness and objective findings similar to those patients who suffer from abnormalities of the hyperplasia involution cycle in earlier years.

With these unusual exceptions the parenchyma of the breast undergoes a slow progressive involution over a period of five or more years following the onset of the climacteric, with atrophy and replacement of parenchyma by fatty infiltration.

The preceding description of interplay of certain steroid hormones on the mammary epithelium is of course a working skeletonization of existing knowledge and is dependent upon a considerably more complex endocrine background involving the pituitary-thyroid-adrenal axis. Some experimental work, for example indicates that in hypothyroidism estrogenic stimulation of the breast produces a cystic type of hyperplasia. In some species of animals after castration, stimulation with estrogen alone will produce considerable acinar hyperplasia which is also of cystic type and in which frequent papillomatous epithelial lesions of hyperplasia occur while with the concomitant administration of progesterone such cystic and papillary changes do not occur. Although experimental, these are findings which one may relate by inference to some of the abnormalities of hyperplasia involution in the human being.

EXAMINATION OF THE BREASTS

It is of primary importance that a woman whose complaints are related to the breast should have a general physical examination, unless she has had one performed by a competent physician in the preceding three months. Pelvic examination is essential because of the not infrequent association of gynecologic pathologic states with breast lesions particularly with "cystic" disease. In functional disturbances of the breast, stigmata of endocrine dysplasia should be searched for such as hirsutism, abnormal deposits of fat and uterine hypoplasia, as well as indications of hypothyroidism. Should the patient have a three-dimensional tumor which may be carcinoma, methodical examination of such accessible areas as may harbor metastatic foci is essential. If a general physical examination is not to be done, the patient should at least be undressed to the waist and draped with a sheet or a gown.

Preliminary examination should be done with the patient sitting comfortably but erect on the end of the examining table, with the hands resting in the lap and the legs uncrossed. With the patient in this position, the breasts should be inspected for equality of size and similarity of contour for disparity in the appearance of the nipples and variation in level of the nipples. It should be obvious that this must be accomplished in a good light.

Approximately one-fourth of women have some minor degree of inequality in the size

of the breasts of developmental nature while in a very occasional woman there may be an obvious unilateral hypertrophy usually unrelated to pregnancy or lactation. It should also be noted that in women who have any degree of scoliosis there will be a distinct lack of symmetry in the breasts, the breast on the side of the convexity of the scoliosis being higher than the opposite breast and the level of the nipples correspondingly altered (Fig. 2)

The color and the consistency of the skin over the breasts should be carefully noted, particularly for areas of erythema or edema. Retraction of the skin due to carcinoma will rarely be noted in this position, except in the instance of an advanced lesion. In fact, distinct retraction of the skin demonstrable with any method of examination is usually an indication of lymphatic permeation and a relatively advanced lesion.

The patient should now be asked to bend forward and thus note whether in a dependent position there is lack of mobility of either breast, alteration in the relative nipple position or evidence of skin retraction in any area.

With the patient sitting straight again, and the arms still adducted and without tension on the pectoral fascia, the axillary folds, the supraclavicular and cervical areas and shoulders should be inspected for any evidence of asymmetry.

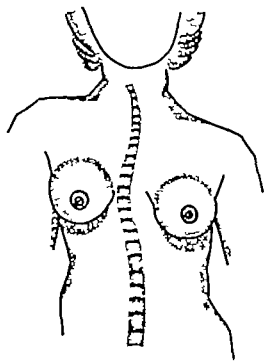


Figure 2. Normal asymmetrical breasts due to scoliosis.

By gentle palpation the neck on both sides and particularly the supraclavicular areas should be searched for abnormal nodes. The thyroid should be palpated for abnormal enlargement or displacement. Next, the infraclavicular area should be palpated for possible nodes.

The axillary area on each side should be palpated for lymphadenopathy, the patient's forearm resting on the opposite forearm of the examiner in such a fashion that the patient's upper extremity is entirely relaxed. With the flat of the fingers and beginning at the axillary apex, the axilla is gently palpated. Special attention should be given to the so-called retropectoral area, where nodes lying behind the converging fibers of the pectoralis major muscle may be palpated.

Palpation of the breast itself with the patient in the sitting position yields much less information than that accomplished with the patient in the supine position, except in the central third of the breast. In the latter area, the nipple and areola are carefully examined for indurative changes. With one hand supporting the breast from below and the other palpating gently from above, abnormality in the ductal system adjacent to the areola may often be detected. The surface of the nipple and areola should be carefully examined for evidences of scaling or erosion, even though of minute degree.

If there seems to be any disparity in the size of the arms, the circumference of each arm should be measured at the same distance from the acromial process on each side, the 5-inch level being practicable.

Still in the sitting position, the patient should now be asked to abduct the arms to a 90-degree angle. The inspection is again made for inequalities in the level of the nipples and for possible areas of skin retraction which may now be apparent with the pectoralis fascia under tension, particularly in lesions located in the upper outer quadrant of the breast.

With the patient in the supine position, each breast is methodically palpated, first with the arm in adduction and then in sharp abduction. In some women, particularly those with bulky breasts this part of the examination may be facilitated by placing a pillow under the scapular area of the side being examined, particularly to obtain better palpation of the lateral portion and axillary tail of the breast. All of this examination should be by the flat hand, remembering that the maximum tactile sensitivity is located in the distal two-thirds of the flexor

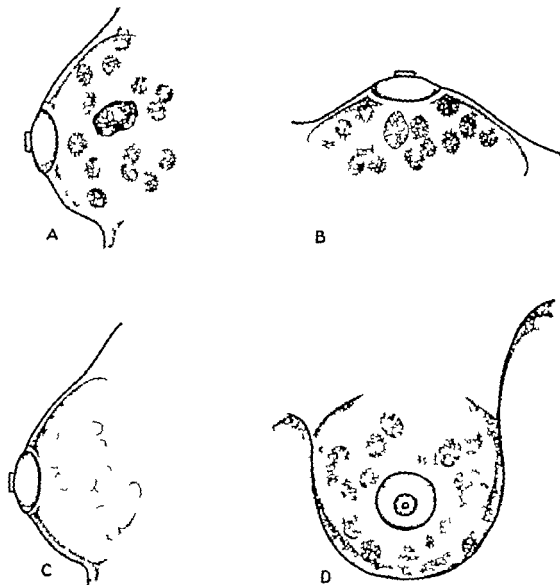


Figure 3. The lesions of both A and B may resemble lumps by finger palpation with the patient in the sitting position. By flat hand palpation with the patient in the supine position, the more advanced lesion in A is detected as a true lump that in B a pseudolump. C, Soft, normal breast lobules not palpable. D Few days premenstrual breast is fuller more dense vague outline of lobules.

surfaces of the fingers. Finger point examination has no place in this examination, for it will frequently produce the illusion of a three-dimensional tumor when none is actually present (Fig. 3 A and B). In order to accomplish systematic palpation of the entire breast, one should begin at some arbitrary axis and, by flat hand palpation around the circumference of the breast, determine variations in consistency and search for space-occupying lumps. The area of the axillary tail of the breast should be examined separately and special attention given to the inframammary fold with upward traction on the breast during examination of the latter area.

As experience is gained, one will be able

to differentiate between varying degrees of consistency in various types of normal breasts. In Figure 3 C, for example, is illustrated a soft normal breast in the first half of the menstrual cycle with the lobules not palpable. In D during the few days premenstrual, the breast is fuller more dense and careful palpation may suggest the vague outline of lobules throughout the breast. Illustrated in Figure 4 E is another type of normal breast with considerably different consistency in which, during the early part of the cycle, the lobules are distinctly palpable in the peripheral two-thirds of the breast, while in the central third vaguely outlined radiating areas may suggest the ductal structure. In such a breast,

the palpable lobular and ductal architecture will have become considerably exaggerated in the premenstrual phase (Fig 4 F). The lobules are now palpable to a more distinct degree and the radiating ductal system may also be more apparent in the form of linear irregular or even slightly beadlike areas. In this sort of breast it is possible by finger point palpation and even more by palpation between two sets of opposing fingers, to obtain the sensation of a tumor in one of these well-developed lobular areas. When such an area is gently palpated with the flat of the fingers, the pseudotumor disappears, blending into the adjacent breast tissues.

In these schematic representations, there is implied a uniformity of palpable lobules and ducts along with their premenstrual exaggeration. In fact, the distribution of such palpable normal lobules either in the resting or hyperplastic stage, usually is extremely irregular.

Represented in Figure 5 are focal areas of persistent abnormal acinar hyperplasia, one represented in the right breast (G) where the lobules are diffusely palpable, and in the left (H) in a soft breast with impalpable lobules. It is possible to interpret these lesions as a lump by fingertip palpation, particularly with the patient in the sitting position. On flat hand palpation with the patient in the supine position, they should be apparent only as abnormally indurated areas without the circumscription or substance of a space-occupying tumor.

More advanced abnormalities of the hyperplasia-involution cycle are portrayed in the lower part of Figure 5. Represented in the left breast (J) are an area of abnormal hyperplasia now large enough in extent to have become a three-dimensional, vaguely circumscribed tumor, another area just above the areola which may also be a three-dimensional lump while the lesser areas indicate indurative changes without the substance of a genuine lump. In the right breast (I) are represented various degrees of the actual cystic phase of "cystic" disease with two gross cysts in the upper hemisphere constituting three-dimensional tumors, while in the lower hemisphere are two small cysts which would constitute distinct nodules only if there was sufficient tension of contained fluid within the cyst. The most medial nodule represents a combination of the cystic and hyperplastic phases of "cystic" disease.

If a three-dimensional tumor has been

satisfactorily outlined its location should be indicated accurately on a diagrammatic sketch on the patient's record. This may be done either on a free-hand sketch of the breast and adjacent axillary and supraclavicular areas or a stamped outline. The dimensions of the tumor should be determined as accurately as possible by measurement with a ruler and written on the diagram. Also shown should be the location and approximate size of enlarged regional lymph nodes, if found or the absence of lymphadenopathy should be indicated. The sketching in of the tumor can also indicate whether it is round or ovoid or irregular in outline.

If possible, one should determine whether the surface of the tumor seems smooth or irregular usually possible in a patient with small breasts or in tumors superficially located. The degree of mobility or its impairment in relation to adjacent breast tissue, deserves description. If the tumor is deeply located or bulky in size the presence or absence of attachment to the pectoral fascia or to the chest wall should be determined and noted. Such a combination of schematic representation and brief description of essential features is informative and accurate, saves time and space and is of particular importance in those patients requiring subsequent observation of questionable lesions.

In the diagnosis of small carcinomas of the breast, significant changes in the over

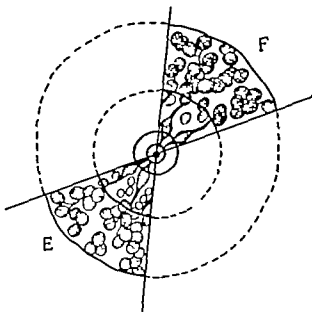


Figure 4. E, Early part of cycle. Lobules palpable in peripheral two-thirds, vaguely outlined in central one-third. Radiating areas may suggest ductal structure. F, Premenstrual phase. Lobules palpable to more distinct degree. Radiating ductal structure more apparent.

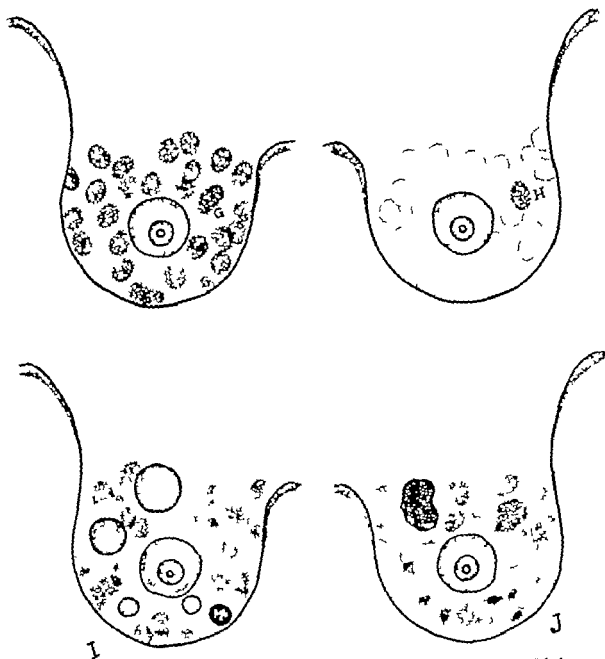


Figure 5. Advanced abnormalities of the hyperplasia-involution cycle. G. Right breast lobules are diffusely palpable. H. Left breast soft with impalpable lobules. I. Right breast. Two gross cysts in upper hemisphere constituting three-dimensional tumors. Lower hemisphere. Two small cysts which would constitute distinct nodules only if there is sufficient tension of fluid within. The most medial nodule represents combination of cystic and hyperplastic phases. Left breast: An area of abnormal hyperplasia now large enough to be a three-dimensional, vaguely circumscribed tumor. Another area above areola may also be a three-dimensional tumor. Lesser areas indicate indurative changes without substance of genuine lump.

lying skin usually are so minimal as to be almost subtle. These include an evident impairment of elasticity of the skin overlying the tumor or minor degrees of skin attachment. Such changes may become evident only on changing position of the breast or altering the degree of tension on the pectoral fascia, as already described in the maneuvers of abduction of the arm and forward bending in the sitting patient. Skin tension

over the suspected area is reduced best when the examiner with the patient lying supine gently displaces the breast upward, laterally or medially. Depending on the location of the tumor the skin may remain taut or slightly depressed over a carcinoma of small size. Many times, decreased mobility of the skin is best seen with a strong source of illumination lighting the area under investigation tangentially. Occasionally too,

inspection under magnification of two or three diameters may bring out alterations not otherwise seen.

Changes more obvious than these, such as obvious areas of retracted skin genuine retraction of the nipple, ulceration of the nipple, distinct erythema or lymphedema, are all signs of advanced disease of the breast, whether inflammatory or neoplastic. The physical signs of early disease of the breast are detected only by exact and meticulous methods of examination and are more suitable for schematic than photographic illustration. Lesions of the breast which are photogenic are so advanced as to be clinically obvious.

Too much emphasis cannot be placed upon development of perceptiveness in the digital tactile end-organs to the point of performing a sort of projected stereognostic function. Such perceptiveness is epicritic, never protopathic. Until such time as more exact methods may be developed, the physician's skill in examination is the only approach to detection of neoplasms of limited, local extent.

Transillumination of the breast is more impressive to the patient than it is informative to the examiner. After having had a considerable vogue some years ago it is now used by very few clinicians of experience. Roentgenograms of the breast for soft tissue detail are informative in some instances but can be misleading. Their net value does not seem commensurate with the effort and expense required. Malignant neoplasms are almost always associated with a distinct increase in vascularity of the affected breast and infrared photographs usually demonstrate rather well this circulatory phenomenon. Malignant epithelial cells in the breast have a somewhat higher uptake of radioactive phosphorus than do non-neoplastic cells, but unfortunately the disparity in uptake is not consistently evident until a carcinoma of the breast is of such size as to be clinically obvious.

For the woman who has a discharge from the nipple the first approach should be routine examination in search of a space-occupying tumor. If none is found, the examination is directed toward localization of the duct or ducts from which the discharge originates. In some instances, the patient herself may have discovered a pressure point. If there has been recent bleeding, inspection of the nipple under magnification may reveal an orifice containing dried blood. The most important part of the examination

is radial stroking of the breast with a finger tip with the patient supine, from the periphery toward the areola, repeating the process in clockwise fashion over twelve or more circumferential segments. If this maneuver does not localize the ductal area of origin it may be repeated with the patient in the sitting position, although the results are not as reliable. If a pressure point is successfully located, it should be marked on the skin with the patient supine and accurately located on the diagram. The location of the ductal orifice on the nipple through which the discharge exudes should also be indicated. It is advisable to repeat the examination five to seven days later to verify the apparent ductal source of discharge.

The procedure described for breast examination represents a preference. Both the techniques employed and the sequence of the examination vary from one physician to another. The essential objective is for the clinician to have an orderly pattern of procedure in examination of the breasts and adjacent regions, with such attention to detail as to accomplish a thorough survey in every patient.

Self Examination of the Breasts. As indicated, one phase of the program of public education directed toward discovery of cancer at an earlier stage is the recommendation that women undertake monthly self-examination of the breasts, particularly after the age of thirty years. While some physicians are critical, there are a number of valid reasons for professional support of such an effort. For obvious psychologic reasons, no more than a small minority of the population will ever pursue such a program consistently but even for this fraction it is less than rational to consider the possibility of monthly medical examination.

A recommended technique of self-examination of the breast is well portrayed in a cinematic film of the same title available from the American Cancer Society. However a certain number of women request instruction in the technique of self-examination from their physicians. The recommended procedure involves self-inspection of the breasts for the same changes described above. This is done with the woman sitting in front of a mirror with adequate illumination, sitting erect and with the arms adducted. Self-palpation of each breast is done in the supine position with the arm abducted, and with a small pillow under the scapular area of the side being examined.

Methodical circumferential palpation is done with the flat of the fingers of the opposite hand.

Obviously the initial achievement of importance is for the woman to become acquainted with mammary texture and consistency and frequently with the indurative abnormalities of "cystic" disease already present. The physician can do much to facilitate this learning process at the time of initial instruction. After several months of experience many women become able to detect new areas of indurative change and sometimes discover space-occupying tumors when they are as small as 1 cm in diameter.

DEVELOPMENTAL ANOMALIES

The least uncommon of the unusual developmental disorders is that of supernumerary nipples or breasts. Practically all such instances of ectopic development occur along the primitive milk line, which extends from the axilla downward across the nipple and thence downward and slightly medially to a point just below the inner third of the inguinal area. The great majority of supernumerary foci occur either in the area above and lateral or below the breast proper. Racial determinism is a factor of importance in location, frequency and relative incidence by sex. A Japanese study showed 1.5 per cent of males and 5 per cent of females with accessory nipples, while a report from a review of 20 000 school children in England demonstrated an incidence of only 0.4 per cent. The ectopic focus may be constituted of nipple alone, of parenchymatous mammary tissue in varying stages of development or more rarely both may be present. From a standpoint of clinical importance the most confusing ectopic foci are those of genuinely separate axillary mammary tissue. Many of the so-called supernumerary axillary foci are simply abnormal prolongations of the axillary tail of the breast. The indicated treatment is surgical excision for cosmetic or diagnostic reasons or both. The necessity for excision in the axillary area is more urgent, as carcinoma has been known to develop in axillary sites of ectopia.

Complete absence of one or both breasts or segmental failure of development of a breast is so rare that it is unknown to most clinicians. Minor degrees of disparity of size of the breasts is usually of developmental origin, while distinct hypertrophy is more reasonably ascribed to postnatal physiologic disturbances.

The most common malformation of the

nipple is that of inversion. Less often, a developmental fissuring of the nipples is seen. Both of these disturbances are with rare exceptions bilateral. Their clinical importance is that they be historically established as of developmental origin and not confused with deformities of the nipple secondary to a pathologic process within the breast.

There is some academic justification for a belief held by some that two intramammary developmental defects may contribute to some unusual neoplasms in later life. Remnants of the nipple pouch have been noted in the area of the large ducts as late as puberty. This may be the origin of rare epidermoid cysts, with or without squamous carcinoma, and also accounts for at least some instances of Paget's carcinoma of the ducts and nipple. Since the mammary gland represents a modified sweat gland structure, remnants of the mammary buds may be associated with the occasional development of carcinoma of the breast which is of distinct sweat gland structure.

TRAUMA AND FOREIGN BODIES

The breast has a remarkable tolerance for injury and usually will achieve restitution to normal even after extensive extravasation of blood into the parenchyma, fatty and other connective tissue. The most disruptive injury short of actual laceration, is also the most common of all, that of surgical incision for biopsy or resection. Resection is commonly followed by a period of organization ending in more or less severe fibrosis which not uncommonly may produce a three-dimensional lump more impressive than the tumor which may have originally been present. These postsurgical sclerosing reactions usually regress slowly over a period of months sometimes completely sometimes leaving a focus of smooth induration under or adjacent to the scar. The most common complication of trauma, either accidental or surgical, is the formation of a hematoma. If the blood is in a fluid state and reasonably well localized, it may be aspirated through a 14-gauge needle. Collections of organizing hematoma of more than minor size require incision and evacuation of the blood clot under local anesthesia.

Occasionally suppurative infection may follow trauma, usually developing in un aspirated or unevacuated areas of hemorrhage. It is of considerable importance that such areas of post traumatic cellulitis have prompt treatment to forestall ramification of the infection. If evidence of suppuration is lack

ing, treatment with sulfonamides or anti biotics should be instituted. If an abscess develops, prompt incision and drainage are indicated, preferably with the patient under general anesthesia to permit thorough evacuation of secondary pockets which may be present. This procedure should be followed by use of an antibiotic, or antibiotics, selected by sensitivity tests continued for a minimum period of ten days.

A more unusual but frequently confusing complication following trauma is that of *fat necrosis*. This lesion is most apt to develop in obese patients or those with pendulous breasts. Although a history of injury is not obtained in some patients, disruption of fat cells is the mechanism by which the process is initiated. A slow aseptic saponification of fat by blood and tissue lipase develops. The lesion may vary from 1 or 2 cm. to as large as 8 cm. in diameter. The surface is firm and irregular and frequently the skin becomes attached to the tumor and even retracted, although there is seldom any retraction of the nipple. Fat necrosis can, therefore, simulate carcinoma with such exactness that this is one of the few lesions for which radical mastectomy has been done on the basis of an apparently inescapable clinical diagnosis of carcinoma. Gross examination of the cut section usually differentiates the lesion from carcinoma because of the diffuse yellowish to orange coloration and the greasy consistency. In some of the lesions, cystic degeneration develops with the formation of pools of fat which may be surrounded by foci of hemorrhage. Eventually in cases of long duration, calcification occurs in the cystic areas. Treatment is simple excision with a margin of adjacent breast tissue. Diagnosis is readily made by frozen sections.

It is pertinent to emphasize that evidence is lacking to implicate trauma as a *causative factor in cancer of the breast*. As an exposed organ resting on the rigid thoracic cage, the breast suffers innumerable traumata during a woman's lifetime. A considerable percentage of women with cancer report prior injury to the affected breast. The breast is probably the most frequent site of cancer in which an allegation of causative trauma becomes the basis for medicolegal action. The fact is that, almost without exception, a minor injury directs attention to a preexisting lesion. While there is an imposing documentation against the thesis of trauma as a causative factor two considerations are sufficiently convincing.

The most frequent injury to the breast, and certainly as severe as any, is incision for resection of part of the organ. There is no recorded instance of cancer developing in an operative site not previously involved by a neoplastic process. Traumatic fat necrosis a particularly severe sequela of trauma resulting in cellular disruption, has not been a precursor of cancer in any recorded instance.

Carcinoma of the breast characteristically is a process of multicentric origin.

The most frequent *foreign body* in past years has been paraffin used for cosmetic reasons, resulting in the development of paraffinoma representing a foreign body granulomatous reaction and producing irregular lumps which may require excision. Some of the nonabsorbable suture materials particularly cotton may produce foreign body granulomas. Currently employed by some plastic surgeons, with preoccupations more esthetic than correctional, are the synthetic substances related to some of the plastic materials used by industry. Experimental evidence in animals as to their hazards is contradictory and sufficient time has not yet elapsed to assess the tolerance of human mammary tissue to these agents.

INFLAMMATORY LESIONS

Pyogenic Infections. Suppurative infections of the lactating breast, fairly frequent prior to the advent of chemotherapy have now become unusual although an occasional patient still progresses to the stage of abscess formation. Very occasionally, a woman with a genuinely cystic phase of dysplasia of the breast will develop a secondary infection in and around gross cysts, an inflammatory process superimposed on the dysplasia. Developing most often in long-standing cystic disease, such infections usually are low grade and slowly progressive. These rare instances represent the one situation where the old misnomer chronic cystic mastitis, becomes an apt and accurate designation. Seldom seen are women who develop a pyogenic infection without trauma, except lactation or gross cystic changes apparently as a result of hematogenous dissemination from some primary focus of infection, apparent or occult.

Abscesses beginning in the subareolar area and involving the central third of the breast have an extraordinary tendency to recur and to be resistant to treatment. Some of these centrally located infections seem to represent an extension of cutaneous axillary

Infections. In recurrent abscess of the breast, wide removal of the previous scar or scars, the underlying chronic abscess cavity and the adjacent ductal system should be undertaken during a period of quiescence of the infection. Such wide excisions should be performed only after repeated incision and drainage and use of antibiotics following which the process may gradually subside over a period of months. Choice of an antibiotic agent by sensitivity tests is of great importance the strain of *Staphylococcus pyogenes* var *aureus* most often present is resistant to penicillin.

Irradiation has been of no value in our experience except in occasional limited, early foci of cellulitis. In these, a tissue dosage of 300 to 450 r is adequate, divided into two or three treatments.

Tuberculosis. Tuberculosis of the breast is now an exceedingly rare disease. It is invariably either secondary to an active distant focus or from direct extension of disease in the underlying costal cage. Advanced tuberculosis of the breast is accompanied by multiple sinuses and frequently by involvement of the regional axillary nodes. Thus, the treatment is radical mastectomy.

Plasma Cell Mastitis. This rare form of mastitis usually occurs in married women under the age of forty years. The dominant symptom is pain. There is usually localized erythema overlying a poorly defined bulky mass in the breast and occasionally there is a milky discharge from the nipple. The skin may become adherent to the mass, is commonly edematous and retraction of the nipple may be present. Thus the lesion often mimics a carcinoma of the acute or inflammatory type. Enlarged axillary lymph nodes of some size may be present. Clinicians neglecting the confirmatory evidence of histologic diagnosis, have performed radical mastectomies for this lesion. Some of these purely inflammatory processes, perhaps most, are highly radiosensitive. When confronted by such findings in a married woman under forty years of age with no recent history of lactation, information of diagnostic value will probably be obtained from one or more biopsies of the involved skin, including the subcutaneous tissue, and smears from the nipple discharge, if present. This can be done as an office procedure under local anesthesia. If histologic study of the material so obtained fails to show foci of carcinoma cells in the skin or subjacent tissue or in the smear one can eliminate the

possibility of genuine inflammatory carcinoma with certainty. However the possibility of carcinoma with erythema of overlying skin cannot be excluded. If the evidence favors the diagnosis of plasma cell mastitis roentgen therapy in moderate dosage is advised, about 600 rads delivered in two weeks time. This amount of irradiation over such a period usually induces complete regression of this form of mastitis. If the physical findings are not entirely consistent with this disorder and in those patients in whom this amount of irradiation does not produce satisfactory regression within three weeks, adequate material for biopsy should be obtained by a surgical approach. Should carcinoma be demonstrated, it is usually biologically inoperable and best treated by intensive roentgen therapy.

Mammary Duct Ectasia (Comedomasitis). This process may simulate carcinoma in elderly women, to whom it is almost limited. The process is characterized by wide dilatation of the ducts particularly of the central third of the breast, in which accumulation of fatty debris is accompanied by ductal and periductal inflammation and eventual sclerosis. With severe sclerosis the ducts may become shortened, thus retracting the nipple so that the physical findings approximate those of subareolar carcinoma of the ducts. In the early stages, the involved ducts can be outlined as individual circoid or vermiform structures with little or no resemblance to a possible neoplasm. In the later stages of sclerosis induration and nipple retraction, excision and histologic diagnosis become necessary.

It is worthy of note that all three of the inflammatory processes which may simulate cancer—fat necrosis, plasma cell mastitis and duct ectasia—are believed by some observers to be variants of the same basic disorder of ductal stasis with trauma playing a determinant part, followed by inflammatory reaction to chemical irritants.

DISORDERS DUE TO ABNORMAL PHYSIOLOGY

Prepubertal Hyperplasia. The earliest example of hyperplasia of endocrine origin is that of *neonatal hyperplasia*. Almost one-half of infants, both male and female exhibit a transient enlargement of the mammary disk as a result of hormonal stimulation through the placental blood. In about one of ten newborn infants, this is accompanied by a scanty secretion of thin fluid from the nip-

ples. This hyperplasia may persist for six months or more, particularly in female nursing infants.

Rare instances of mammary hyperplasia and hypertrophy during the first five years of life, or so-called precocious mammary development are almost invariably due to hormone-producing neoplasms of the ovary, adrenal cortex or hypophysis or to hypothalamic lesions.

Later in life, and most often from the ages of eight to twelve years, unilateral hypertrophy unassociated with any recognizable endocrine abnormality is not uncommon. Such female prepubertal hypertrophy may be transient or may persist until puberty, during which period the opposite breast undergoes development equal to that of its fellow.

At the time of puberty, there are rare examples of hypertrophy of one breast or even of both. Attempts at control of such hypertrophic abnormalities have been unsuccessful and when they are disfiguring, appropriate plastic surgery is indicated.

Gynecomastia. Enlargement of the breast in the male, either unilateral or bilateral is most common during the period of puberty and after the age of forty years. Gynecomastia developing during puberty is ordinarily transient and unilateral, with an average duration of twelve to eighteen months. It represents an adolescent, mammary dysplasia of functional type, secondary to the profound changes in steroid hormone levels characteristic of adolescence. The process may be a diffuse, slightly lumpy or even nodular enlargement of the entire gland, in which case the diagnosis is obvious. More often, however, there is a discrete tumefaction, usually subareolar in position and the area of localized indurative change may be from one to several centimeters in diameter. The finding of a measurable lump frequently arouses such apprehension in the clinician that he performs an excision for microscopic diagnosis. No such action is necessary because malignant neoplasia in the mammary gland of the adolescent male is unknown. The differential diagnoses of remote chance in boys at this age are dermoid cyst, lipoma, hemangioma and lymphangioma. The only indication is for observation at intervals of several months. In only two instances in my experience has adolescent gynecomastia persisted and been of such degree as to require treatment, which is subcutaneous mastectomy. Attempts at control of adolescent gynecomastia by hormone

treatment are unnecessary, ineffective and not without some possible hazards.

Gynecomastia developing in the adult male is usually seen in those of middle or late life, is almost invariably unilateral and is also due to functional endocrinologic disturbance, except for rare instances secondary to interstitial cell tumor of the testis, atrophy of the testis, hypophyseal adenoma or adrenocortical lesions. In later life gynecomastia may be secondary to hepatic cirrhosis with or without a history of alcoholism in which instances the lesion presumably is due to the inability of the liver to accomplish intermediate metabolism of steroid hormones. Examination in the adult should include careful survey of the involved breast for a three-dimensional tumor, palpation of the testes and inquiry as to the possibility of cirrhosis of the liver. If a distinct circumscribed, three-dimensional tumor is present, it should be excised to rule out carcinoma, although, in the male, fixation of the skin and pectoral fascia is almost invariably present when the diagnosis of carcinoma is first made. Otherwise, no treatment is indicated or useful, unless subcutaneous mastectomy becomes necessary for unsightly enlargement.

Dysplasias of the Female Breast ("Cystic" Disease) The dysplasias of the breast have achieved an unenviable distinction from the unnecessary complexity bestowed upon them in the past fifty or more years by contributors to the literature of surgery and pathology. A few years ago a rough count of the nomenclature employed in several textbooks and a score or more of articles showed thirty-eight separate, frequently ambiguous, terms used to designate individual facets of the abnormalities of the hyperplasia involution cycle in the breast, all of which are readily divisible into three principal phases. Some of the nomenclatural labels thus coined by fertile minds are polysyllabic and rather accurate, others are eponymic and misleading. Even the name of a symptom, mastodynia, has been conferred on a phase of this complex of dysplastic pathology. Some of these hair-splitting tags have come to mean different things to different people. An example is the eponymic designation of one situation as Schimmbelbusch's disease, a popular term with clinicians, probably for the suggestion of erudition implied in the use of so unusual a name but which nowadays is seldom used to indicate the process described by Schimmbelbusch.

The most reasonable hypothesis is that all of these dysplasias no matter how disparate their gross and microscopic appearance may be after years of secondary changes, are nevertheless part of an over-all pattern. The highly variable patterns which develop can be correlated in terms of a single complex of abnormalities in epithelial and stromal components. Valid etiologic factors are endocrine dysfunction and atypical response in the mammary target tissue.

No satisfactory generic term is apparent, except the rather ambiguous expression employed here, *dysplasias*. The most inaccurate misnomer of all is "chronic cystic mastitis" for these changes are in no sense inflammatory. Cystic changes are frequently absent, and if present are more often minor than major so that "chronic" is the only valid word in this traditional title. "Cystic" disease is a less offensive misnomer for at least it disposes of the implication that the process is inflammatory but quotation marks should be used around cystic for the reason indicated.

The division of the dysplasias into three general groups is accomplished naturally by considering the three dominant abnormalities in the hyperplasia involution cycle.

Probably the earliest phase of dysplasia in most instances and the persistently dominant phase in some women, is that of failure of involution, or persistent epithelial hyperplasia.

The reverse of failure of involution is hyperinvolution of the epithelium, rather than returning to the resting phase undergoes progressive involutionary changes with flattening and blunting. With this there is apt to develop a shedding off (mazoplasia) of the involuted epithelium and some secretory tendency. Cystic dilatations appear which at first are microcystic; as the process advances, probably by coalescence of small adjacent cysts, gross cysts of varying size may develop. The designation for this phase of hyperinvolution is its dominant manifestation, that of cystic development.

The stromal components of the breast are rarely unaffected by the epithelial changes of dysplasia. In the florid phase of proliferative activity there is a concomitant though minor degree of fibroplasia, particularly in the periacinar and periductal layers and this stromal reaction is also subject to either involution or hyperinvolution. When the latter change becomes dominant, progressive hyalinization of the connective tissue occurs, with eventual dense sclerosis. Generally the

sclerosing changes occur in perilobular fashion, but the changes of advancing sclerosis may be extremely irregular. Foci of acinar hyperplasia or microcystic change may become circumscribed and compressed by sclerosing reaction. The same sort of sclerotic reaction may proceed within the walled-off area, isolating contained foci of epithelial elements which may be hyperplastic or cystic, or both. Such circumscribed areas may vary in size from microscopic foci to large areas representing space-occupying tumors of several centimeters in diameter. In an advanced area of sclerosing reaction, formerly hyperplastic epithelium becomes compressed and pyknotic and these compressed, distorted, deeply-staining cells are the only residual evidence of the florid phase of hyperplasia of former years now completely overcome by the violent intensity of the sclerotic stromal reaction. In this phase of dysplasia, as in the others there may be a histiocytic response less often some minor foci of lymphocytic infiltrate, probably, responsible for the original designation of mastitis. These are the prominent changes in the sclerosing phase.

The least frequent phase of dysplasia is the secretory or exudative response, in which the secretory acinar function is dominant. Whole lobules of mammary tissue become microcystic, the secretory products drain freely through the ductal system and appear at the nipple in the form of a pseudolactiferous exudate which is rarely profuse. This form of exudative dysplasia constitutes less than 2 per cent of all cases and may be regarded as a variant of the cystic phase.

It should be emphasized that pure examples of hyperplastic, cystic or sclerosing dysplasia are rarely seen. Some degree of all three phases generally will be found in long-standing cases of dysplasia. It is common for one phase to be dominant and it is the variegation of gross and microscopic appearance of acinar ductal and stromal response which has given rise to the bewildering complexity of nomenclature. For example dilatation of terminal tubules and acini with the formation of microcysts, usually diffuse is the hallmark of so-called Schimmelbusch's disease. With this, there is frequently an accompanying epithelial proliferation in the terminal ducts to form intra ductal hyperplasia, and some degree of proliferative acinar hyperplasia which may spill into the adjacent stroma and resemble, superficially an infiltrative process. These changes of ductal papillomatosis adenosis

and microcystic formation are to be regarded only as various phases of hyperplasia and involution respectively. In some areas ductal proliferation produces tubules with a blind ending, referred to as blunt duct adenosis. The formation of gross cysts which are clinically palpable occurs in 15 to 20 per cent of women with dysplasia. In some instances a cyst, when exposed surgically, has a bluish color which promptly disappears when the cyst is incised or when the investing layer of tissue is removed. The coloration is simply an optical illusion through the surrounding fat and parenchyma but has acquired a special and unwarranted dignity by the designation of "blue-domed cyst." In 1 or 2 per cent of women with dysplasia, the sclerosing phase will become so overwhelming as to produce a hard fixed tumor occasionally with attachment of the skin, readily mistaken for carcinoma. Even on microscopic examination, especially of frozen sections the extreme distortion and pyknosis of the formerly hyperplastic cells may mimic carcinoma. This lesion of *sclerosing adenosis* or *adenomatosis*, has probably been the lesion most frequently misdiagnosed as carcinoma. It is most reasonably interpreted as an end phase of a severe sclerosing reaction in dysplasia.

Incidence Clinically recognizable dysplasia of the breast is probably present in one-fourth to one-third of white women, based on the recognition of abnormal indurative changes. The process is practically always bilateral by objective criteria, although in some instances only one breast may be affected and occasionally but one segment of a breast. The process may appear immediately after puberty but the great majority of dysplasias are seen in women beyond the age of thirty years and up to the climacteric. In the postmenopausal years, the process undergoes gradual resolution with rare exceptions. Pregnancy has a beneficial effect, particularly in the nulliparous woman. The disorder may improve remarkably after pregnancy or even fail to recur entirely.

Symptoms. The most frequent symptoms are soreness, tenderness or even severe pain beginning five to seven days premenstrually and subsiding rapidly after the onset of menstruation. In some patients the period of distress is longer beginning about the time of ovulation each month. Less often, the presenting complaint is the self-discovery of a lump in the breast with little or no soreness. It is very common for the

symptoms to occur in an irregularly periodic pattern with months or even a year or more between periods of distress. In many women the severity of symptoms is profoundly influenced by emotional stress such as domestic difficulties or the death of a relative or friend from cancer of the breast.

Physical findings. When the dysplastic process is in its early phase most often seen in younger women, the dominant change is most apt to be *hyperplastic*. The first evidence of such change is the appearance of small foci of abnormally granular or beady thickening, frequently with tenderness sharply localized to the area of altered tissue. In more diffuse forms larger segments of the breast show granular or beady or almost vermiciform indurative changes, often linear in arrangement. Not infrequently these changes vaguely resemble a string of beads of varying size. The hyperplastic phase of the process is that in which pain and soreness are apt to be most distressing.

More advanced changes are those in which some of the proliferative changes have evoked a sclerosing reaction of some degree and the larger lesions may now constitute three-dimensional tumors. Such indurative changes, however, can reach a size of even several centimeters in diameter and still be distinguishable as diffuse induration without the qualities of a space-occupying tumor.

When it is difficult to differentiate between advanced changes of dysplasia and a three-dimensional tumor in one or more areas, the physical findings may be related to the time of the menstrual cycle. Examination of questionable areas is best done a few days to one week after the onset of menstruation. Not infrequently what seemed to be a three-dimensional lump during the premenstrual period will have decreased in size or disappeared entirely on re-examination after the onset of menstruation. For the woman who has the symptomatology and clinical findings of dysplasia, a good working rule is to re-examine "tumors" of 2 cm diameters or less before proceeding with surgical approach for biopsy.

When the *cystic phase* of dysplasia is in the microcystic stage the physical findings are indistinguishable from those of the hyperplastic or proliferative phase. If the cysts begin to acquire some gross size, even when less than 1 cm. in diameter they are usually evident as smooth-surfaced moderately movable nodules without attachment to the adjacent breast. Their consistency

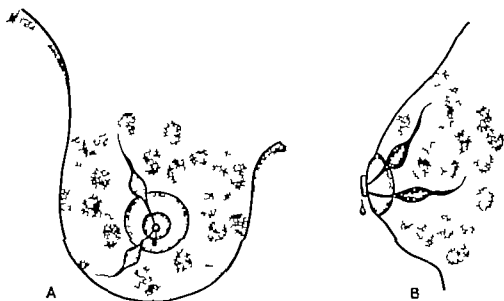


Figure 6 A, Exudative phase with discharge from nipple. Also indicated is the presence of palpable duct or ducts draining involved segment or segments of breast. No space-occupying tumor B Development of ductal papillomatosis in central third of breast.

depends on the amount of contained fluid. When gross cysts reach a size of several centimeters, they may be indistinguishable from solid tumors if the tension of the intracystic fluid is sufficient to distend the wall of the cyst. By this time, too, the surface may seem to be irregular because of overlying indurative change in the surrounding breast, although the largest cysts usually remain mobile and unattached to the skin.

In Figure 6 A is shown the exudative phase with discharge from the nipple. Also indicated is a not infrequent finding in such patients the presence of a palpable duct or ducts draining the involved segment of the breast from which the pseudolacteal exudate originates. Lacking a space-occupying tumor the only requirement in this situation is a smear of the nipple discharge to be examined only for red blood cells. If red blood cells are not found, the only indication is for periodic observation.

Figure 6 B illustrates the development of ductal papillomatosis in the central third of the breast, a ductal hyperplasia producing papillary excrescences of epithelium. Such areas of nodular epithelial ductal hyperplasia are extraordinarily fragile and a considerable percentage of them give rise to minute amounts of serosanguineous discharge from the nipple. In this situation, one's first effort should be directed toward the search for a space-occupying tumor. If none is found, an effort should be made to locate the ductal area from which the bleeding is originating. If the origin of the bleeding

can be consistently demonstrated on two or more examinations the involved ductal area should be explored as described below. Otherwise, if the patient is premenopausal, continued observation is justifiable, as only once in twenty times will bleeding from the nipple before the menopause indicate the presence of malignant change in ductal epithelium.

Relation of dysplasias of the breast to carcinoma. The relation of the dysplasias of the breast to carcinoma is still controversial, but the evidence for dysplasia as a precancerous process is flimsy indeed. If a patient with dysplasia has one breast predominantly affected, she has just as much chance of developing cancer in the less affected breast. Our experience includes over 600 women with objective criteria of dysplasia who were followed for periods of from two to twelve years. Only two of these patients developed carcinoma of the breast under observation; one had had an excess of mammary carcinoma in her family history. Various degrees of dysplasia are found in surgical specimens showing carcinoma, the frequency depending on the liberality with which the pathologist makes the diagnosis of dysplasia, but the only significance of such coexistence is that women with dysplasia may also develop carcinoma.

From our present state of knowledge, the following statements seem permissible in relation to this problem.

The microscopic lesion of blunt-duct adenosis may be a precursor to carcinoma.

The woman with objective changes of dysplasia deserves careful observation for the development of a dominant tumor which may be carcinoma. The development of such a space-occupying tumor particularly if the dysplasia process is severe may be masked by the chronic indurative changes. The intervals of observation should be four to six months.

For the woman with well-established dysplasia presenting multiple space-occupying lumps, good management is frequent observation and measurement of the tumors, particularly if the diagnosis has been established by prior excisional biopsy. Many of these lumps will disappear under observation.

Simple mastectomy is rarely if ever indicated for mammary dysplasia as a prophylactic procedure. From what has been said above, the only reasonable prophylaxis would be bilateral mastectomy, a therapeutic absurdity.

Treatment of mammary dysplasias. Three main problems are most pertinent.

The patient with symptoms and signs of dysplasia without a three-dimensional or dominant lump.

The patient with a space-occupying solitary tumor.

The patient with nipple discharge.

WHEN NO DOMINANT LUMP IS PRESENT The patient without a dominant lump usually consults a physician because of soreness, tenderness or pain. For at least one half of such patients a careful examination followed by adequate discussion and explanation of the process, is in itself adequate therapy. The reassurance of learning that the symptoms and the process are not related to cancer will satisfactorily diminish the severity of the symptoms. If the indurative changes are more than minimum, examination twice yearly should be recommended. In women with any degree of pendulousness of the breasts adequate support should be recommended in the form of a fitted brassiere. During the period of greatest discomfort prior to menstruation, the majority of patients obtain great relief from wearing a brassiere at night, for which purpose a "sleeping" brassiere may be obtained if desired. Fitted brassieres should be so designed as to provide adequate uplift to the breasts and relaxation of the suspensory fascia.

These measures of reassurance and proper support of the breasts will produce satisfactory relief of symptoms in at least eight

or possibly nine of every ten patients. In the remainder hormonal therapy is usually beneficial, either in the form of small doses of androgenic hormone in the first half of the cycle or progesterone in the latter half. If an androgen is employed methyltestosterone by mouth is as effective as other preparations. It is desirable for the first two months to employ testosterone propionate by intramuscular injection, in dosage of 25 mg. two or three times weekly for two weeks to measure adequately the response to treatment.

WHEN A DOMINANT LUMP IS PRESENT When a dominant three-dimensional lump is present with physical findings characteristic of dysplasia elsewhere in the breasts, a very useful preliminary diagnostic measure is aspiration of the tumor as an office procedure under local anesthesia. The primary objective of this maneuver is to determine whether the tumor is solid or cystic. If it is cystic and can be evacuated completely it will fail to refill in at least eight out of ten instances and thus the patient is spared the distress and expense of a surgical procedure for excision. In all such instances of successful aspiration of a cyst, the patient should be instructed to return in six weeks for re-examination. If the cyst has refilled, it is preferable to proceed with excision rather than to repeat the aspiration. Reaccumulation of fluid may indicate the presence of papillary changes in the epithelial lining. The development of carcinoma in a gross cyst developing in dysplasia is extremely unusual, not more than 0.5 per cent of all carcinomas of the breast give evidence of intracystic origin.

The procedure is simple and rapid. A site on the skin several centimeters from the tumor is selected, prepared with Zephiran solution, and an intracutaneous wheal is raised with injection of about 0.5 cc. of 1 per cent procaine solution. A small stab wound with a no. 11 scalpel is made through the wheal to permit passage through the skin of a no. 16 needle attached to a 20-cc. metal tipped syringe. With the tumor held stationary by one hand, the needle is advanced through the breast until it encounters the periphery of the tumor. The needle is then slowly pushed into the tumor. If the tumor is cystic, the needle usually passes suddenly into the cavity after the preliminary resistance of the fibrous wall. As thorough an aspiration as possible is done, the last of the fluid being obtained by combining further suction with some flat hand pressure on the

tumor A small dressing on the puncture wound can be removed in twenty four hours. Our experience with cytologic study of the centrifuged sediment from such aspirations has been so unsatisfactory that it has been abandoned. The most reliable indication for further action in a successfully aspirated cyst is the reaccumulation of fluid.

If the solitary tumor proves to be solid rather than cystic surgical exposure for biopsy is mandatory

WHEN THERE IS DISCHARGE FROM THE NIPPLE. As indicated if the patient with discharge from the nipple is found to have a space-occupying tumor the nipple discharge should be disregarded except for cytologic study and prompt excision of the tumor for diagnosis recommended For the patient with nipple discharge and without a three-dimensional lump the next most important step is to determine whether or not the exudate contains red blood cells. If so and the segment of the ductal system from which the bleeding originates has been located, the involved area should be exposed through a circumareolar incision with the patient under general anesthesia. Not infrequently by careful palpation through the wound, nodular involvement of a duct by papillary change can be digitally determined. Prior to incision, it is possible in some instances to pass the blunt end of a straight skin needle into the orifice on the nipple through which the bleeding exudes and to pass the needle gently into the duct. With or without such localization, a wedge-shaped resection of the involved part of the ductal system should be done the radial extent of which should be one-third of the distance from the nipple to the periphery of the breast. If this excised portion of the ductal system fails to show a lesion and the patient is premenopausal, further action is not indicated, as the ductal papillomatosis coincident with dysplasia of the breast is not known to be a precancerous process. If the patient is postmenopausal the likelihood of bleeding from the nipple being due to ductal carcinoma increases proportionately with each decade past the menopause. In this instance, if a preliminary conservative ductal resection fails to demonstrate a benign papilloma, simple mastectomy should be done, particularly if the patient is ten or more years past the menopause. If the excised portion of the ductal system shows a fully developed malignant lesion, radical mastectomy is indicated.

Pseudoneoplastic Hyperplasia. There are

both ductal and acinar states of dysplasia which may resemble closely a genuine neoplastic process: there is no intent to lend to these lesions the dignity of separate entities. The fact is however that the histologic appearance of some of the more florid of these processes may be so atypical as to make interpretation difficult for the most experienced pathologists. This is to say that there are occasional situations where the differentiation between hyperplasia, with considerable atypism and neoplasia is difficult indeed.

Reference has already been made to papillomatosis of the ducts and when there are focal areas of atypical nodular hyperplasia of ductal epithelium the differentiation from genuine ductal papilloma may be extraordinarily difficult. Actually the differentiation is largely academic, for there is no present evidence that either hyperplastic papillomatosis or the genuine papillomas are precancerous lesions.

Atypical acinar hyperplasia is most often seen where there are areas of long standing cystic involution with development of papillary excrescences in the epithelial lining of such cysts a condition referred to by some as papillary cystophorous hyperplasia. As in the ductal lesions interpretations of such atypical changes are best deferred until permanent sections are available. The distinct criteria of malignant neoplastic change should be agreed upon by several pathologists before radical treatment is undertaken.

BENIGN NEOPLASMS

Fibroadenoma (Adenofibroma) As indicated in Tables 1 and 2, this compound neoplasm of fibroblastic and epithelial origin exceeds all other benign neoplasms of the breast by a large margin. It is the only common mammary neoplasm in young women, with a peak of incidence between the ages of twenty and twenty five. Fibroadenomas occasionally develop during adolescence a few are seen in women from thirty years of age up to the age of the menopause and they are a curiosity in women in the postmenopausal years. Usually there are no subjective symptoms occasionally there may be slight tenderness. This neoplasm has highly characteristic physical criteria in its round or ovoid shape smooth surface and most of all in its extreme mobility within the tissue of the breast. As indicated in Figure 7 the encapsulation of fibroadenomas is such

Table 2. Common Mammary Neoplasms

ORIGIN	BENIGN VARIANTS	APPROX. INCIDENCE (PER CENT)	MALIGNANT VARIANTS	APPROX. INCIDENCE (PER CENT)
I Epithelium a. Ductal b. Acinar	Papilloma Adenoma	18 2	CARCINOMA Lobular Carcinoma	93.0 5.0
II. Connective tissue	Lipoma Rare neoplasms, including granular cell myoblastoma, mesodermal tumors	3	Rare lesions, including liposarcoma, hemangiosarcoma, lymphosarcoma	0.5
III Compound	FIBROADENOMA	75	Cystosarcoma phyllodes	1.5
		100.0		100.0
Hyperplasia stimulating neoplasm	DYSPLASIAS Cystic disease principal phases (a) proliferative (b) cystic (c) sclerosing			

that gentle pressure over the tumor with a finger will cause easy displacement of the lump within the breast. Multiple fibroadenomas are seen in some 15 per cent of patients with this tumor. Approximately one in ten patients develops one or more additional fibroadenomas after the appearance of an initial tumor.

There is some reasonable evidence to support the thesis that fibroadenomas are related to hyperestrinism, for these neoplasms can be induced in animals by administration of estrogenic steroids and it is not uncommon to notice their rapid enlargement during pregnancy. Most of the malignant neoplasms known as cystosarcoma phyllodes have their origin in pre-existing fibroadenomas which usually have been present for periods of five or more years.

Treatment is that of any undiagnosed three-dimensional tumor of the breast—surgical excision. Because of the characteristic physical findings and their predilection for occurrence in younger women, the accuracy of diagnosis for fibroadenomas is greater than for other space-occupying lesions of the breast.

Intraductal Papilloma. Genuine benign neoplastic growths in the form of papillomas of the ductal epithelium are a poor second in frequency. They occur in women of almost any age over twenty years. The majority or about three of four are located in

the ductal system of the central third of the breast, the remainder more peripherally. Minute papillomas several millimeters in diameter may cause significant bleeding from the nipple. Larger papillomas often produce ductal obstruction which, if followed by bleeding into the obstructed portion, will result in severe distention with tenderness, pain and a palpable soft enlargement. Occasionally there will be periodic, rather profuse bloody discharge from the nipple as a duct alternately is obstructed and drains. Infection may complicate the picture, producing retraction of the nipple and secondary axillary lymphadeni-

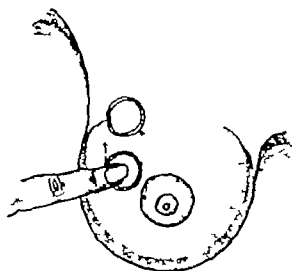


Figure 7. Mobility of an encapsulated fibroadenoma.

tumor. A small dressing on the puncture wound can be removed in twenty four hours. Our experience with cytologic study of the centrifuged sediment from such aspirations has been so unsatisfactory that it has been abandoned. The most reliable indication for further action in a successfully aspirated cyst is the reaccumulation of fluid.

If the solitary tumor proves to be solid rather than cystic, surgical exposure for biopsy is mandatory.

WHEN THERE IS DISCHARGE FROM THE NIPPLE. As indicated, if the patient with discharge from the nipple is found to have a space-occupying tumor the nipple discharge should be disregarded except for cytologic study and prompt excision of the tumor for diagnosis recommended. For the patient with nipple discharge and without a three-dimensional lump, the next most important step is to determine whether or not the exudate contains red blood cells. If so and the segment of the ductal system from which the bleeding originates has been located, the involved area should be exposed through a circumareolar incision with the patient under general anesthesia. Not infrequently by careful palpation through the wound, nodular involvement of a duct by papillary change can be digitally determined. Prior to incision, it is possible in some instances to pass the blunt end of a straight skin needle into the orifice on the nipple through which the bleeding exudes and to pass the needle gently into the duct. With or without such localization, a wedge-shaped resection of the involved part of the ductal system should be done the radial extent of which should be one-third of the distance from the nipple to the periphery of the breast. If this excised portion of the ductal system fails to show a lesion and the patient is premenopausal, further action is not indicated, as the ductal papillomatosis coincident with dysplasia of the breast is not known to be a precancerous process. If the patient is postmenopausal, the likelihood of bleeding from the nipple being due to ductal carcinoma in creases proportionately with each decade past the menopause. In this instance, if a preliminary conservative ductal resection fails to demonstrate a benign papilloma, simple mastectomy should be done, particularly if the patient is ten or more years past the menopause. If the excised portion of the ductal system shows a fully developed malignant lesion, radical mastectomy is indicated.

Pseudoneoplastic Hyperplasia. There are

both ductal and acinar states of dysplasia which may resemble closely a genuine neoplastic process: there is no intent to lend to these lesions the dignity of separate entities. The fact is however that the histologic appearance of some of the more florid of these processes may be so atypical as to make interpretation difficult for the most experienced pathologists. This is to say that there are occasional situations where the differentiation between hyperplasia, with considerable atypism, and neoplasia is difficult indeed.

Reference has already been made to papillomatosis of the ducts and when there are focal areas of atypical nodular hyperplasia of ductal epithelium, the differentiation from genuine ductal papilloma may be extraordinarily difficult. Actually the differentiation is largely academic, for there is no present evidence that either hyperplastic papillomatosis or the genuine papillomas are precancerous lesions.

Atypical acinar hyperplasia is most often seen where there are areas of long-standing cystic involution with development of papillary excrescences in the epithelial lining of such cysts, a condition referred to by some as papillary cystophorous hyperplasia. As in the ductal lesions, interpretations of such atypical changes are best deferred until permanent sections are available. The distinct criteria of malignant neoplastic change should be agreed upon by several pathologists before radical treatment is undertaken.

BENIGN NEOPLASMS

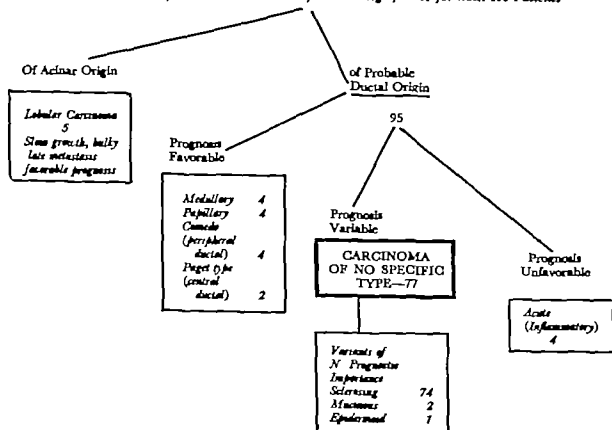
Fibroadenoma (Adenofibroma) As indicated in Tables 1 and 2, this compound neoplasm of fibroblastic and epithelial origin exceeds all other benign neoplasms of the breast by a large margin. It is the only common mammary neoplasm in young women, with a peak of incidence between the ages of twenty and twenty five. Fibroadenomas occasionally develop during adolescence a few are seen in women from thirty years of age up to the age of the menopause and they are a curiosity in women in the postmenopausal years. Usually there are no subjective symptoms occasionally there may be slight tenderness. This neoplasm has highly characteristic physical criteria in its round or ovoid shape, smooth surface and most of all in its extreme mobility within the tissue of the breast. As indicated in Figure 7 the encapsulation of fibroadenomas is such

specific type (Table 3) From the standpoint of gross pathologic changes, however, almost all of these common carcinomas provoke a sclerosing stromal reaction of variable intensity which is responsible for the hard, irregular consistency of almost eight of every ten carcinomas on clinical examination. When the sclerosing process is advanced, the neoplasm will exhibit a stony hard consistency. This sclerosing reaction led to their separation in older classifications under the designation of *scirrhous carcinoma* with the now discredited implication that intense sclerosis implied a more favorable prognosis. The informative value of the "scirrhous" reaction lies not in prognosis, but in gross diagnosis. It produces on cut section a retracted, gummy surface with yellowish foci representing minute islands of fat which have become surrounded and isolated. There are two minor variants of this group those with mucinous degeneration and those with epidermoid differentiation representing approximately 2 per cent and 1 per cent respectively of all carcinomas.

There are four recognizable cell types of ductal carcinoma which, when predominant, may permit a favorable prognosis. These are, in order of descending importance from a

prognostic standpoint medullary papillary comedo and Paget types of carcinoma. The *medullary carcinoma* is more apt to occur in women under the age of fifty. The tumors are frequently bulky in size and may appear to be encapsulated and often are mistaken grossly for fibroadenomas. Although they may appear to be highly malignant microscopically their prognosis is five times more favorable than average because of their inherent tendency to remain localized to the breast and regional nodes for considerable periods. *Papillary cystadenocarcinoma* is an unusual neoplasm with a tendency to spread throughout the ductal system and to involve a considerable portion of the breast often a quadrant or more. Inadequate excision of these neoplasms may be followed some years later by local recurrence without metastasis to regional nodes. The *comedo carcinoma* is an intraductal process in the beginning and may remain so limited for many months. Necrotic material is extruded into the lumina of the cancerous ducts and on cut section, compression will cause extrusion of wormlike masses of necrotic tumor consequently its nomenclatural prefix. Eventually these neoplasms invariably invade mammary stroma and metastasize to regional nodes

Table 3 Mammary Carcinoma—Variants of Clinical Significance for Each 100 Patients



tis thus imitating a carcinoma of the central third of the breast.

Treatment is the same as for dysplastic ductal papillomatosis. Radical management of this lesion is entirely unjustifiable, for the great majority are localized to a single quadrant and the process does not undergo malignant transition. This latter fact was well documented by a study of seventy six patients with intraductal papilloma treated by local excision in whom follow-up for five to ten or more years showed no single instance of subsequent carcinoma.

Other Benign Tumors. Other benign neoplasms are so unusual as to occur in a total of not more than 1 per cent of all patients with lesions of the breast. Included are genuine intramammary lipomas (as compared to those developing in the subcutaneous layer of fat) adenomas and a few other esoteric items.

MALIGNANT NEOPLASMS

Carcinoma of the Female Breast. Although there are some striking racial variations of incidence, carcinoma of the breast is the most common form of cancer in white women, amounting to about 22 per cent of all cancers. Uterine cancer ranks second, contributing about 19 per cent of total incidence. In women beyond the age of forty the relative incidence of mammary carcinoma increases progressively with advancing age, when plotted against the size of the female population in each decade. Because of its natural history, with long duration of the disease in many women who do not obtain a curative result from treatment, the prevalence of this disease or the yearly incidence plus the carry-over of previously diagnosed cases far exceeds that of any other form of cancer in the female. A study of annual morbidity rates in upper New York state indicated five living patients with cancer of the breast for each death from the disease. The increase in mortality from carcinoma of the breast in the United States since 1920 has been progressive and the rate of increase considerably exceeds that of the total population. In 1920 there were 8685 recorded deaths in a population of 105,710,000. In 1948 the mortality was 18,928 in a population of 146,113,000. Currently mortality exceeds 20,000 yearly. Nevertheless the age-adjusted mortality is not as dismal as these figures would indicate and actually in some areas there is evidence of a slight decrease. In California, for example, the

age-adjusted mortality rate per 100,000 increased from 19.7 in 1910 to 28.0 in 1940 but in the decade from 1940 to 1950 decreased to 25.3.

Etiologic factors: The proximate cause of mammary carcinoma is unknown, but there are certain factors which are either almost requisite or appear in excess in the background of women developing this disease. A presumptive requisite is the exposure of the mammary epithelium to the long-continued stimulus provided by estrogenic and perhaps other steroids for the development of carcinoma of the breast in women deprived of active ovarian function before the age of thirty is rare. Carcinoma of the breast also occurs in relative excess with the following historical features: family history of cancer of the breast, nulliparity, failure of the breast to perform its expected physiologic function (lactation) following full term pregnancy, complications of lactation and late onset of the climacteric. It has been statistically demonstrated that carcinoma of the breast is more prevalent in women of better than average economic circumstances, but this may be related to a lesser degree of fertility. The evidence from a number of well-controlled studies in different countries leads adequate support to the belief that human mammary carcinoma is frequently a disease of genetic determination, although evidence is yet lacking that it is associated with a maternally transmitted, specific agent.

Speculation as to the possibility of administered estrogens acting as a causative factor is entirely unverified, although it is established that significant amounts of exogenous estrogen can produce augmentation of an existing, sometimes occult, carcinoma. For this reason, it is advisable to withhold the use of estrogens in women who have a family history of mammary carcinoma unless their use is considered essential. Women with a family history of mammary carcinoma deserve careful survey of the breasts at intervals of no less than six months. If the necessity for administration of estrogens arises it is advisable to examine the breasts even more frequently during the first few months of medication.

Clinical-pathologic correlation. About ninety-five of each 100 mammary carcinomas are considered by consensus to be of ductal origin. Of this dominant group nearly eighty are without distinctive morphologic features and may be designated as of no

rants, with only 5 per cent of lesions originating in the last-named area. Approximate figures are as indicated in Figure 8.

EVOLUTION OF LOCAL GROWTH. Evidence now available indicates that most, if not all carcinomas of the breast have their inception as multicentric foci of microscopic neoplasia, whether of ductal or acinar origin. Most frequently a single area of parenchyma is irreversibly involved and with progression of the process there is a confluence of the separate foci, usually with some stromal productive reaction, to produce a single gross tumor when the phase of clinical (palpable) recognition is reached. In a few instances, widely separated areas of parenchyma undergo simultaneous cancerization and formation of multiple gross tumors, most often observed in lesions developing during pregnancy or lactation. In about 1 per cent of all patients, bilateral carcinoma is present on first examination. In patients with only a single lesion demonstrable by gross examination, if scores of microscopic sections are examined from the remainder of the breast, other microscopic foci of carcinoma will occasionally be demonstrated (7 per cent). Further evidence of the biologic abnormality which permits the genesis of mammary carcinoma, is the subsequent development of a second carcinoma in the remaining breast in 5 per cent of women surviving mastectomy for five or more years. In fact, the only mammary pathologic condition which appears with any frequency as a recognizable precursor of carcinoma is mammary carcinoma.

The interval of time between the inceptive phase of preinfiltrative neoplasia and its achieving such size as to be clinically palpable (1 cm. or even less in diameter) is

unknown though it must be highly variable. It seems certain that in at least a few patients, the preinvasive phase is of long duration. In the average lesion available evidence indicates that from the phase of earliest infiltrative growth to that of a space-occupying tumor is at least six months and more probably twelve or more months.

After reaching three-dimensional size, progressive extension into adjacent mammary tissue usually occurs and most often in an irregular pseudopod-like fashion producing a highly irregular surface and poorly demarcated edge. The rate of growth is not an exponential of time periods of active local growth alternating with intervals of little or no increase in extent. With advance of the lesion the skin becomes attached, infiltrated and eventually ulcerated, with subsequent secondary infection, necrosis and hemorrhage. Deep extension becomes attached to the pectoral fascia, which usually acts as a barrier to further direct spread, but extension through fascia and muscle may result in solid fixation to the thoracic cage. In the area of the axilla, the neoplasm may extend directly into and blockade the axillary space.

Notable variations from this average of local growth and extension are seen. In some fortunate patients, a dilatatory progressive ductal process will require several years to produce a palpable mass or to appear clinically as Paget's disease. In such women, dissemination of the disease beyond the breast is long delayed or may never occur. In lobular (acinar) carcinoma and in the more favorable variants of ductal carcinoma (medullary carcinoma, papillary cystadenocarcinoma and comedocarcinoma) the tendency is toward a walled-off or even pseudo-encapsulated tumor which may be bulky but is not actively infiltrating. Such neoplasms are expansile rather than infiltrative. Acute, or inflammatory carcinoma represents a florid overwhelming variant of infiltrative carcinoma, with multiplicity of origin, rapid extension and the circulatory congestive changes peculiar in such degree, to this lesion.

A rare variant in local growth is one that may be of great diagnostic difficulty: the primary carcinoma of the breast which remains occult while disseminating widely. In some of these patients, the original lesion can be found only by serial sectioning of the breasts at autopsy. Less striking, but more frequent, are women whose first symptoms are those of either regional or distant metastasis in whom the primary lesion is so small in size



Figure 8 Distribution of carcinoma in various parts of the breast.

The term *Paget type* is employed here to designate those ductal carcinomas developing in the central third of the breast, in which the first manifestation of the disease may be the development of scaly indurative, erosive or actual ulcerative changes of the nipple and areola, the lesion first described by Paget. Such involvement of the areola is invariably a surface manifestation of an underlying ductal carcinoma, even though no tumor may be palpable. In some instances the same type of ductal carcinoma may be present without involvement of the areola. This type of mammary carcinoma may remain limited to the central ductal area for varying periods before metastasis occurs.

The most ominous type of mammary carcinoma is that referred to as *acute* or *inflammatory carcinoma*. The lesion is a highly anaplastic variant with a rapid rate of growth and wide lymphatic embolization at an early stage of the process. Obstruction of the subdermal lymphatics by tumor emboli produces congestion with erythema and commonly some degree of lymphedema of the overlying skin. In some instances, no definable three-dimensional tumor is present, as the rapidly advancing neoplasm produces a diffuse indurative change in the consistency of the affected portion of the breast. The only excuse for recognizing this as a separate form of mammary carcinoma is the ominous prognostic implication, for this is a surgically incurable form of the disease. Acute carcinoma is more often observed in premenopausal women and is particularly frequent in those carcinomas developing during pregnancy or lactation. It is largely for this reason that carcinoma of the breast in young women is believed by many to be of uniformly bad prognosis. The fact is that, excluding those carcinomas developing during pregnancy or lactation, the prognosis of mammary carcinoma in patients under the age of thirty is somewhat more favorable than for those of all other age groups combined.

Carcinoma of acinar origin. Approximately 5 per cent of mammary carcinoma exhibits histologic features consistent with an acinar origin and these lesions are designated as *lobular carcinoma*. Most often they are slowly growing neoplasms which usually have attained some considerable size when first seen. They have a distinct predilection for long-delayed metastasis to regional nodes. This neoplasm is seen more commonly in younger women than in those of the older age group. It typifies an occasional

situation where the clinical setting appears highly unfavorable by reason of a large primary lesion of long duration yet it has a far better prognosis than the average, indifferent mammary carcinoma of small size and short duration. Experience has demonstrated that in situ lobular carcinoma should be treated by simple mastectomy rather than local excision.

Grading of mammary carcinoma. Apart from recognition of the 20 per cent or more lesions of special types described above, the pathologist cannot grade carcinoma of the breast with the prognostic accuracy he achieves in most carcinomas of squamous or epidermoid variety. The histologic inconsistencies are well illustrated by the medullary variant in which an "anaplastic" microscopic appearance is belied by a favorable natural history. The pathologist's recognition of these variants frequently is based on a combination of gross and microscopic features. For the remaining majority of carcinomas it is possible by histologic criteria, to label a few as low grade in degree of malignancy, and another small group as having a high grade of malignancy because of striking anaplasia. These relative extremes of histologic architecture account for probably not more than 10 per cent or 12 per cent of the nonspecific carcinomas. Thus a major fraction of some 65 per cent are processes which the pathologist can identify only as carcinoma, possibly using the word "ductal" to indicate probable epithelial cell type. In these instances information of more prognostic value can be secured from the pathologic extent of the disease and the combined facts of history and physical examination, all of which permit a limited evaluation of the biologic pre-determinism inherent in the host-neoplasm relationship for each individual patient. A further reflection on the limitations of microscopic grading is that, even in the few mammary carcinomas histologically separable as high or low grade in degree of malignancy the subsequent course of the disease occasionally belies the histologic assumptions.

Patterns of local growth, regional and distant metastasis. A distinct excess of carcinoma is found in the upper outer quadrant of the breast, over 40 per cent originating in this quadrant. Next most frequent are the lesions of subareolar origin, accounting for over 20 per cent. In rapidly diminishing sequence of situational frequency are the upper inner lower outer and lower inner quad-

planation for the areas of skeletal involvement is a spread through the paravertebral venous system. The same route probably accounts for cerebral metastasis as it does for cerebral involvement in bronchogenic carcinoma and pulmonary abscess.

The variegations in patterns and timing of the appearance of metastasis are inexplicable. Activation of skeletal metastasis may occur thirty or more years following radical mastectomy. In some patients a solitary focus of metastasis will become evident, particularly in bone, one month or years after primary treatment, and will come under prompt control by palliative management, with a subsequent period of years of clinical quiescence ensuing. At the other extreme, innumerable sites of metastasis may erupt with explosive biologic force after apparently successful treatment, with lethal effect in months or even weeks.

Finally, there are not infrequent instances of spontaneous arrest and regression of metastatic mammary carcinoma, even when widespread. In some instances, the phenomenon is due to a "biologic adrenalectomy" or total replacement of the adrenal glands by metastasis. More often, spontaneous control is of short duration and due to inexplicable changes in host-tumor relationship.

Biologic predeterminism—the determinant of natural history. The traditional philosophy of surgeons and other clinicians in respect to the curability of cancer in general in past decades has been epitomized by an almost ritualistic slogan, "early" diagnosis and "early" treatment. "Early" has carried a dual implication—that if the elapsed time from first recognition of the primary neoplasm to initiation of treatment is short, the extent of the disease is more likely to be limited and the prognosis more favorable. The more extreme adherents of this form of clinical evangelism have reduced the natural history of cancer to a situation of enviable simplicity: the extent of a neoplasm is in direct ratio to the lapse of time since recognizable onset of symptoms or signs.

Actually, there are a few forms of cancer in which such a direct spatial-time relationship exists, but mammary carcinoma is one of a number of major human cancers which provides convincing refutation of such clinical

pragmatism. As indicated previously, there are rare instances in which a primary carcinoma of the breast never becomes of such size as to be clinically discoverable while the occult focus disseminates widely with lethal effect. In such an extreme instance as far as the original source of disease is concerned, there is zero-delay on the day of death in terms of ritualistic adherence to "early" diagnosis. Or the patient's initial symptom may be pain due to skeletal metastasis from a carcinoma of the breast so insignificant in size as to escape any but the most meticulous examination.

At the other extreme of the biologic scale are the mammary carcinomas so dilatory in growth, so late in development of metastatic potential that simple mastectomy within three years of recognition of a local abnormality usually could achieve a cure if one could recognize clinically such biologic sluggishness of growth. About 20 per cent of all carcinomas of the breast make up this group of biologic sluggards of neoplasia. Reference to Table 3 will indicate that a substantial portion of the slow-growing neoplasms are pathologically recognizable as processes of more favorable prognosis. While the variants so indicated in Table 3 add up conveniently to 19 per cent, it is proper to recognize that not all of these neoplasms actually pursue a more favorable course. Also some of the genuinely slow growing carcinomas are of nonspecific type, and no clinical or pathologic criteria are available to characterize these less ominous, neoplastic mavericks.

Mammary carcinoma provides the most variegated pattern of human cancer of any major organ site. Relative accuracy in evaluation of "early diagnosis" of the disease can be secured only by determining the total extent of involvement in terms of time (duration) and local space-occupation (size of primary lesion).

Of these two factors, duration is least reliable, for obvious reasons common to most historical data. With an adequate sample however such data have some validity. The more objective factor is provided by actual measurement of the primary neoplasm in a series of surgical specimens provided by radical mastectomy. Both of these factors of "earliness" are most readily tested by correlation with the presence or absence of axillary nodal metastasis in the same surgical specimens for this is the important single prognostic criterion in operable lesions.

When the axillary nodes are without evi-

The expressions "curability" and "cure" are used in their absolute sense, i.e., the survival of the patient following definitive treatment until her death from some unrelated cause and without demonstrable evidence of residual or recurrent carcinoma by autopsy.

as to be detectable only by careful examination.

Secondary changes within mammary carcinoma are most dependent on rapidity of growth and size and are proportional to both. They include areas of hemorrhage zones of necrosis and secondary infection.

In a few patients, satellite cutaneous nodules develop from subdermal lymphatic tumor emboli, an ominous sign.

REGIONAL METASTASIS. Axillary lymph-nodal metastasis has been of predominant interest for two reasons: one anatomic and the other clinical. With the largest group of carcinomas originating in the upper outer quadrant, initial regional metastasis most frequently occurs through the axillary (principal) pathway to the axillary and retropectoral nodes. Carcinomas primary in the lower outer quadrant have a similar predilection for axillary spread. Even the lesions primary in the medial hemisphere may take the axillary route of earliest dissemination; those in the upper inner quadrant are as apt to take the axillary as the internal mammary pathway while those of the lower inner quadrant more often spread by the latter route. The primary neoplasms of subareolar origin are unpredictable and may, as seems logical, spread through the subareolar lymphatic plexus.

From a surgical standpoint the standard procedure of radical mastectomy produces only the axillary group of lymph nodes for histologic study. The presence or absence of demonstrable involvement of axillary nodes determines the final (pathologic) staging of the operable case and also offers criteria of considerable value in prognosis.

Current investigative studies of the value of internal mammary node dissection, in addition to conventional mastectomy have focused interest on the frequency of metastasis in these superficially located, intrathoracic nodes. Of those carcinomas originating in the medial quadrants and operable over 50 per cent have set up such deposits, while about 20 per cent of those in the lateral quadrants have so disseminated.

The subclavicular nodes are least commonly involved of all the regional nodes and such spread is usually concomitant with supraclavicular nodal deposits.

It should be emphasized that both the supraclavicular and other cervical nodes, as well as the internal mammary chain, are not regional, but distant nodes in the true sense, both by anatomic and biologic considerations.

The mechanism of lymphatic spread is usually by embolization, seldom by permeation.

DISTANT METASTASIS. For those instances in which a sequential sort of spread is the pattern, axillary nodal involvement is followed by supraclavicular deposits, then by mediastinal nodal metastasis. Or through the internal mammary pathway direct spread to the pleura and perhaps lymphangitic pulmonary involvement represent an anatomic sequence.

The vagaries of remote dissemination are such that any orderly progressive sort of spread is seldom demonstrable. From a practical, clinical standpoint, *it is essential to remember that any spread beyond the true regional nodes is almost invariably an indication of generalized disease.* Thus metastasis in a single supraclavicular node, histologically proved, is as ominous as spread to other more distant sites. It seems equally certain that occupation of a single node in the internal mammary chain is of similar import. The dissection of such nodes will probably produce no increase in curability and little in the way of palliation.

Of greatest importance to the clinician is a knowledge of the most frequent sites of distant metastasis, so that an intelligent search may be made both in presumably operable, primary cases and in the follow-up of postoperative patients. From combined information provided by clinical and autopsy studies distant sites may be listed in the following order of descending frequency:

Mediastinal nodes, lung and pleura

Bone

Liver peritoneum

Adrenal

Brain

Ovaries

It is also helpful to have in mind the sites of predilection of skeletal metastases which by order of diminishing frequency are

Ribs

Lumbar spine, pelvis and upper femora

Dorsal, cervical spine

Skull

Upper humeri and scapulae

Skeletal metastasis from carcinoma of the breast is predominantly osteolytic, although some osteoblastic reaction is common at the advancing periphery of bony lesions. Occasionally osteoblastic metastasis is seen, though rarely is it as sclerosingly emburated as that common to deposits of prostatic carcinoma.

It is of interest that the only rational ex-

less secure basis one may speculate that the determining factors governing neoplastic aggressiveness or host resistance may reach back to the genetic inheritance of the host. This is to say that the behavior of a neoplasm is determined by genetic factors.

From the clinical standpoint it seems obvious that the growth pattern is established long before the symptomatic stage is reached and thus for practical purposes is predetermined. The growth potential of the neoplasm, arrayed against whatever defensive reactions are excited in the host, even tuates in a biologic complex which determines the natural history of the disease. For the clinician, this constitutes biologic predeterminism.

From all of this, it is apparent that biologic factors, more than the time of treatment, the size of the neoplasm or even the type of treatment, most profoundly influence prognosis. This does not mean however that reasonably prompt, effective treatment is without importance to the woman with carcinoma of the breast, both for curative and palliative purposes.

Indicated in Table 5 is an estimated distribution of 100 patients based on factors inherent in biologic predeterminism. Twenty individuals would have a favorable result, usually curative in effect, even with something short of ideal treatment, at any time

up to three years after onset. About fifty five of each 100 have neoplasms of high growth potential and inadequate host resistance in whom regional or distant metastasis is established before the primary lesion is clinically apparent. In the remaining twenty five there is the relationship between time, space-occupation and metastasis implied in the traditional concept of "early" diagnosis. For these twenty five women the extent of their disease is indeed a function of time. It is for this group that not only prompt, but adequate, effective treatment may mean the difference between cure and failure between life and an early death.

These concepts of mammary carcinoma lead to the following conclusions:

Only in some twenty five of each 100 patients will "early" treatment, by detection of a small tumor of short (known) duration contribute to curability.

In seventy five of each 100 patients, curability is established by natural history (biologic predeterminism) not by factors of duration or local spatial extent. Twenty of such patients are readily curable, fifty five are incurable, and for biologic reasons in both instances.

Clinical evaluation of curability which is to say operability should not be influenced by duration or the size and extent of the primary neoplasm. Some lesions of long

Table 5 Natural History of Mammary Carcinoma

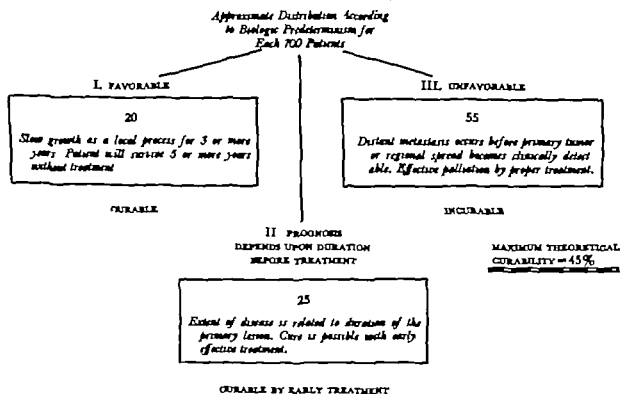


Table 4. *Mammary Carcinoma—Incidence of Stages I and II by Known Duration of Primary Tumor*

DURATION (MONTHS)	STAGE I (PER CENT)	STAGE II (PER CENT)
1 month or less	55	45
>1-6 months	35	65
>6-12 months	48	62
>12 months	22	78

dence of metastasis by histologic examination of all discoverable nodes, the neoplasm is from the standpoint of the surgical specimen, limited to the breast and designated as stage I P (pathologic as compared to clinical staging). If axillary nodal metastasis is proved histologically the process is stage II P.

The relative incidence of stages I P and II P is related to the known duration of the tumor in Table 4, based on data from several sources. It is obvious that lesions of very short duration (thirty days or less) have a distinctly better prognosis than do long-neglected tumors: the relative incidence of favorable (stage I P) cases being 55 per cent and 22 per cent respectively. However after a delay of one month, there is no significant change in the incidence of axillary extension between the remainder of the first six-month period and the next full period of six months.

For successive increments in size of the primary carcinoma, as shown in Figure 9 a comparable situation is demonstrated. With carcinoma of the breast just within the range of palpability (1 cm diameter or less) the prognosis is most favorable, but even in such very "early" neoplasms, over 50 per cent have already seeded into one or more axillary nodes. The obverse of this biologic coin of chance again reveals that fraction of less ominous growths when the primary tumor has achieved the impressive dimensions of more than 5 cm. diameter 23 per cent of such patients are yet without regional (axillary) nodal metastasis. The progress in spatial extent from more than 1 to 3 cm. and on to 5 cm., is a period of growth during which no change in incidence of regional metastasis is evident.

One reason for this seeming paradox is that the more rapidly growing, more ominous neoplasms tend to provide the patient with

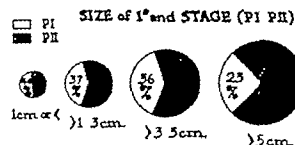


Figure 9. Relation of size of primary carcinoma of breast to involvement of axillary nodes by microscopic examination (stage I P nodes negative; stage II P nodes involved)

more disturbing evidence of local or regional abnormality than do the less aggressive tumors. Such striking signs as rapid local growth, erythema or edema of the skin or a large node in the axilla bring most women with unfavorable lesions to treatment in the first few weeks or months after onset. More of the group with slowly progressive tumors, procrastinate and after many months of delay may still present evidence of local disease only. Thus the "early" cases of short duration include a disproportionate number of unfavorable cancers obscuring the true value of "early" treatment for some patients.

The natural history of untreated carcinoma of the breast now becomes easy to comprehend when considered in the light of the foregoing information. From several sources, the percentage of patients surviving without treatment are, in round figures as follows:

YEARS	PER CENT
1	75
3	40
5	20
10	5

Here again, the figure of 20 per cent survival at five years is most impressive, pointing once more to that fraction of dilatory neoplasms of low growth potential and little or even no capacity for metastasis.

The sum of this evidence constitutes inexorable proof of a host-neoplasm relationship by which a balance of power is established between the invaded and the invader. The biologic factors involved in this struggle for survival are unknown, but the evidence also indicates that the struggle is decided in the preclinical phase of the disease and probably during its earliest inception. On a

involvement, and evidence of low growth potential.

- B More extensive axillary and especially apical nodal involvement*
 Axillary nodes, bulky or adherent to each other or to the skin.

Lymphedema or erythema of the mammary skin, including "inflammatory" carcinoma.

Other evidence of a pattern of rapid growth.

III. All other cases Regionally remote or distant metastasis

This group includes patients with

Fixation of the breast to the chest wall

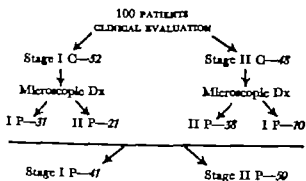
Metastasis to supraclavicular nodes

Contralateral axillary metastasis

Those with distant metastasis

Clinical staging of lesions in operable cases is only a working evaluation of the disease in respect to axillary metastasis. The histologic determination is the final diagnosis. Where differentiation of the two is necessary a capitalized C or P is used, for Clinical or Pathologic Staging

The inaccuracy of clinical evaluation by competent individuals is apparent in numerous reports, of which the following data are representative. For each 100 women with carcinoma of the breast undergoing radical mastectomy clinical impression concerning axillary nodal disease will be in error to the extent indicated below



The clinical error is greater in the direction of inability to detect lymphadenopathy found by gross or microscopic examination. In brief the experienced clinician's "false negative" evaluation of axillary nodes is 40 per cent, while his "false positive" error is only 15 per cent. The usual cause of a "false positive" conclusion is the presence of large nodes secondary to inflammatory reaction.

In a few centers effort toward securing

more accurate information on the extent of the disease is pursued by obtaining lymph nodes for biopsy from the scalene and internal mammary areas, prior to radical mastectomy. If such nodes are found to contain metastasis, palliative treatment is employed.

Initial clinical investigation. The patient with a tumor of the breast which may be carcinoma deserves careful preliminary study the extent of which will be indicated by symptoms and physical signs. The reaction of many physicians toward an undiagnosed lump in the breast is as emergent as that of a fireman toward a blaze in a gasoline refinery. From what has been said above concerning its natural history it is obvious that carcinoma of the breast does not metastasize overnight or for that matter in a fortnight. The "fire engine" approach to the undiagnosed lump in the breast is more apt than not to be a disservice to the patient, both in respect to proper management and the patient's psychologic reaction.

The following information, all of which is relevant to phases previously described in detail should be secured

1. Known duration of tumor
2. Observation of change in size, over what interval
3. Observation of redness of skin, change in size of breast
4. Change in appearance of nipple or discharge therefrom
5. If premenopausal, whether the breast is sore or lumpy premenstrually and the present phase of the cycle
6. Fertility lactation and abnormalities thereof
7. Previous operations for mammary or pelvic pathologic conditions
8. Family history of mammary carcinoma
9. Inquiry concerning symptoms which might indicate metastatic involvement in common sites

a. Persistent cough, or dyspnea (mediastinal, pulmonary)

b. Pleuritic type of pain (pleura, ribs)

c. Pain of recent origin or altered character particularly if persistent, which may be referred to sites of bony metastasis (ribs, spine, pelvis)

d. Headache, vertigo or visual disturbance (brain)

In addition to meticulous survey of the breasts and regional areas general examina

duration with extensive local lesions are curable

Effort should be made, in each patient, to evaluate the biology of the lesion in terms of historical (approximate rate of growth) and physical (local, regional and distant) indications of curability or incurability

If it were possible to provide the most effective treatment now possible for all patients with a minimum period of delay (one month or less) the maximum theoretical curability would be 45 per cent.

Stages of disease Staging is the effort to express the extent of the disease and is of vital importance for it determines both treatment and prognosis. There are two approaches to staging, clinical and pathologic.

I Clinical stages Every primary (untreated) tumor of the breast which may be carcinoma and apparently operable should be regarded by the clinician preoperatively as cancer and such survey methods as may be indicated are employed for evidence of possible metastasis. Clinical staging is highly inaccurate even in the most competent of hands.

2 Pathologic stages The pathologists' effort in determining spread of disease is more accurate than the clinician's but usually is limited to a study of the operative specimen from operable cases. Most often, of course the specimen is the product of radical mastectomy from which the most important prognostic information secured is the presence or absence of axillary, nodal metastasis. Not less than fifteen and up to thirty or more axillary nodes should be found by careful search, without clearing of the specimen, after an adequate axillary dissection. The prognostic value of the findings can be increased considerably if the surgeon tags the axillary apical portion of the specimen during the dissection. The axillary contents then become divisible into three approximate areas:

Level I--Below the tendon of the pectoralis minor

Level II--Posterior to this structure

Level III--Apical area, above the pectoralis minor

The prognosis becomes progressively worse with the demonstration of nodal metastasis from level I to level III

Occasionally histologic study is necessary to determine spread of disease beyond the area of conventional radical mastectomy most frequently through excisional biopsy of suspected supraclavicular nodes, contralateral axillary nodes or skin nodules

The most valuable and most logical clinical classification of any cancer when its mode of dissemination permits is that which expresses three possible stages of advance of the disease

I Limited to primary site

II Regional nodal involvement apparently present

III Distant metastatic foci are demonstrated beyond reasonable doubt

In some situations as in carcinoma of the breast some subdivision of regional spread to indicate degree of such extent is useful and practical. For clinical usefulness adherence to the basic concept of three stages seems of obvious value although some classifications of more complex design, such as Portmann's, are perhaps more accurate and are widely employed.

The deficiency of any clinical staging is that it represents only the demonstrable extent and location of disease at the moment of evaluation. It gives no hint of the kinetic phase or the growth potential of the neoplasm. For example, two patients may exhibit primary tumors of the same size, and comparable degrees of axillary lymphadenopathy yet in one there will be historical information indicating no change in size or extent in six months while in the other the mammary tumor has doubled in size in three months and the nodes have developed in a matter of weeks. If the history of the latter patient is underscored by objective findings of edema or erythema, one is confronted with an entirely different neoplasm, biologically considered, than in the other patient, even though the spatial occupation by neoplasm is nearly identical.

Incorporated in the following outline of clinical staging are some considerations of biologic variability in stage II. This outline has been used for some years at the Los Angeles County Hospital.

Clinical stages

I Disease limited to the breast

May include patients with minimum changes in the skin (early retraction) without actual cutaneous infiltration.

II Primary lesion of breast with clinical evidence of metastasis to axillary nodes or more than minimum involvement of skin, or both

A. Axillary nodes are discrete. No axillary lymphadenopathy

Cutaneous involvement with or without ulceration, but concomitant with little or no axillary nodal

diagnostic measure, prior to palliative radiotherapy or chemotherapy.

Its success is entirely conditioned on the interest and cooperation of the pathologist. This implies either an interest in cytology or attention to detail in the fixation and subsequent processing of the slivers of tissue submitted. In contrast to routine procedure for larger blocks of tissue.

CLINICAL LABORATORY PROCEDURES. Unfortunately there is as yet no specific test for any type of cancer. Nor do any of the current efforts in refinement of nonspecific serologic reactions offer sufficient promise of relative accuracy for routine clinical use.

The *sedimentation rate* in the absence of concomitant and particularly inflammatory disease, is of some value as it is usually elevated in mammary carcinoma if the primary lesion is bulky or if there is more than minimum axillary nodal spread. Remote metastasis is almost invariably accompanied by highly abnormal rates.

While not as valuable an indication of skeletal metastasis as is acid phosphatase in prostatic carcinoma, the alkaline form of this enzyme is present in concentrations above the average range in some six out of ten women with bony metastasis. When a patient with a known carcinoma of the breast has symptoms consistent with such metastasis, consistently elevated levels of *serum alkaline phosphatase* on two or more determinations are almost confirmatory of skeletal or hepatic metastasis, regardless of roentgenographic findings in respect to bone detail.

The woman who faces the possibility of an operative procedure for carcinoma of the breast requires both physical and psychologic evaluation.

Physical evaluation. Only severe concomitant disease or extreme senility and deterioration should interfere with the choice of surgical treatment. In women with cardiovascular disease, adequate medical work up is indicated, but rarely should it constitute a contraindication to radical mastectomy. Careful medical management before, during and after operation is, of course, mandatory. The same attitude should hold for patients with diabetes or other major medical problems.

Age, as in any other major surgical venture, is more a matter of biology than chronology. Some women of sixty-five are more senescent than others a decade or two older. The mere fact that a woman has survived

past the biblical three score and ten is usually evidence of a biologic fortitude which will carry her safely through a radical mastectomy no matter how frail she may appear. We have had an experience of two patients past ninety years of age, both of whom had a smooth convalescence following a hurried but radical mastectomy, one of them surviving four years to die of coronary thrombosis without clinical evidence of recurrence.

Psychologic evaluation. To many women the prospective mutilating effect of mastectomy seems far more drastic and much more difficult to accept than the most extensive sort of intra-abdominal operation. To such women the breasts are more closely identified with physical integrity than are any other structures save those of the head and neck. Such an attitude is not peculiar to younger women or to those whose physical attributes would command admiration.

In any woman there will be with rare exceptions more than a little reluctance to face the situation. Curiously enough, the problem is apt to be less difficult when the patient has had prior though unrelated, major operations.

For his own protection the surgeon must inform the woman with an undiagnosed tumor of the breast that a mastectomy may be required, no matter how remote the possibility may seem. However in most patients it seems desirable to create the impression that the necessity of a radical operation is unlikely even in the presence of almost certain clinical signs to the contrary: an unverified clinical diagnosis of cancer is psychically damaging to the patient and hazardous for the clinician.

When the diagnosis of cancer is established, the frankness with which the situation is discussed, or the evasions which are sometimes almost mandatory must depend on the clinician's attempt to evaluate the emotional status of each patient, with help from the family when available. The clinician's effort in psychotherapy, amateurish though it may be, is as integral a part of management as the technical details of definitive treatment. Large doses of optimism warranted or not, are salutary adjuncts to physical therapy.

Methods of Treatment. Since the early years of this century radical mastectomy as originally described by Willy Meyer and Halsted, has been the definitive therapeutic measure for operable carcinoma of the breast. In the past twenty years a growing

tion occasionally shows signs of possible importance in relation to metastatic sites.

Hepatomegaly

Ascites

Neurologic, such as Horner's syndrome due to pressure on the cervical sympathetic chain by enlarged, deep cervical nodes, or signs of cranial neuropathy from cerebral metastasis

Ovarian mass

Abnormal signs in examination of the chest with advanced intrathoracic disease are relatively unimportant, as the roentgenogram of the chest is far more accurate

RADIOGRAPHIC STUDIES. The absolute preoperative requirement in every patient with an undiagnosed tumor of the breast is roentgenograms of the chest, preferably anterior stereoscopic and lateral views. Some clinical authors emphasize routine skeletal surveys but the yield of positive findings is so low in primary asymptomatic cases as to be entirely out of proportion to the burden imposed on the radiologic laboratory and the cost involved. If the tumor is of questionable operability a simple form of bone survey should be done. This may consist of a large (14- X 17 inch) anteroposterior film of the lumbosacral spine and pelvis and a similar large film taken laterally. Providing the previously mentioned roentgenograms of the chest have been made with adequate penetration, the ribs and thoracic spine are reasonably visible. If the patient has symptoms which may be due to skeletal metastasis, additional appropriate studies should be ordered.

It should be remembered that the radiographic method is a study in shadows and considerable destruction of bone may be present without evidence of radiographic abnormality even with technical work of high quality and expert interpretation. It has been estimated, by study at autopsy, that two-thirds of the diameter of a vertebral body in the exposed plane is usually destroyed before the metastatic focus becomes apparent on the roentgenogram. Clinical evaluation of pain is thus of great importance: persistent, localized pain of increasing intensity with the added evidence of elevated serum alkaline phosphatase may be as significant as a questionable area in a roentgenogram.

NEEDLE BIOPSY. The theoretical dangers of needle biopsy are entirely speculative and several reports concerning long-term follow up of many patients with cancer of the

breast subjected to the procedure fail to indicate any adverse influence.

The value of needle biopsy is limited to those instances in which the material secured is diagnosable as cancer either by histologic or cytologic techniques. *Negative findings are valueless.* When carcinoma is determined by needle biopsy, the procedure obviates the necessity of a preliminary biopsy and rapid frozen section in the operating room reducing the duration of anesthesia and operation by fifteen to thirty minutes.

For almost fifteen years the resident staff in Surgery and Radiology of the Tumor Surgery and Radiation Therapy Services of the Los Angeles County Hospital has employed needle biopsy as a routine measure in tumors of the breast clinically consistent with carcinoma. The relative simplicity of the technique is evident from the fact that after a month or so of experience, performing several needle biopsies weekly the residents usually obtain samples as satisfactory as those secured by more experienced individuals.

Needle biopsy may be done by one of two methods

Aspiration of the tumor by a large-caliber needle and cytologic study of smears from the aspirated material

Securing a core of tissue from the tumor by one of several special needle-type devices of which the most commonly used is the Silverman needle. The core of tissue is placed in fixative and processed by histologic technique

Of the two methods the histologic method is preferred by most pathologists.

Our considerable experience, as indicated above permits these conclusions concerning needle biopsy

Successful use of the technique requires reasonably frequent practice. He who tries a needle biopsy several times in a year is likely to have a low opinion of the procedure. Unless one can use needle biopsy weekly rather than monthly it is better to rely on open, surgical biopsy.

In tumors subsequently established as carcinoma, needle biopsy should produce a positive report seven out of ten times.

It is preferable to limit the technique to tumors clinically suspect and 2 cm. or more in diameter.

It is of greatest value in confirming a diagnosis of carcinoma, for the record, in patients with inoperable disease and without superficial lesions suitable for conventional biopsy. It is, therefore a highly appropriate

If the biologic concepts enunciated are valid, by which supraclavicular node involvement is as ominous as more distant metastasis, such surgical heresim may be only of palliative, certainly not of curative, value.

Extreme pessimism has been the keynote of recent writings of some biometricians who maintain that no difference in survival statistics is reflected following any method of treatment. Such conclusions are iconoclastic both by the statistical methods employed and the failure to differentiate between survival of long duration with and without disease.

All of these viewpoints are inspired by a growing recognition of the inadequacy of presently available treatment for mammary carcinoma. Nevertheless until more effective, physiologic or chemotherapeutic methods become available, conventional radical mastectomy remains the definitive measure for operable, possibly curable cases. The clinician's primary responsibility then is to separate *operable* from *inoperable* cases. In operability may be established either on technical or biologic considerations, an effort which is attended only by relative success.

Referring now to the clinical classification of mammary carcinoma previously outlined, stages I and III present no problem. Stage I without clinical evidence of spread beyond the breast, requires immediate radical mastectomy. Stage III, with extension beyond the homolateral axilla, is eligible for palliative treatment.

The varying degrees of local growth and axillary nodal involvement constituting stage II may tax the judgment of the most experienced clinician. Combining considerations of physical findings and attempts to assess the rate of growth, the classification employed here divides stage II lesions into two categories:

IIa. Eligible for immediate radical mastectomy

IIb. Categorically inoperable

The primary problem in stage IIb cases is to filter out some lesions which, though clinically unfavorable especially in relation to the extent of the primary lesion, may nevertheless secure a better palliative and even an occasional curative result by radical mastectomy.

This filtering process can be accomplished with a fair degree of accuracy by employing irradiation as a therapeutic test. In general, radiosensitivity indicates biologic incurability and, conversely, radioreistance may mean a more favorable prognosis in a clin-

ically extensive process. At least failure of a stage IIb lesion to regress with a moderate dosage of irradiation will indicate that better palliation will be secured by operative measures either simple or radical mastectomy.

In practice the therapeutic test of radiosensitivity in stage IIb cases may be employed as follows. The primary lesion is treated through two opposing portals: the axillary nodal disease through a single direct field. Daily treatments are given to a total of 1600 r to 2000 r tumor dose to the primary (breast) lesion and 1000 r to the axilla, in a period of fourteen to eighteen days. At the end of this trial period, the primary radiosensitive carcinoma should show a 40 per cent or more regression by measurement, with similar reduction in the size of axillary nodes. This degree of radiosensitivity indicates genuine incurability as well as the probability of satisfactory local and regional palliative control by irradiation; a full course of roentgenotherapy is then employed. Failure to respond to the therapeutic test usually is an indication of a less ominous growth pattern and radical mastectomy may be undertaken with suitable additional postoperative irradiation depending upon microscopic findings in the removed axillary nodes.

Histologic confirmation of the diagnosis of carcinoma should precede any therapeutic test of radiosensitivity, even though the clinical evidence seems unmistakable. Stage IIb cases, owing to the extent of the disease, usually present areas of involvement permitting biopsy under local anesthesia if the clinician does not employ needle biopsy.

Surgical biopsy. The surgical approach to undiagnosed tumors of the breast whether clinically suspected of cancer or not, should be made with the patient under general anesthesia by a surgeon who is competent in radical mastectomy with the stage set for the latter procedure if it is indicated. The key figure in this setting is the pathologist, who must be skilled in the preparation and interpretation of frozen sections. An experienced surgical pathologist can make an exact frozen-section diagnosis in a high percentage of breast lesions, but such accuracy is a product of training, experience and, above all, interest in assuming this responsible role. Failing these requirements, either because of disinterest or absence of a full time pathologist in smaller hospitals, it is best to do excisional biopsies and await the preparation of paraffin sections. If the surgeon has had adequate training in gross

recognition of unfavorable biologic factors, such as outlined by Haagensen and Stout, has emphasized a group of patients technically operable but categorically inoperable. Still more recently the therapeutic pendulum has swung in two opposite directions. One philosophy represented by the work of McWhirter and his associates at Edinburgh, favors operative treatment by simple mastectomy followed by intensive roentgenotherapy to the regional lymph nodal areas. The end results after five and ten years reported by McWhirter are equal to those after radical mastectomy but two practical objections to his procedure are apparent.

The level of dosage (3700 r tumor dose in three weeks) employed by McWhirter is far more intensive than that customarily employed by American radiologists. In actual practice only a small minority of radiologists in the United States whose major or exclusive interest is in radiotherapy will be willing to employ the intensive treatment and meticulous planning which are required if the results reported by McWhirter are to be achieved.

External irradiation of this intensity regardless of the quality of the beam, results in some sequelae in a fair percentage of patients, the hazard of which becomes greater with survival beyond five years after treatment. Although the incidence and severity of pulmonary fibrosis are considerably less than were predicted, and although radiation osteitis usually is not of significant importance, the telangiectasia and atrophy of the skin are a source of distress in some women in whom such changes become advanced.

The main disadvantage of the McWhirter technique is the fact that every woman with carcinoma of the breast is subjected to the same intensive radiotherapy regardless of the absence or presence of involvement of axillary lymph nodes, which cannot be determined in the absence of axillary dissection. As has been described above at least 20 per cent of patients even with the lesions of largest local size and longest duration, still have no evidence of spread to the regional, axillary nodes. When the primary tumor is small (in the range from 1 to 1.5 cm.) 50 per cent or more of patients are without axillary spread. Thus, the uniform application of radiation therapy without actual knowledge of the presence of regional metastasis is an unnecessary exercise for somewhere between twenty five and fifty of each 100 women. It is equally ob-

vious that the routine use of radiotherapy is of no value in those patients in whom spread is by humoral rather than by lymphatic channels. The McWhirter technique also employs routine irradiation of the chest wall in the customary operative area of a radical mastectomy. Inasmuch as only 5 per cent to 15 per cent of women in most series of cases exhibit chest wall recurrence, this also indicates an unnecessary amount of irradiation for about eighty five of each 100 patients. Most clinicians who employ postoperative x ray therapy following radical mastectomy prefer that the chest wall not be irradiated so that in the event of local recurrence there will have been no impairment of skin tolerance for local irradiation.

Assuming that crude five-year survival following radical mastectomy with intelligent use of postoperative irradiation is equal to that reported for simple mastectomy and routine irradiation, radical mastectomy remains the preferable procedure in view of the preceding considerations. Two recent reports concerning significant numbers of patients do indicate that the standard radical mastectomy produces results equal to or superior to those reported from Edinburgh. In 1957 Watson reported on 629 patients treated in Saskatchewan from 1944 to 1949 and, although smaller in number than the group reported by McWhirter it is nevertheless a comparable experience, as it represents over 85 per cent of all the breast cancers in this Canadian province. The five-year crude survival was 48.3 per cent with radical mastectomy and almost routine use of postoperative irradiation, compared to 42 per cent for the Edinburgh series. In 1958 a report of 8396 proved cases for 1935 to 1953 in Connecticut (Ryan et al.) showed an improvement in five-year survival from 46.3 per cent for the first seven-year period to 51.0 per cent. There was a significant difference favoring the survival of patients treated by radical surgery over those treated by limited surgery.

The reverse of this approach is the super radicalism currently employed by some surgeons in the United States, in which the standard procedure of radical mastectomy is extended to include one or more of the following:

- Homolateral neck dissection
- Dissection of the internal mammary chain of nodes
- Contralateral mastectomy

The same may be said of axillary dissection, but one obtains thereby important prognostic information, as well as guidance for proper

Various incisions may be employed in radical mastectomy depending on the location of the primary lesion. When feasible, variants of the transverse (Stewart) or arrowhead (Greenough) incisions are more acceptable from a cosmetic standpoint than the Meyer Halsted type.

Irrigation of the operative area before closure with a solution of some chemotherapeutic agent has been employed to an increasing extent since 1957 although conclusive evidence of its value is not yet established. Various alkylating agents have been employed, but the substance which seems most promising for this purpose is Chlorpactin (monoxychlorosene).

Closure of the wound must be done without undue tension on the thin skin flaps. Split thickness skin grafts may be required, but in the majority of patients a more satisfactory closure is obtained by using the adjacent skin. This is accomplished by sliding flaps, usually from lateral and lower costal areas.

POSTOPERATIVE CARE. Postoperative care is concerned mainly with two important objectives: wound healing and function of the arm. Although the primary contribution to healing is wound closure with minimum tension, a departure from the customary occlusive type of pressure dressing has produced a distinct increase in primary healing. A no. 28 Robinson (hard rubber) catheter with four or five additional openings cut into the distal 8 cm. of the tube, is inserted through a stab wound below the axilla in the posterior axillary line for dependent drainage. The tube is fixed to the skin with a single suture, which should also make the stab wound airtight. Negative suction through the tube is started in the recovery room or even in the operating room if there has been more than an average amount of generalized bleeding. Negative suction is applied, preferably through a power-operated source such as the Pratt-Chaffin unit, maintaining a constant pressure of about -118 mm. of mercury, although the usual two-bottle Wengensteen suction can be substituted. This achieves early fixation and revascularization of skin flaps with far greater effectiveness than is accomplished by pressure dressings. Suction is maintained for three to five days or until the amount of exudate recovered in the trap bottle is less than 100 cc. in 24 hours. On the second postoperative day the patient is taught to disconnect the Robinson catheter from the tube to the suction machine so that she

can have increasing periods of ambulation and no restriction in bathroom privileges.

Negative suction is employed and no dressing of any sort is used, as it has been demonstrated that complete exposure of the wound provides a combination of drying, cooling and electrolyte and protein environment which is most hostile to bacterial growth. Assuming that there has been meticulous edge-to-edge closure the wound becomes sealed in less than two hours. During this time a nonsterile bath towel may be placed over the wound to absorb the early exudate. In the recent experience of one group this combination of negative suction and exposure of the wound has produced better than 80 per cent primary healing in over 100 radical mastectomies. Antibiotics have not been employed and there is no conclusive evidence of their value as a routine postoperative measure.

Active motion of the arm should begin on the day of operation. The most valuable single exercise in prompt recovery of function of the arm is brushing the hair. The patient should have a hairbrush within reach constantly and be encouraged to spend much of her time using it. She should aim at being able to make the full sweep of the brushing motion on the opposite side of the head in five to seven days after the operation. More varied exercises are of value later.

Ambulation should begin on the evening of operation and increase daily.

CHEMOTHERAPY SUPPLEMENTARY TO RADICAL MASTECTOMY. Cole and his associates have provided convincing evidence that in animal experiments free floating cancer cells can be destroyed by chemotherapeutic agents given intravenously or intraperitoneally. The early results of controlled human tests, in which randomly matched postoperative patients who have or have not received such chemotherapy are compared, appear promising. It is still too early to evaluate the results.

Radiation therapy supplementary to radical mastectomy. This is a therapeutic issue with contradictory statistical evidence concerning its value. The issue is clouded by the reports of some surgeons who, with a lamentable lack of objectivity, order radiation therapy only for patients with stage II P cases with extensive axillary disease. Such bias results in comparing unfavorable with favorable series of cases rather than measuring the effectiveness of irradiation in all stage II P cases. The techniques of post

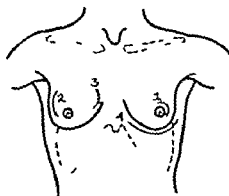


Figure 10 Incisions for exploration and biopsy. 1 Curved areolar for lesions in the central third. 2 and 3 Curved peripheral incisions. 4, Inframammary incision for deeply located tumors.

pathology he will be able to make a diagnosis on the physical features of the cut surface of at least seven of every ten carcinomas. Whether he proceeds with a radical mastectomy without histologic confirmation is his own responsibility, for there will be rare instances in which the most typical gross findings are not verified by histologic findings.

Incisions for tumors in different areas of the breast are indicated in Figure 10. Radial incisions should be avoided, for they are more prone to keloidal reaction and stretching than any other and are frequently sensitive, even painful. For exploration of the central third of the breast, including the ductal area, the curved areolar incision permits wide exposure by reflection of skin flaps and the resultant scar is almost invisible after a few months in most patients. An inframammary incision, to expose the under surface of the gland for a deeply located tumor is disadvantageous if a radical mastectomy is indicated.

For tumors with the physical criteria of a benign process, or of small size (in order of 1 cm. in diameter) excisional biopsy is preferable. In all instances, a margin of adjacent, apparently uninvolved tissue should be removed with the lesion, even though it may seem encapsulated. In tumors clinically suspect of carcinoma, incisional biopsy has theoretical advantages: fewer lymphatic radicles are divided and there is a lesser hazard of cancer cells being spread in the wound or on gloves or instruments.

TECHNIQUE OF INCISIONAL BIOPSY After preparation and draping of the area required for radical mastectomy the surgeon does a secondary draping to leave exposed only the site of incision. An adjacent drape is spread, on which the instruments em-

ployed are placed, to be discarded if cancer is present. Incision is made through the skin and directly down to the surface of the tumor. Retractors are placed to expose the lesion adequately and, with a scalpel, a wedge of the tumor is excised. If the gross appearance suggests carcinoma, the site of biopsy may be electrocauterized, hemostasis completed and the skin closed tightly while awaiting the frozen section report. If the tumor is not carcinoma, skin flaps are reflected and the entire lesion excised with a margin of adjacent tissue.

If carcinoma is confirmed, tight closure of the skin is followed by electrocoagulation of the line of incision. Biopsy instruments are discarded, as are the drapes around the incisional area and any wash basins used, gloves are changed and the entire operative area is repainted with antiseptic.

If rapid frozen-section diagnosis is not available, at least one study has indicated that there is little, if any influence on prognosis by first excising the tumor and performing radical mastectomy for carcinoma two or more days later. With either approach, the most important consideration is meticulous attention to the "aseptic" sort of technique outlined above for cancer cells can be implanted, even in donor sites while grafting skin. Some postoperative local recurrences are probably due to a similar mechanism.

Radical mastectomy This procedure requires a precise knowledge of anatomy and meticulous adherence to operative detail. Competence in the procedure is acquired only by training and experience. Cardinal requirements include

1. Wide reflection of thin skin flaps in the area of the primary lesion.

2. Reflection of skin flaps beyond the mid line anteriorly to the posterior axillary line posteriorly to the clavicle above and the upper area of the rectus sheath below.

3. Sacrifice of both pectoral muscles major and minor.

4. Meticulous axillary dissection, including the sheath of the axillary vein. If multiple enlarged nodes are found around the axillary vein, its resection will permit a more thorough dissection than is otherwise possible. Resection of the axillary vein entails no increased hazard of postoperative edema of the arm, provided x-ray therapy is not employed.

Some studies indicate that five-year survival increases in proportion to the number of lymph nodes found in the surgical specimen.

sions are those with recurrent or metastatic disease following definitive treatment who also require palliative care. As almost one-half of operable patients develop recurrence or metastasis within five years after radical mastectomy, it is obvious that palliative management is the clinician's major problem.

At present palliative treatment depends on control of the disease by irradiation and therapeutic alterations in steroid metabolism with surgical procedures for removal of local or regional disease occupying a minor but sometimes useful position. In general, the best palliation will be secured by conservation of the therapeutic ammunition reserving the larger-caliber and more drastic bullets until the less spectacular shots have outlived their usefulness. Thus, many women respond well to roentgenotherapy of successive areas of metastatic involvement over a period of years, before the disease becomes so disseminated as to indicate the need for hormonal therapy.

With this philosophy in mind, the following outline of the management of inoperable mammary carcinoma was recently developed by the Subcommittee on Breast and Genital Cancer Committee on Research, American Medical Association.

A. Palliative Radiotherapy

With one exception inoperable breast carcinoma, whether primary or recurrent and/or metastatic postoperative should have preliminary palliative treatment by radiotherapy if the dominant lesions are encompassable by this modality. The exception is that of metastatic lesions (not local recurrence) of soft tissue in women five years or more postmenopausal. For these patients primary palliative treatment by estrogens is preferable. The postmenopausal phase is defined as that following the last menstrual period, or what may reasonably be accepted as such.

Even in this situation, however moderate doses of irradiation will produce more rapid regression than if estrogens alone are employed. It should be noted that "soft tissues" is employed to designate such sites as lymph nodes, contralateral breast, chest wall or subcutaneous areas in any part of the body and does not include visceral metastasis. When employed concurrently with the initiation of hormone therapy a tumor dose of 1000 rads to not more than 1500 rads seems to be entirely adequate.

All major areas of skeletal involvement with significant weight bearing function should have the benefit of x-radiation regardless of other management. Large tumor doses are not necessary for control of most metastatic lesions in bone. Tumor dosage in the range of 1500 rads will be adequate in most instances and, in some patients will permit re-treatment of the same area at a later time without concern over skin tolerance.

B. Alterations of Steroid Metabolism

I Indications for Artificial Menopause
Induction of menopause should precede the use of androgenic hormone in premenopausal patients. In the absence of medical contraindications oophorectomy seems preferable. Roentgen induced menopause is a satisfactory substitute in women from 40 to 60 years of age provided that a dosage of 500 to 1200 r is delivered to the midpelvis in a minimum period of ten days to two weeks. In younger women, proportionately larger dosage is necessary. About one-third of the patients will exhibit early objective regression of disease after artificially induced menopause. Androgens should not be used in these responsive subjects until clinical evidence of active disease is again apparent.

II Hormones

The hormone of choice is related entirely to the patient's physiologic age in reference to the menopause. Up to, and for a period of three years after the menopause whether natural or by artificial induction the initial steroid of choice is androgenic; following this arbitrary period, estrogens are consistently superior to androgens at all postmenopausal ages and in any and all sites of metastasis.

C. Initial Trial of Treatment

Any attempt to alter steroid metabolism should be pursued for a minimum period of three months before objective criteria of effectiveness are evaluated. During this time observation at intervals of one to three weeks is essential, with careful inquiry as to symptomatic response and development of side effects. Objective changes in breast cancer occur slowly with altered steroid metabolism and may not be apparent until later than three months. Therefore, if the disease remains static, treatment should be continued.

If objective evidence of unaltered progression of the disease is apparent during the trial period, the method of treatment

operative irradiation employed also influence its possible effectiveness. With these reservations as a guide in evaluation of reported results it seems evident that postoperative roentgenotherapy in stage II P carcinomas increases five-year survival with clinical control by 10 per cent.

In a study of 8396 proved cases in Connecticut from 1935 to 1953 the one group of patients showing a significant improvement rate of 8.7 per cent in later years were those having irradiation after radical surgery. The improvement in patients in stage II was significant at the 5 per cent level.

Preoperative x-ray therapy in operable cases (stage IIa C) is not of value in the opinion of most American surgeons and radiologists although of value as a therapeutic test of radiosensitivity in more advanced (stage IIb C) cases, as outlined earlier in this section.

There is no reasonable basis for postoperative roentgenotherapy of patients with stage I P lesions with two possible exceptions. If evidence indicates that an inadequate axillary dissection has been done the limited number of nodes removed may constitute inadequate evidence that the process is actually stage I P. For lesions located in the medial quadrants irradiation to the medial axillary area is a logical sequel to operation even though all axillary nodes are clear. In most instances postoperative roentgenotherapy is best directed toward regional lymphodal areas (axillary supraclavicular and mediastinal). Irradiation of the chest wall in the operative area is inadvisable either the dosage given is so small as to be of no value or if adequate is hazardous in respect to the poorly vascularized skin and the frequency of pulmonary fibrosis. Should local chest wall recurrence develop later its control by focal irradiation is frequently most effective but is not possible if the tolerance of the skin has been lost by prior irradiation of large portals, even in small dosage.

If postoperative radiotherapy is to be employed, its intent should be for its possible cancericidal, not psychologic, effect. This implies the desirability of its direction and planning by one whose interest in radiation therapy is more than casual. It also requires conditioning of the patient to a course of treatment given daily for several weeks with the subsequent acute though transient, cutaneous reaction over the treated areas. Intensive irradiation can sterilize mammary carcinoma, when employed as primary treat-

ment in operable cases the original focus is destroyed in about one third of the subjects. According to recent reports, a cancericidal effect in lymphodal deposits may be obtained with a similar frequency. Thus, although only 5 to 7 per cent of recurrences are axillary in origin the effectiveness of radiotherapy supplementary to surgery is somewhat greater than this figure, but only after adequate treatment uniformly employed for all patients with axillary nodal involvement.

Treatment of pregnant or lactating patient. Carcinoma of the breast developing during pregnancy or lactation is usually attended by symptoms and signs indicating an unfavorable biologic pattern, such as rapid growth, bulky primary lesion, cutaneous erythema or extensive axillary nodal involvement. So common is this pattern that the patient with carcinoma of the breast which becomes manifest during pregnancy or lactation came to be regarded as categorically inoperable and incurable. More recently a number of reports have proved the error of this assumption, with figures as favorable as 30 per cent five-year survival. The same criteria for separation of operable and inoperable cases should be employed as for any carcinoma of the breast, with the expectation that about two-thirds of these patients will present inoperable, incurable disease best treated by palliative irradiation. For patients developing mammary carcinoma during pregnancy the following outline seems to be a satisfactory guide.

At any stage of pregnancy the carcinoma should be treated as though the patient were not pregnant. Thus if radical mastectomy is indicated, the procedure is done with little danger of abortion or premature delivery.

If the fetus has not reached the stage of viability therapeutic abortion should be recommended, the decision being left to the patient and her husband. If the pregnancy is five months or more in duration, little if any benefit will accrue from its interruption.

Palliative treatment. Over 40 per cent of all patients with mammary carcinoma are incurable at the time of initial examination. In some institutions for the medically indigent, the proportion is considerably higher. The basic reason for incurability of most primary untreated lesions, however, is the unfavorable biologic characteristics of the disease in this fraction of patients. Added to the patients with inoperable primary le-

is necessary before clear-cut and conclusive indications can be established for their use.*

End Results. As in any form of cancer valid measurements of effectiveness of treatment require observance of certain criteria, some of which are

1. The total experience (absolute survival rate) of the institution or individual must be reported, thus avoiding the factor of selection.

2. The sample must be of significant size and homogeneity

3. Successful follow-up of patients should exceed 90 per cent and untraced patients should be counted as failures.

4. Differentiation must be made between "survival" and "survival without disease" as determined by clinical examination

5. Survival after treatment should be compared with survival of untreated patients. Therapeutic success is represented by the extent to which survival of treated patients exceeds that of untreated or by the percentage of survivors without clinical evidence of disease at various intervals.

6. A method of presenting survival in terms of yearly "survivorship function" is more enlightening from a biometric view as developed by Berkson and Gage. The logarithm of survival is plotted as ordinate against time as abscissa to produce a curve giving the death rate for any given year. The same curve for a normal population of similar age may be shown also for contrast.

Results of "curative" treatment. A further consideration in mammary carcinoma is its variability and tendency to late recurrences and appearance of metastasis. The arbitrary yardstick of five years following treatment is thus of limited significance; end results in terms of clinical control at the end of ten years are of significant value.

End results in operable cases following definitive treatment, in terms of survival

Editor's note: There is a difference of opinion as to whether hypophysectomized patients are controlled more easily with replacement therapy than are adrenalectomized patients. Removal of the hypophysis can be accomplished surgically with a reasonable mortality rate. Surgical removal is a more satisfactory procedure than attempts to destroy the hypophysis by the implantation of a radioactive substance such as yttrium⁹⁰. Section of the hypophyseal stalk is not a satisfactory substitute for hypophysectomy. A series of patients is now under study in whom the hypophysis is removed before the development of metastases is clinically evident. It is hoped that some evidence may be offered concerning the permanency of the capacity of the patient to respond favorably to endocrinologic ablations.

without clinically evident disease, are of value provided total experience is reported and grouping of operable cases is clearly defined. Most of the apparent improvement in end results reported in recent years by some surgical centers is the result of selection stemming from recognition of criteria of inoperability.

In respect to *total experience* an approximation of the best results of treatment recently reported from various centers, compared with the natural history of the disease, is as follows:

	SURVIVAL	
	5 YEARS (PER CENT)	10 YEARS (PER CENT)
Untreated	20	5
All patients treated, including those with recurrent disease	30	15
All patients, primary treatment	40	25

For considerations of prognosis, end results as related to the stage of the disease are important. The usual distribution of pathologic stages reported by most authors is

	PER CENT
Stage I	30
Stage II a	25
Stage II b	30
Stage III	15

In other classifications (such as Forstmann's) stages II, III and IV are equivalent to II a, II b and III above.

Representative results of definitive primary treatment are

	SURVIVAL	
	5 YEARS (PER CENT)	10 YEARS (PER CENT)
Untreated	20	5
Stage I	75	60
Stage II a	40	25
Stage II b	20	10
Stage III	0	0

In stage II cases, the extent of axillary lymph node involvement by histologic examination is a determinant of survival. If the axilla is divided into three levels in relation to the pectoralis minor muscle, five-year results bear the following relation to ascending metastatic involvement

AXILLARY NODAL METASTASES	SURVIVAL—5 YEARS (PER CENT)	
	Level I only (lower third)	65
	Level II (mid-axilla)	45
	Level III (apical nodes)	20



Figure 11 A, Photomicrograph of biopsy tissue from a primary inoperable carcinoma of breast before treatment. B, Photomicrograph of biopsy tissue from same neoplasm after steroid (estrogenic) hormone therapy with major regression. Note diffuse fibrosis and cellular changes.

should be changed. Increase in pain during the first two weeks of treatment is not uncommon and is not an indication for change of treatment. Later in the course pain may be significant indication of progression.

D Plans of Treatment with Steroid Agents

The standards of reference for treatment are

1. Androgen—testosterone propionate in oil in dosage of 50 mg. three times weekly by intramuscular injection.
2. Estrogen—oral diethylstilbestrol, 15 mg. daily usually in three divided doses. In the rare patient who cannot tolerate oral medication, injectable material may be substituted.

If objective response is achieved, these dosage schedules may be maintained until reactivation of disease is apparent, some patients do well on lesser maintenance doses. The Committee has no data indicating any superiority in tumor-suppressing effect by any steroids over that obtainable with testosterone propionate and diethylstilbestrol (Fig. 11).

E. Secondary Plans of Therapy

For the patients not responding to initial therapy or those escaping control after initial regression, the following sequence of further management is advised.

1. Discontinue hormone therapy for one month, as some patients will then exhibit regression and should have no further treatment until reactivation of disease is evident.
2. Patients nonresponsive to androgen who have not had bilateral oophorectomy should now be so managed. If

oophorectomy has been done, experience has shown that a cautious trial of estrogen is indicated.

3. Postmenopausal patients with disease not controlled by estrogens may have a trial of testosterone, given in the same manner as already described.

F Hazards

Emphasis should be placed upon the most dangerous side effects of steroids: fluid retention in the early phase of treatment and hypercalcemia. Both of these complications are the most frequent in patients with cardiovascular renal disease, which in elderly patients may become apparent only after the initiation of steroid therapy. Such concomitant disease makes these complications more ominous and demands more frequent observations.

G Experimental Procedures

Both adrenalectomy and hypophysectomy are in an experimental phase of development and should not be employed generally. It seems evident that almost one-half of women showing evidence of a steroid-dependent neoplasm, as indicated by prior response to administered steroids or oophorectomy will secure a further interval of control of their disease following adrenalectomy.

Some investigators have published results indicating that hypophysectomy as a primary palliative measure is more effective than adrenalectomy with or without oophorectomy. There is, as yet, insufficient evidence to establish a significant difference between the two procedures in control of the disease or longevity. Further experience with larger groups of patients

dissemination Axillary nodal metastasis is usually evident and pulmonary metastasis may also be demonstrated at the initial examination

If axillary or other metastasis is not evident, radical mastectomy is indicated. More commonly with axillary extension already obvious, the disease is incurable and the most effective management is castration reserving stilbestrol for later use when the disease escapes the customary postorchietomy period of control

Survival for five years is less than 10 per cent.

Sarcoma. Sarcomas constitute about 1 per cent of malignant neoplasms. The most frequent connective tissue neoplasm is the *cystosarcoma phylloides* generally accepted as arising in a pre-existing fibroadenoma. As is consistent with such an origin this tumor is actually of compound structure, although the epithelial component is with rare exceptions largely overwhelmed by the fibroblastic process. There is divided opinion on the nature of the neoplasm some authors maintain that *cystosarcoma phylloides* in defiance of its ancient nomenclature, occurs in benign and malignant variants, while others state that if a sufficient number of areas are histologically examined genuine sarcoma is invariably found. In rare instances, both epithelial and fibroblastic elements contribute to the structure of a compound, malignant neoplasm. The tumor is characteristically bulky, nodular and expansile, of slow growth but capable of attaining an impressive size in cases of long duration, giving rise to the term "giant fibroadenoma." Encapsulation is the rule. Metastasis is infrequent and then usually by hematogenous rather than lymphatic routes, with the lungs the most frequent site of deposit. It would seem best to employ the term *adenofibrosarcoma* for this dilatory neoplasm. For the more bulky variants simple mastectomy is adequate treatment. When relatively limited in extent, wide resection of the breast is a proper procedure. Axillary dissection is not indicated.

Specific types of sarcoma occur in the breast, but their total number is considerably less than the adenofibrosarcomas. Included are mesenchymoma, even true teratomas, liposarcoma, hemangioendothelioma and sarcoma exhibiting metaplasia to simulate chondrosarcoma and osteosarcoma. The treatment of these rare neoplasms is surgical, of such scope as may be indicated by the extent of the individual lesion. Lym-

phatic dissemination is so unusual as to preclude concomitant axillary dissection. Prognosis is highly variable but is usually related to the degree of differentiation or anaplasia exhibited by the neoplasm

READING REFERENCES

- Ackerman, L. V., and del Regato, J. A. *Cancer: Diagnosis, Treatment and Prognosis*, 2nd ed. St. Louis, C. V. Mosby Company 1934
- Adair, E. E. Plasma Cell Mastitis—a Lesion Simulating Carcinoma. *Arch. Surg.* 26:735 1933.
- Berkson, J., and Gage, R. P. Calculation of Survival Rates for Cancer. *Proc. Staff Meet. Mayo Clin.* 25:270 1930
- Ciba Foundation Colloquia on Endocrinology. Vol. I. Steroid Hormones and Tumor Growth. Philadelphia, The Blakiston Company 1932.
- Daland, E. M. Untreated Cancer of the Breast. *Surg. Gynec. & Obst.* 44:264 1927
- Dao, T. L., and Higgins, C. Metastatic Cancer of the Breast Treated by Adrenalectomy. *J.A.M.A.* 165:1703, 1937
- Endocrine Aspects of Breast Cancer: Proceedings of a Conference held at the University of Glasgow July 1937. Edinburgh, E. and S. Livingstone Ltd., 1938
- Foot, F. W., and Stewart, F. W. Comparative Studies of Cancerous versus Noncancerous Breasts. *Ann. Surg.* 121:6, 1945
- Geschickter, C. F. *Diseases of the Breast, Diagnosis, Pathology, Treatment*. Philadelphia, J. B. Lippincott Company 1943.
- Goss, L. W. : The Problem of Bilateral Independent Mammary Carcinoma. *Am. J. Surg.* 89:171 1954
- Haugensen, C. D., and Stout, A. P. Carcinoma of the Breast. *Ann. Surg.* 116:601, 1942.
- Haugensen, C. D., Stout, A. P., and Phillips J. S. The Papillary Neoplasms of the Breast. *Ann. Surg.* 133:18, 1951
- Kilgore, A. R., and Fleming, R. Abscesses of the Breast, Recurring Lesions in the Areolar Area. *California Med.* 7:190, 1932.
- Levin, M. L. Cancer Reporting in New York State. *New York J. Med.* 44:880 1944.
- Mackdonald, I. Mammary Carcinoma: a Review of 2,636 Cases. *Surg. Gynec. & Obst.* 74:75, 1942.
- Mackdonald, I. Biological Predeterminism in Human Cancer. *Surg. Gynec. & Obst.* 92:443, 1951
- Mackdonald, I. The Role of Extirpative Procedures in Cancer of the Breast. *Cancer* 10:805, 1957
- McWhirter, R. The Value of Simple Mastectomy and Radiotherapy in the Treatment of Breast Cancer. *Brit. J. Radiol.* 21:569, 1948.
- Moore, O. S., Jr., and Foot, F. W., Jr. The Relatively Favorable Prognosis of Medullary Carcinoma of the Breast. *Cancer* 2:635 1949
- Nathanson, I. T. and Welch, C. E. Life Expectancy and Incidence of Malignant Disease. I. Cancer of the Breast. *Am. J. Cancer* 28:40, 1936.
- Portmann, U. V. Cancer of the Breast. Classification of Cases, Criteria of Incurability and Treatment. *J.A.M.A.* 144:513, 1950.
- Proceedings of the Third National Cancer Conference, Breast Panel, pp. 87-177. American Cancer Society Inc., and National Cancer Institute of the U. S. Public Health Service, 1956

All of the foregoing figures are of course approximations of currently reported, statistically valid results. Some individual authors for example have reported five-year clinical control in stage I cases as high as 90 per cent, but such results usually reflect either a small sample or factors of selection.

Representative of those performing combined radical mastectomy and mediastinal node dissection, J. A. Urban reports as follows on his first seventy-five cases in a personal communication

	TOTAL NO.	5-YEAR SURVIVAL
All nodes clear	29	25
Axillary nodes only invaded	19	14
Internal mammary nodes only invaded	3	1
Both axillary and internal mammary nodes invaded	24	10
Entire group	75	50

If these patients had had a standard radical mastectomy the study of axillary nodes only would have divided them as follows with five-year survivals

Stage I — 29 + 3 = 32
5-year survival = 26 or 81.2%
Stage II — 19 + 24 = 43
5-year survival = 24 or 55.8

These figures for five-year survival exceed those commonly reported, but individual reports of larger series treated by radical mastectomy are not significantly different, e.g., Haagensen's results of 86.2 per cent in 160 stage I cases and 50.5 per cent in 196 stage II cases. The overall five-year survival is identical for Urban and Haagensen or 66.6 per cent. In view of the fact that, at least by microscopic findings the mediastinal dissection was unproductive in forty-eight of Urban's seventy-five patients a more impressive increase in survivals at the ten year level seems required to justify the combined procedure.

Local recurrence of carcinoma, i.e., in the operative area, is variously reported at 6 per cent to over 20 per cent, the usual figure ranging from 10 per cent to 14 per cent. The more rigid criteria of operability produce lower rates of local recurrence and the adequacy of the operative procedure is also an important factor.

Results of palliative treatment Occasional patients experience long-term control of recurrent and advanced metastatic disease with irradiation and steroid therapy but over-all results have to be measured in terms

of months rather than years. The most important result of palliative treatment is the relief of symptoms and rehabilitation, even when longevity is not significantly increased. Relief of pain from skeletal metastasis occurs in 70 per cent or more of patients both with irradiation and steroids though less than half of such patients show objective evidence of healing. Long term control by steroid therapy is most frequent in soft tissue peripherally located metastasis in postmenopausal women treated with estrogens. In some of these patients the disease is inactivated for two or more years.

The final report of the American Medical Association Subcommittee on Breast and Genital Cancer is an analysis of 945 women with disseminated mammary carcinoma, of whom 581 were treated by androgens and 364 by estrogens as the only form of palliative therapy. Follow-up was successful in 86.5 per cent (818). The following summary of the Subcommittee's report should be a guide to the proper choice of hormonal therapy most notably in its rejection of the notion that androgens are superior to estrogens for skeletal metastasis.

Androgens produced objective regression in 81 per cent of premenopausal patients, and in from 13 to 28 per cent of postmenopausal women. Estrogens, limited properly to postmenopausal patients, induced regression in 16 to 40 per cent of patients. Both types of hormones were more effective after the eighth postmenopausal year at which time both reached a plateau of performance, but with the estrogens inducing a higher relative frequency of regressions (40 per cent) than the androgens (27 per cent).

In cases with unsystemic dissemination, estrogens produced a greater degree of control in soft tissues at all postmenopausal ages, and were equal, or superior to androgens for skeletal and visceral metastasis.

Responsive patients had significantly longer survival after both androgenic and estrogenic therapy while the unresponsive individuals in all groupings had identical, average survivals characteristic of untreated patients.

Steroid sex hormones are sufficiently effective in postmenopausal women to deserve a primary trial in the treatment of disseminated mammary carcinoma. After the fourth postmenopausal year estrogenic substances are the agents of choice.

Carcinoma of the Male Breast. Less than 1 per cent of all mammary carcinoma is of the male breast. The pathologic condition and natural history are the equivalent of the disease in the female. Unfortunately the lesion is usually relatively advanced and clinically obvious when first seen, probably because of the rudimentary structure of the thin mammary plate with early lymphatic

THE MEDIASTINUM

By JOHN M. DORSEY M.D.

JOHN MICHAEL DORSEY the son of a doctor in a small community in central Illinois, attended the University of Chicago and Rush Medical College. Trained in surgery at the Mayo Clinic his attention has become focused upon the surgical problems best solved by a combined expert knowledge of the abdominal and thoracic cavities. A Professor of Surgery at Northwestern University he is Chairman of the Department of Surgery at Evanston Hospital.

The mediastinum is a potential space enclosed within the parietal investing layers of the right and left pleural spaces laterally, the sternum in front, the vertebral column behind and the diaphragm inferiorly. The thoracic mediastinum becomes the cervical mediastinum above the level of the thoracic inlet. The mediastinum is important surgically because within it are located the heart, giant vessels, trachea and the tissues of its bifurcation, the esophagus, important nerve trunks, the thoracic duct and the thymus gland.

It is convenient to divide the mediastinum into anterior and posterior divisions. The anterior division is further divided into superior and inferior compartments; the middle mediastinum can be best considered as part of the posterior division. Figure 1 diagrammatically illustrates these divisions in the sagittal view.

It is obvious that the heart and pericardium are the principal occupants of the inferior division of the anterior mediastinum. The structures about the tracheal bifurcation are likewise the essential occupants of the middle mediastinum, whereas the aorta, esophagus, thoracic duct and important nerve trunks obviously lie in the posterior compartment.

The pressure within the thoracic mediastinum is less than atmospheric. It is important to the normal physiologic function of the mediastinal structures that this be relatively undisturbed. Trauma with extravasation of the fluid contents of one of the mediastinal structures (i.e., air, blood, lymph, inflammatory exudate, gastroenteric fluid) new growths, congenital deformities and disturbances in pulmonary function, as by atelectasis or pneumothorax, may alter this normal pressure directly or by displacement of structures so as to seriously threaten life.

MEDIASTINAL EMPHYSEMA

The escape of air into the mediastinal space may occur from the esophagus or from the tracheobronchial tree. It is a possible complication of such respiratory disorders as asthma, influenza and pneumonia. Violent coughing associated with these illnesses is responsible. It may follow heavy lifting or straining at stool. Air escaping, usually from the tracheobronchial tree, dissects along the interstitial tissue and bronchial vessels to the hilum of the lung. There may be associated pneumothorax. Accompanying substernal pain may be severe, radiating to the neck and arms and suggesting the symptoms

- Robbins, C. F., Brothers, J. H. III, Eberhart, W. F., and Quan, S.: Is Aspiration Biopsy of Breast Cancer Dangerous to the Patient? *Cancer* 7:774, 1954.
- Ryan, J. A., and others: Breast Cancer in Connecticut, 1935-53. *J.A.M.A.* 167:298, 1958.
- Shimkin, M. B., Lucia, E. L., Low Beer, B. V. A., and Bell, H. G.: Recurrent Cancer of the Breast. *Cancer* 7:29, 1954.
- Shimkin, M. B., Lucia, E. L., Stone, R. S., and Bell, H. G.: Cancer of the Breast, Analysis of Frequency Distribution and Mortality at the University of California Hospital, 1918 to 1947. Inclusive. *Surg. Gynec. & Obst.* 94:645, 1952.
- Smithers, D. W., Rigby Jones, P., Calton, D. A. G. and Payne, P. M.: Cancer of the Breast, a Review. *Brit. J. Radiol.* (supp. 4) 1952.
- Stewart, F. W.: Tumors of the Breast. Subcommittee of Oncology Committee of Pathology National Research Council, Section IX, Fascicle 34. Washington, D. C., Armed Forces Institute of Pathology 1950.
- Subcommittee on Breast and Genital Cancer Research Committee, Council on Pharmacy and Chemistry American Medical Association. Androgens and Estrogens in the Treatment of Disseminated Mammary Carcinoma (a Retrospective Study of 945 Patients) *J.A.M.A.* In press.
- Treves, N. and Sunderland, A.: Cystosarcoma of the Breast: a Malignant and a Benign Tumor. *Cancer* 4:1286, 1951.
- Urban, J. A., and Adair, F. E.: Sclerosing Adenocarcinoma. *Cancer* 2:1236, 1949.
- Watson, T. A.: Results of Treatment of Cancer of the Breast. *Surg. Gynec. & Obst.* 104:106, 1957.
- Wood, D. A. and Darling, H. H.: Cancer Family Manifesting Multiple Occurrences of Bilateral Carcinoma of the Breast. *Cancer Research* 3:509, 1943.

may compress the mediastinal structures to a dangerous degree making immediate surgical relief imperative. The cervical fascial layers should be divided, loosely packed open and, if necessary, their abnormal air content evacuated by suction pump.

Crushing injuries to the chest wherein fluid, blood, lymph and inflammatory exudate quickly accumulate give the patient a most characteristic appearance. There is a progressive increase in swelling over the soft tissues of the thorax and in those of the neck and face. The eyelids may swell so as to obscure the eyeballs. There are subcutaneous petechial hemorrhages and the skin may be the dusky hue of that characterizing cyanosis of varying degrees. The victim is unrecognizable. Shock both from the primary injury and from the developing hypoxia must be combated.

The measures to control hypoxia due to whatever cause must always be kept in mind. They are maintenance of a clear airway by suction, by bronchoscopy or by tracheostomy, oxygen administration, paravertebral procaine block, correction of the structural deformity and incision and drainage for relief of abnormal pressure plus other measures for shock prevention.

The anatomy of the cervical mediastinum, as well as the thoracic mediastinum, and the relationship of the deep cervical fascia of the neck to the thoracic inlet are important in the consideration of the management of air and inflammatory fluid accumulation.

MEDIASTINITIS

The principal routes by which infection involves the mediastinum are in the order of their frequency: extension directly from structures within the mediastinum, extension from the neck by way of the fascial planes and through lymphatic drainage. The advent of chemotherapy and of the many new antibiotics has completely changed the course, the seriousness and the outcome of acute mediastinal infections. Whereas at one time pulmonary infections leading to suppurative pleurisy and in turn suppurative mediastinitis, both diffuse and localized, were not uncommon, today these processes are relative rarities and seldom are presented for their definitive care. The most frequently encountered cause of acute mediastinitis today is from foreign body trauma either through ingestion or by accidental injury in the many diagnostic and therapeutic endoscopic procedures. It is no reflection on the techniques themselves. In the

vast majority of instances it mirrors the increasing use of these diagnostic measures and their inevitable risk.

In considering mediastinitis arising from infected mediastinal lymph nodes, one visualizes the acute or subacute lymphadenitis seen for example in tuberculous infections. Obviously mediastinal as well as other lymph nodes may be involved in the drainage from any focus of infection. This is a localized process. It may go on to become chronic. An example is seen in the formation of traction diverticula due to inflammatory contraction and outpouching of the esophageal wall about chronically inflamed, even calcified, lymph nodes. Under certain circumstances, these diverticula have perforated. Whereas it is conceivable that they might produce spreading, phlegmonous mediastinitis, experience has been that the inflammatory process in the mediastinum has prevented such spread. However the intrapleural manifestation of disease such as empyema and even tension pneumothorax may predominate.

Acute inflammatory disease such as arises from infected teeth or from retropharyngeal abscess has in the past spread to the mediastinum from the cervical region along the fascial planes. It is important to understand the basic pathologic processes, the manifestation of their spread and the most effective measures in their treatment even though modern therapy has almost entirely altered the index of their danger.

Visceral perforation in the neck, as well as within the confines of the thorax, is similarly minimized as a serious threat to health through the prompt and adequate employment of antibiotics. Here too and of even greater importance, must the proper therapeutic measures be understood. Antibiotic sensitivity through previous employment, often for trivial affections, has led to a definite percentage of the population becoming sensitized. Whereas new antibiotics are being developed yearly, unless desensitization means are likewise developed there will be times when antibiotic usage will be valueless and even dangerous. For comparison, one must keep in mind the wisdom of the philosophy expressed by Evans Graham about the treatment of thoracic empyema. In general, it was to this effect: when the infecting organism is relatively nonvirulent almost any treatment suffices, but when the infecting organism is virulent then only will the best treatment be effective.

As in thoracic empyema, the best treat-

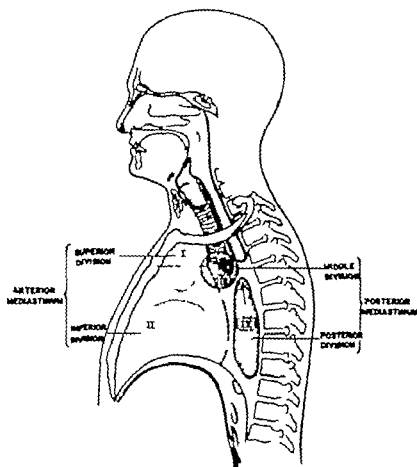


Figure 1 The mediastinum is presented in sagittal view; it is divided into anterior and posterior compartments. The anterior division is further subdivided into superior and inferior spaces. The mediastinum is considered as a portion of the posterior compartment.

The Roman numeral ovoids designate the more common location of specific tumors. In the region of ovoid I should be found the tumors of the anterior superior mediastinum which would be largely those of thyroid and thymic origin. Dermoids would occupy this area, too, as well as the area of ovoid II. Also, in ovoid II would be the pericardial cysts, lipomas and the more rarely encountered new growths. In ovoid III would be the tumors at the hilum of the lung and in ovoid IV the posterior mediastinal tumors which, as has been stated, are principally neurofibromas, but which may also be chondromas, chondrosarcomas and chondrosarcomas. Not illustrated are those lesions in the thorax which occur in the mediastinum through upward extension of intra-abdominal processes. These may be foramen of Morgagni diaphragmatic hernias which are confused with anterior mediastinal tumors, and partial evolutions of the diaphragm containing spleen or liver which are confused with mediastinal tumors in more posterior locations.

of coronary occlusion. Dyspnea and cyanosis may be present. Fortunately the characteristic to and fro crunching or clicking sound audible over the precordium synchronous with respiration (Hamman's sign) is distinctive.

Trauma to the esophagus or tracheobronchial tree either direct or by increase in intraluminal pressure is the most common cause of mediastinal emphysema. The reaction associated with the escape of intra-esophageal content is more damaging than that from air alone. On the other hand, a sizable rent in a major bronchus will allow the escape of enough air to alter seriously the pressure relationships within the mediastinum to the point of critically embarrass-

ing the normal function of its contents. The more peripheral the source from the tracheobronchial tree, the slower is the accumulation of air. There may be no serious embarrassment due to pressure change, yet distortion of the normal body appearance due to subcutaneous emphysema may be extreme. Crepitation the sign of crackling of the subcutaneous tissue on palpation will be detected at times as far as the terminal phalanges of the fingers. The neck and face may be enormously swollen, yet the interference with respiration and circulation may be quite minimal.

On the other hand, the rapid escape of air along the fascial planes of blood vessels, together with the extravasation of fluid

ment is adequate drainage. For infection above the level of the fourth dorsal vertebra cervical mediastinotomy will be adequate. Infections whose level is below that of the fourth dorsal vertebra will require mediastinotomy either through a posterior approach or through a transpleural approach (Fig. 2)

The most effective treatment begins with early recognition. The diagnosis is suspected if in a patient whose temperature, pulse and leukocyte count are normal before an endoscopic procedure, there develops thereafter a substernal epigastric or shoulder pain with

an elevation of temperature of 1° or 2° F and of the leukocyte count from levels of 11 000 to 25 000 cells per cu mm. of blood. If for several hours this pain persists and is made worse by deep inspiration, then the suspicion is clinically confirmed. The additional physical signs which may be present are fullness in the cervical region if the perforation has been in the upper dorsal or lower neck level tenderness, edema, in duration and crepitation due to subcutaneous emphysema.

Röntgenograms made with a portable apparatus are of little help and should not be

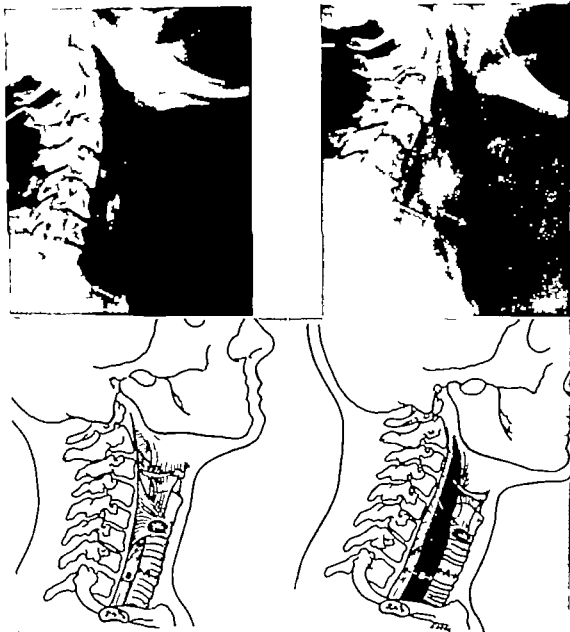


Figure 4. The lateral roentgenograms of the neck are further explained by the illustrative drawings. On the left, it is noted that the width of the tracheal column of air is greater than the distance between the posterior tracheal wall and the anterior vertebral body. This space contains the esophagus. On the right, the space between the anterior vertebral body and the posterior tracheal wall designated as B' has been considerably widened by the presence of air and exudate due to mediastinitis. It is definitely wider than the tracheal column of air labeled A.

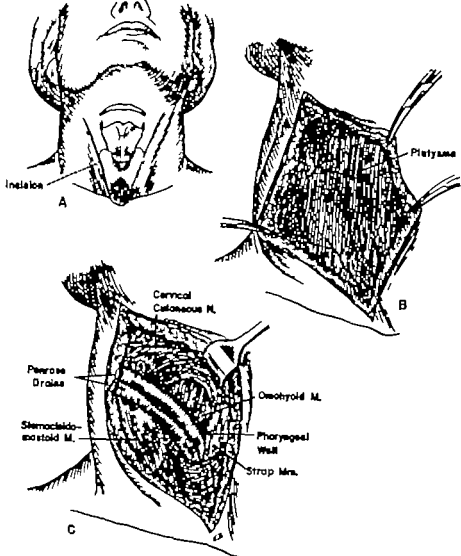


Figure 2 Cervical mediastinotomy for effective drainage as low as the level of the fourth dorsal vertebra. A, Skin incision along the anterior border of the right sternomastoid muscle. B, Liberal exposure of the platysma muscle before division. C, Penrose drains are illustrated as having been inserted to the depth of the visceral compartment lateral to the strap muscles below and medial to the omohyoid and well into the superior mediastinum. Right-sided drainage avoids possible injury to the larger left thoracic duct.

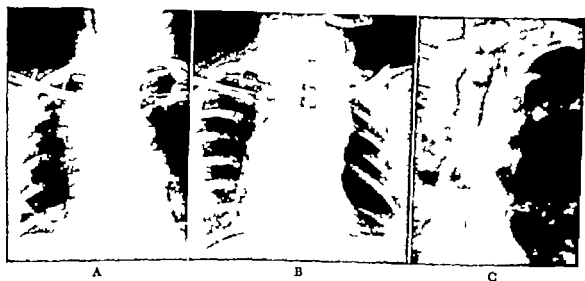


Figure 3 A, Anteroposterior roentgenogram of the thorax illustrating a right, upper mediastinal abscess. B, Oblique roentgenogram of the thorax after barium swallow demonstrating extravasated droplet of barium in the mediastinum. C, Roentgenogram of the esophagus after barium ingestion illustrating a pharyngo-esophageal diverticulum which had been perforated at the time of endoscopy producing the abscess illustrated in A and B. It was not until after the mediastinal abscess had been drained by posterior mediastinotomy that this was recognized. This must be kept in mind as a cause of esophageal perforation when endoscopy is carried out before preliminary barium study.

ment is adequate drainage. For infection above the level of the fourth dorsal vertebra cervical mediastinotomy will be adequate. Infections whose level is below that of the fourth dorsal vertebra will require mediastinotomy either through a posterior approach or through a transpleural approach (Fig. 2).

The most effective treatment begins with early recognition. The diagnosis is suspected if in a patient whose temperature, pulse and leukocyte count are normal before an endoscopic procedure, there develops thereafter a substernal epigastric or shoulder pain with

an elevation of temperature of 1° or 2° F and of the leukocyte count from levels of 11 000 to 25 000 cells per cu. mm of blood. If for several hours, this pain persists and is made worse by deep inspiration, then the suspicion is clinically confirmed. The additional physical signs which may be present are fullness in the cervical region if the perforation has been in the upper dorsal or lower neck level, tenderness, edema, in duration and crepitation due to subcutaneous emphysema.

Röntgenograms made with a portable apparatus are of little help and should not be

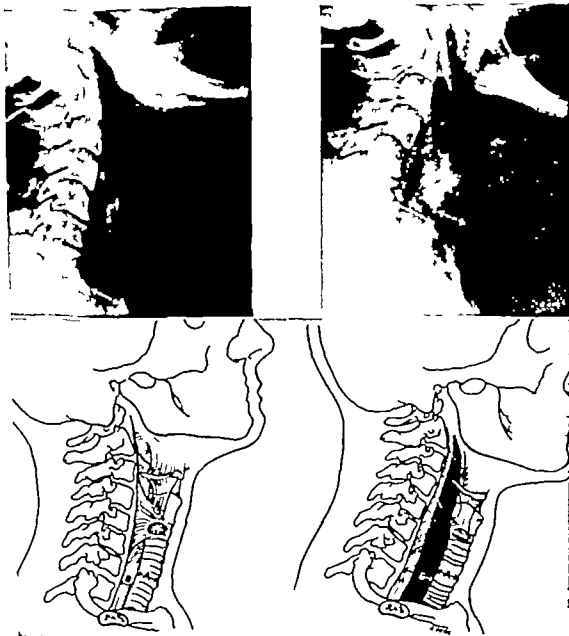


Figure 4. The lateral roentgenograms of the neck are further explained by the illustrative drawings. On the left, it is noted that the width of the tracheal column of air is greater than the distance between the posterior tracheal wall and the anterior vertebral body. This space contains the esophagus. On the right, the space between the anterior vertebral body and the posterior tracheal wall designated as B' has been considerably widened by the presence of air and exudate due to mediastinitis. It is definitely wider than the tracheal column of air labeled A.



Figure 5 Barium study of the esophagus and esophagogastric junction, demonstrating a fistulous tract leading from the left wall of the esophagus just above the esophagogastric junction. This is the characteristic location of so-called spontaneous perforation of the esophagus, which leads to fulminating mediastinitis. Clinically it is frequently represented by the triad of (1) rapid respiration, (2) abdominal rigidity and, most pathognomonically (3) subcutaneous emphysema in the cervical region.

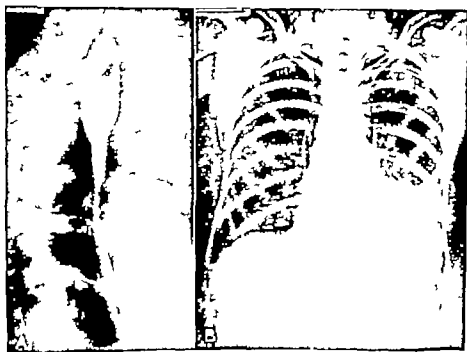


Figure 6 A, Barium study of the esophagus in a patient with lye stricture. B, Anteroposterior view of the thorax in the same patient with evidence of barium extravasation and clouding of the left lower thorax. These were incontrovertible evidences of esophageal perforation and mediastinitis. This patient had tried to forcefully swallow an impacted food bolus.

relied upon. The patient should be taken to the x ray room and given a radiopaque material for fluoroscopic studies. If there is no perforation harm will not follow. If a droplet of fluid escapes from the esophageal lumen (Fig 3) then immediate operation should be performed. Lateral roentgenograms of the neck in addition to revealing air in the tissue spaces, will demonstrate any change in the normal width of the space between the posterior tracheal wall and the anterior vertebral body (Fig. 4) Under ordinary conditions this should not exceed the width of the tracheal column of air. If perforation has occurred, the space is in-

creased beyond this measurement. Other roentgenologic signs include widening of the mediastinal shadow, elevation of the diaphragm and obliteration of the costophrenic or cardiophrenic angle. Small quantities of fluid may be present.

As in perforation of abdominal viscera, there is sometimes a temporary period of apparent improvement with subsidence of reaction between six and eighteen hours after injury marked by a decrease in pain and tenderness and a lowering of the temperature and leukocyte count. This may give a false impression and cause surgery to be delayed, thereby adding to the gravity of the situation.

Figures 4 5 and 6 serve to illustrate some of the essentials in the manifestations and treatment of mediastinitis from various causes in representative cases.

The creation of a high index of suspicion is necessary to the prompt recognition of spontaneous esophageal perforation. It may conceivably be related to peptic esophagitis. The associated pathologic processes are very much like those seen in peptic duodenitis. When perforation does occur its manifestations are explosive. X-ray examination may be inconclusive. Because of discomfort, it is difficult for the patient to cooperate so that an informative roentgenogram can be made. The "interpretive roentgenograms" in Figures 7 8 and 9 illustrate three modes of progression from the initial perforation.

Peptic Esophagitis with Perforation.—
Signs Predominantly of Mediastinitis

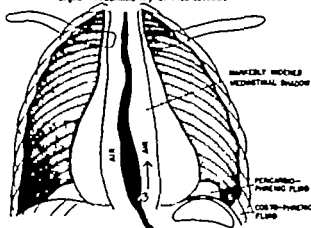


Figure 7 If escaping esophageal content is confined to the mediastinum there may be minimal pleural reaction. The mediastinal shadow may progressively widen. In these individuals detection of cervical emphysema by the discovery of crepitation is of utmost importance

Peptic Esophagitis with Perforation
Mediastinitis and Left Pleural Effusion

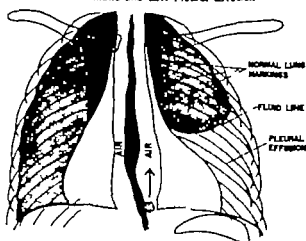


Figure 8. There may be a moderate unilateral or even bilateral pleuroparietal inflammatory reaction if with the esophageal rupture there is perforation or extension into the pleural space or spaces. There will be the combined symptoms of mediastinitis and pleuritis.

Peptic Esophagitis with Free Perforation Into Left
Pleural Cavity Hydrothorax and Tension Pneumothorax

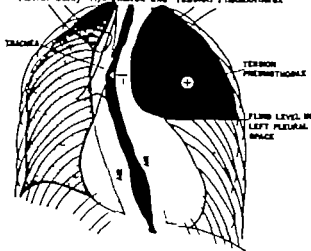


Figure 9 The pleural manifestations may progress to the point of marked respiratory embarrassment due to the development of tension pneumothorax. The increasing intrapleural pressure from accumulated fluid, both air and inflammatory exudate, may lead to death if recognition is not prompt. Immediate thoracentesis is indicated while preparing the patient for surgical intervention.

MEDIASTINAL TUMORS

In general it can be said that tumors within the mediastinum may have their origin from one or all of the primordial germ layers or their adult derivatives which have contributed to the normal structures of the region Heuer and Andrus have classified these tumors as follows: dermoid cysts, teratomas, cysts of entodermal and mesodermal origin, cystic lymphangiomas, echi-
nococcus cysts, fibromas, lipomas, leiomyomas, xanthomas, chondromyxomas, chondromas, chondrosarcomas, neurofibromas, ganglioneuromas, neuroblastomas, neuroepitheliomas, benign and malignant tumors of the thymus gland, lymphosarcomas, Hodgkin's disease, endotheliomas, primary and secondary sarcomas, primary and metastatic carcinomas and, finally, goiters (Fig. 10).

Assuming that any intrathoracic new growth may be one of these, what generalizations are helpful in guiding diagnostic thought and therapy? Certainly the location of the tumor in the mediastinum helps to establish its identity. Characteristically we know that specific tumors frequently arise

in arbitrarily designated mediastinal divisions. Perhaps the most common mediastinal tumor is the adenoma of the thyroid, when it is substernal in location (Fig. 11). It may be deeper intrathoracically than might be ordinarily suspected and may less commonly lie posteriorly in the mediastinum. It is more characteristically located anteriorly. There need not be an enlarged cervical thyroid.

The next most common anterior mediastinal tumor is the so-called dermoid (Fig. 12). Tumors of this nature containing elements of one germ layer are epidermoids; those with two layers are true dermoids; and those with three germ layers represented are teratomas. It is convenient as well as profitable to consider the more usually encountered anterior mediastinal tumors in a differential diagnosis of lesions so located by x-ray examination. Tumors may arise in a given mediastinal compartment and by growth extend to occupy other mediastinal compartments; nevertheless the point of origin is of extreme importance in determining the nature of these neoplasms. An an-

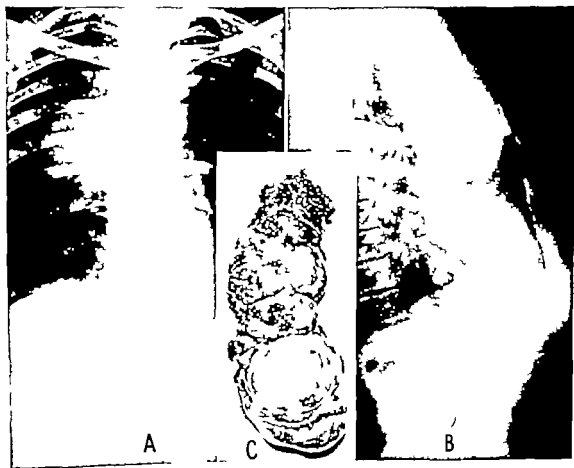


Figure 10 A and B, Anteroposterior and lateral roentgenograms of the thorax illustrating a rounded anterior mediastinal tumor in which there is a calcium deposit. C, Photograph of the gross specimen which includes the cervical thyroid above and its intrathoracic extension.

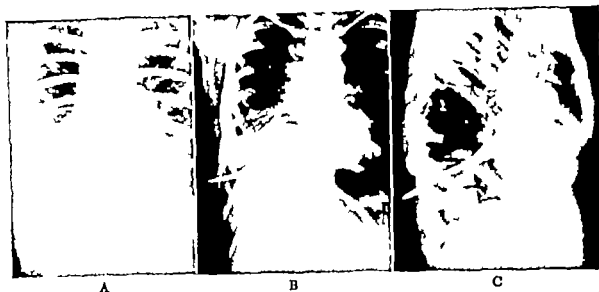


Figure 11. A, Anteroposterior roentgenogram of the chest with clouding in the right lower thorax indicative of pleural empyema. B, Anteroposterior roentgenogram of the chest with catheter instillation of radiopaque Liptodol. The sinus tract obviously communicates with the bronchus and extends toward the midline. C, Lateral roentgenogram of the chest at the time of this diagnostic procedure, showing that the catheter passes anteriorly and that the communication is with the anterior mediastinal tumor. The empyema, in this instance, was secondary to the rupture of the dermoid tumor of the anterior mediastinum into the bronchus.



Figure 12. A, Artist's conception of the anterior approach for removal of the dermoid tumor. This patient was operated upon before the days of chemotherapy and antibiotics. B, Dermoid tumor with the characteristic hair apparent on its surface. C, Photomicrograph of the tumor showing epidermal elements as well as mesodermal elements represented by fat cells.

terior mediastinal dermoid may become complicated through communication with the tracheobronchial tree by rupture and produce pleural empyema which will manifest itself posteriorly. Such a situation is illustrated in Figure 11, which also demonstrates the diagnostic procedures employed to establish the anterior location of the tumor characteristically a hair-covered dermoid bathed in a sebaceous type of secretion and microscopically exhibiting evidence of its two germ-layer origin. Figure 13 presents an interesting comparison to Figure 14. The oblique view in Figure 13 indicates the relationship to the pericardium. This was established as being a so-called

spring water or pericardial (coelomic) cyst, according to the classification one chooses to follow. In contradistinction, the microscopic nature of the tumor in Figure 14 for comparison indicates that it is solid and of thymic origin.

Whereas, anatomically the midmediastinum may not be recognized as a separate compartment, still the region about the tracheal bifurcation is the origin principally of tumors arising in the lymphatic glandular structures or metastatic to them. The lymphomas, Hodgkin's disease and the leukemias constitute one group. Among the metastatic lesions, bronchogenic carcinoma involves the regional lymph nodes which

include the carinal group. The sharpness of the carina as demonstrated by bronchoscopic examination, or by planographic view is of considerable diagnostic significance.

The most common posterior mediastinal tumor is generally agreed to be the neurofibroma. It arises from the neurilemma sheath of intercostal nerves. These tumors undergo a malignant change and become neurofibrosarcomas. The related neoplasms of central nervous system origin such as the ganglioneuromas and neuroblastomas are also found in the posterior mediastinum. Figure 15 illustrates the x-ray appearance both in the anteroposterior and lateral views of a posterior mediastinal neurofibroma, the gross appearance of the tumor and its mi-

croscopic nature are evident. This is a benign tumor and the prognosis after its surgical removal is obviously excellent. Contrast this with the tumor illustrated in Figure 16. The working diagnosis in this instance was also neurofibroma because of the characteristic posterior location of the tumor. At operation, however this proved to be a chondromyxosarcoma arising from the region of the intervertebral disk. Because of its malignant infiltrative nature, it was impossible to resect it completely. Death from local extension resulted. It is quite obvious that the prognosis of all mediastinal tumors depends upon their microscopic nature. This determination can only be made by exploratory thoracotomy and excision of the tumor when possible.



Figure 13. A, Anteroposterior roentgenogram of the thorax illustrating a right mediastinal shadow B, Oblique view of the thorax, after barium swallow demonstrating the relationship of the shadow to the pericardium. This proved to be a so-called spring water cyst.

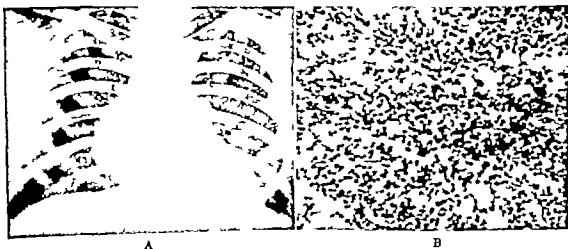


Figure 14. A, Anteroposterior roentgenogram of the thorax illustrating a right mediastinal shadow for comparison with Figure 13. B, Photomicrograph of the tumor establishing it as a thymoma.

The symptoms of mediastinal tumors are obviously variable, depending upon the size and origin of the tumor. Occasionally a symptom may be pathognomonic of a particular type of tumor; for example, coughing up hair is indicative of the presence of a dermoid cyst. Certain manifestations of probable incurability are recognized. Important among these are involvement of the cervical sympathetic nerves producing a Horner's syndrome, hoarseness from recurrent laryngeal nerve invasion and elevation of the diaphragm as an evidence of phrenic nerve damage.

Certain signs, both from laboratory studies, including x ray examination, and physical examination, are of diagnostic importance. It has long been recognized that a circumscribed, spherical tumor with smooth outline is not necessarily benign.

Hard cervical or axillary lymph nodes must be searched for and studied when they are present in association with a mediastinal x ray shadow. The fluid level sometimes seen in anterior mediastinal tumors caused by fat floating on fluid is pathognomonic of dermoids. The use of a test dose of roentgen ray therapy as evidence in the diagnosis of a tumor has very little to offer. Except when surgical intervention is contraindicated, it should not be employed because of the associated delay.

The diagnosis of indeterminate mediastinal shadows should be carried out in a very systematic way. Careful history taking, physical examination, blood studies including bone marrow examination, sputum for direct smear and culture, paraffin block and Papanicolaou study, appropriate x ray examination, including planographic views, radio-



Figure 18. A, Anteroposterior roentgenogram of the thorax demonstrating the right mediastinal neurofibroma. B, Lateral roentgenogram of the thorax proving the tumor to be posterior in location. C, Photograph of the gross specimen. These tumors are ordinarily firm and yellow-white. They may be vacuolated as herein illustrated. D, Photomicrograph demonstrating the characteristic microscopic appearance. Fibrous connective tissue cells are without mitotic figures.



Figure 16 A, Anteroposterior roentgenogram of the chest illustrating the large spherical, right, intrathoracic tumor. B Lateral roentgenogram of the chest demonstrating its posterior location. C, The photomicrograph establishes its microscopic nature as a chondromyxosarcoma. The prognosis in this instance was far more serious than for the benign neurofibroma illustrated in Figure 15.

active iodine survey occasionally diagnostic pneumothorax and needle biopsy and, more frequently advocated than ever before, exploratory thoracotomy are recommended.

A tumor of the mediastinum, as any other intrathoracic tumor should be treated as is a tumor of the breast. The exact nature of the lesion must be determined and the removal accomplished, except when obviously contraindicated.

A surgical approach by a posterolateral incision is commonly employed because it allows the most adequate exposure of the mediastinum. The disadvantage in transgressing the pleural cavity is compensated for by the adequacy of the exposure of the lesion to be resected. The level of the incision depends upon the level of the growth to be exposed. The anatomy of these incisions should be carefully studied. In the young, a rib need not be resected, but in those individuals of the middle and later periods of life, it is better to resect a rib and to divide ribs above or below in order to secure adequate working room. Airtight closure of the thorax is advisable with complete re-expansion of the lung through the use of catheter drains attached to a negative pressure apparatus. When drainage ceases and the postoperative roentgenogram discloses that the lung on the operated side is completely expanded, then the catheter may be removed and a pressure dressing applied over the point of its withdrawal from the chest wall.

More use is being made of an anterior surgical approach by sternum-splitting in

cisions. These incisions may be made as extensions downward from the cervical collar incisions, as extensions upward from upper abdominal approaches or as primary transverse divisions. The sternum should be carefully divided with a Lebschke knife or Gigli saw so as to protect the structures of the anterior mediastinum from inadvertent injury. The sternum may be spread over its entire length or the incision may be carried into an intercostal space if the more extensive exposure is not necessary. In so doing, it is important to protect against injury to and hemorrhage from the internal mammary arteries. The sternum may be reapproximated with multiple stainless steel wire sutures placed through its entire thickness or simply through its anterior table. The anterior fascia of the sternum can be approximated with either fine silk or catgut sutures to cover the projecting end of the wire. If there is any anticipated difficulty from retention of bronchial secretion, it is preferable to do a prophylactic tracheostomy in association with the sternum-splitting incision. This is an innocuous procedure. It is far easier and better to do it electively than ineffectively as an emergency. Because of the reported incidence of malignant change in thyroid adenomas a word could be said for the advantage of the sternum-splitting incision wherein complete removal without morcellation can be carried out. Morcellation would tend to implant the tumor in the event that such adenomas are malignant. Nevertheless, sternum splitting should be reserved for selected cases.

Posterior mediastinotomy has been largely

abandoned in favor of the transpleural posterolateral approach. However, it has a definite place in the armamentarium of the thoracic surgeon. If mediastinotomy is to be accomplished without tearing of the parietal pleura, a long segment of the rib directly over the area to be investigated should be removed, with additional shorter resections of ribs above and below. Open pneumothorax is thus avoided.

Because tumors of the anterior mediastinum may be of thymic origin and because tumors of the thymus are frequently accom-

panied by at least incipient symptoms of myasthenia gravis, curare and its derivatives should be avoided in producing surgical anesthesia. Should respiratory difficulty develop in the postoperative period after removal of anterior mediastinal tumors, it would be well to consider the administration of ephedrine or any of the antimyasthenic drugs which have been developed.

The place of tracheostomy as an elective rather than as an emergency procedure in these surgical procedures cannot be over emphasized.

THE THORACIC WALL, PLEURA AND LUNGS

The Thoracic Wall and Pleura

By WILLIAM E. ADAMS, M.D.

WILLIAM ELIAS ADAMS was educated at the State University of Iowa but has been associated with the University of Chicago's Department of Surgery in progressive ranks since 1928. He is the chief of Thoracic Surgery and Raymond Professor of Surgery. His experimental and clinical investigations helped to establish the specialty of thoracic surgery on firm physiologic principles.

PHYSIOLOGY OF THE THORACIC CAVITY

For a better understanding of the clinical features observed during the management of various abnormalities of the chest, a knowledge of pulmonary function under normal conditions and its alteration under adverse circumstances is desirable. Since pulmonary function is likely to be altered by both extrathoracic and intrathoracic operations, it is important to know the status of the lungs prior to any major operation. Although lung function assists in temperature regulation and in electrolyte balance, its principal objectives are oxygenation of the blood and removal of carbon dioxide.

Many patients have adequate pulmonary reserve to meet the body needs during illness, during and following surgery and following injury. However, in persons of advanced years or in the presence of extensive

bilateral pulmonary disease, lung function studies may be of significance in both medical management and prognosis.

Pulmonary function may be subdivided into four principal parts, namely, ventilation, distribution, perfusion and diffusion. The requirements for performance of these functions include a normal bellows action of the chest wall and diaphragm with a negative intrapleural pressure of 6 to 8 cm. of water, adequate pulmonary capacity and patent air passages for the free exchange of gases and adequate vascular bed for perfusion of the pulmonary tissue. Under normal circumstances, a considerable reserve of pulmonary function is available for performing these activities. During rest, only approximately one-fourth of the lung capacity is required for normal pulmonary function.

Although a large percentage of patients have adequate pulmonary reserve for toler-

ating operative procedures, in individuals of advanced years or who have bilateral pulmonary disease, pulmonary reserve may be considerably reduced. Tests for determining the status of pulmonary ventilation and diffusion are important, especially when pulmonary reserve may be further decreased as a result of pulmonary resection. For ventilatory tests, the timed vital capacity (maximum volume exhaled after maximum inhalation measured in seconds) and the maximum breathing capacity (maximum amount of air inhaled and exhaled while breathing deeply and rapidly for fifteen seconds $\times 4$) have been found to be of practical value. Tests of pulmonary diffusion are considerably more complicated and difficult to make. However in patients for whom pulmonary resection is necessary when the results of tests for pulmonary function are correlated with clinical information they are a valuable aid both in prognosis and in planning therapy.

Interference with pulmonary function may occur during and following operations and following trauma, particularly to the chest, and it may be associated with some degenerative diseases of the lungs. Ventilation of the lung may become decreased through interference with chest wall activity as seen following an injury or by obstruction of the air passages with foreign material such as blood, pus or mucus. Reduction of lung capacity by infection, neoplasm or injury may seriously reduce pulmonary reserve so that the diffusion of gases is inadequate. Chronic emphysema and fibrosis may materially interfere with internal mixing of gases or also produce a physiologic shunt of blood through un-aerated lung and thus lead to some degree of hypoxia. Advanced stages of fibrosis with intrapulmonary vasculitis may markedly impede the flow of blood through the lungs giving rise to cor pulmonale with varying degrees of cardiac decompensation. A knowledge of the degree of the above changes prior to pulmonary surgery is especially important in planning therapy.

Following operations on the upper abdomen or thorax, ventilation of the lung is decreased because of pain on respiration and restriction of motion of the chest wall. In pulmonary resection, there is an additional factor i.e., decreased lung capacity. Following operation one of three complications may result which may materially affect the postoperative convalescence of the patient by reducing pulmonary capacity

atelectasis, pneumonitis and physiologic shunting through an un-aerated lung distal to an obstructed bronchus. When mucopurulent secretions are retained in the air passages one of the above conditions or a combination of two or more may develop. With the absorption of entrapped air by bronchial obstruction atelectasis occurs which leads to an elevation in temperature and leukocyte count. If this condition involves an entire lung or lobe some retraction of the mediastinum toward the side of involvement and elevation of the diaphragm on the involved side result. These findings aid materially in differentiating atelectasis from lobar pneumonia, pleural effusion, pulmonary embolism, coronary thrombosis, acute dilatation of the heart or diaphragmatic hernia. If atelectasis persists it will usually lead to pneumonitis of the obstructed lung.

When secretions intermittently obstruct a large bronchus and the air beyond the obstruction is not absorbed, a physiologic shunt occurs. This means that blood passing through that portion of obstructed nonventilated lung does not pick up oxygen. Thus this venous blood returns to the left side of the heart, causing lowered saturation of the arterial blood oxygen. The resultant hypoxia may be sufficiently great to produce cyanosis. Since only approximately one-fourth of the lung capacity in normal individuals is necessary to oxygenate the blood adequately if dyspnea and cyanosis accompany retained bronchial secretions a physiologic shunt rather than atelectasis is present. The principles of management of these three conditions may be briefly enumerated as follows: *prophylaxis* should include early ambulation, postoperative breathing and coughing exercises, avoidance of restriction of respiration and the use of various aids for removal of excess secretions. *active therapy* requires stimulation of cough, removal of secretions by catheter suction or bronchoscopic aspiration, the use of expectorants postoperatively and the employment of tracheotomy when the patient is extremely ill and unable to help himself in eliminating secretions.

During intrathoracic operations on the normal chest, the lung on the side of operation tends to collapse because of its normal elasticity. In order to maintain adequate ventilation of the lung and diffusion of gases during surgery a mild intratracheal positive pressure is necessary in the majority of patients. This may be obtained by admin-

istering oxygen and anesthetic gases through a snug fitting face mask or by means of an endotracheal catheter. By properly employing either of these methods, ventilation of the lungs for maintenance of normal blood oxygen and pH levels can be adequate.

INJURIES OF THE THORAX

Injuries of the thorax may vary considerably in type and in degree of severity. In military casualties as well as in persons injured in automobile and industrial accidents wounds of the thorax have constituted one of the major causes of death. During World War I, 20 to 30 per cent of the deaths on the field of battle resulted from this cause. Only 4 to 8 per cent of the total wounded admitted to the clearing stations had chest injuries. Early mortality among those seen at advanced stations who had escaped immediate death was 25 per cent. Since fatalities usually occur early following a chest injury, it is indicated that prompt first-aid measures may materially reduce the rate of mortality. This was proved to be a fact for during World War II, when first-aid measures were given near the front lines to men having chest injuries deaths from these wounds were reduced to only a small fraction of those occurring during World War I. In the management of severe wounds of the chest, shock and hemorrhage from additional sites must be kept in mind as likely causes of the presenting clinical features. Disturbance of cardiorespiratory function, however may prevent improvement following adequate treatment of shock due to other causes.

For convenience of discussion, wounds of the thorax may be divided into nonpenetrating injuries in which only the chest wall is involved, without an opening into the pleural cavity penetrating injuries of the chest in which the entire chest wall as well as the pleura is involved, perforating injuries of the chest in which the entire thorax has been traversed by a foreign body, and blast or concussion injury of the lung in which damage of the pulmonary tissue was brought about by sudden exposure of the chest to extremely high pressure within a small enclosed area.

Injuries of the thorax may threaten life because of shock, hemorrhage alteration of cardiopulmonary function and infection. Early first aid measures of most importance are administration of oxygen, control of hemorrhage, correction of physical disturbance of cardiopulmonary function, replace-

ment of blood loss by the use of whole blood or plasma and prevention of infection.

Chest Injuries Requiring Immediate Attention. *Open or sucking wound with pneumothorax.* The ill effects accompanying this type of wound are chiefly those due to disturbance of cardiopulmonary function. The lung on the side of injury becomes collapsed and airless the mediastinum deviates toward the opposite side and the contralateral lung becomes hypoinflated. If the opening through the chest wall is larger than the size of the trachea, the condition is usually incompatible with life. Paradoxical respiration develops in which the mediastinum swings from side to side with respiration, thus preventing normal or adequate gaseous exchange in the remaining inflated lung. In addition to the ill effects on pulmonary function hemorrhage from the chest wall or lung may add to the clinical picture. Dyspnea and cyanosis in varying degrees of severity cough and hemoptysis are usually present.

First-aid measures should include immediate closure of the opening through the chest wall by whatever means are available, thus converting the open pneumothorax into a closed pneumothorax. Aspiration of air from the pleural cavity on the injured side will increase the pulmonary capacity and give some immediate relief of symptoms unless serious hemorrhage has occurred. Definitive treatment, when available should include hemostasis, débridement of the wound with removal of foreign material from the chest wall or intrathoracic area and removal of air from the pleural cavity by continuous suction drainage after closure of the chest wound. Replacement of blood loss and the use of oxygen and antibiotics are an important part of the treatment.

Tension pneumothorax. This condition is produced by a penetrating wound of the chest, in which the lung is injured by a fractured rib or a foreign body. As air escapes into the pleural cavity and is trapped, the pressure within the pleural cavity gradually increases, thus collapsing not only the lung on the side of injury but, to somewhat lesser extent, the lung of the opposite side (Fig. 1). In addition to the deleterious effects from the markedly reduced pulmonary capacity symptoms may also be due to hemorrhage within the pleural cavity. Ventilation of the opposite aerated lung is also diminished because of deviation of the mediastinum toward the contralateral side. This condition is recognized by the presence

of dyspnea, cyanosis and increased peripheral venous pressure. Physical findings are self-explanatory.

Management of tension (pressure) pneumothorax includes the use of continuous suction applied to the pleural cavity through an intercostal catheter for re-expansion of the lung and replacement of blood loss if hemorrhage has occurred. A small foreign body in the lung may be inconsequential and require no therapy. Foreign bodies over 1 cm. in size, however, especially when located near larger vessels, should be removed when facilities are available. The use of oxygen and chemotherapy is valuable in treating these subjects.

Unstable chest wall "flail chest." Multiple fractured ribs are frequent, however a double fracture of several ribs occurs infrequently. When several ribs are broken in two places, covering a segment of the chest wall, this area may become unstable. If the region is sufficiently large and tends to move inward on inspiration, thus interfering with ventilation of the lung, the condition is called a "flail chest." This injury usually results from a crushing blow to the chest by a flat object. It is accompanied by hemorrhage into the chest wall as well as into the pleural cavity. Lacerations of the underlying lungs

by the fractured ribs with resultant pneumothorax may occur. The clinical features attending this type of injury are due to interference with ventilation of the lung, decrease in pulmonary capacity from hemothorax and collapsed chest wall and loss of circulating blood volume.

In simple fracture of the rib, paravertebral blocking of the involved intercostal nerve roots with procaine is the treatment of choice. Control of pain by this method is preferable to strapping the chest with tape, since the latter tends to interfere with respiratory activity. Early therapy for flail chest entails stabilization of the chest wall by whatever means are available. Adequate ventilation of the remaining functioning lung tissue is then re-established. If a large segment of the chest wall is involved, massive collapse of the lung on the ipsilateral side may occur. This may be both from compression of the chest wall and from bronchial obstruction by blood or secretions (Fig. 2). Definitive treatment entails elevation and stabilization of the flail segment by traction. By this means, the underlying collapsed lung will be re-expanded and the normal size of the thoracic cage will be re-established. Additional therapy should include replacement of blood loss by transfusion and removal of air and blood from the pleural cavity. Oxygen and antibiotics should be used as indicated.

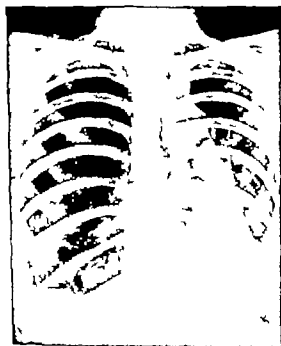


Figure 1. Tension pneumothorax. X-ray appearance of the chest in tension (pressure) pneumothorax in a twenty-eight year old woman who had developed a bronchopleural fistula secondary to cystic disease of the lung. Note the marked shift of mediastinal structures to the left with complete collapse of the right lung and partial collapse of the left lung.

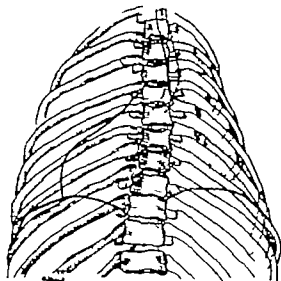


Figure 2. Flail chest. A "double fracture" of the right second to sixth ribs, inclusive, with massive collapse of the right lung is shown. The chest wall was staved in as a result of an automobile accident. Note also some shift of the upper mediastinal structures to the right, indicating obstruction of the air passages on that side. The drawing was made from an x-ray film of the chest of a fifty-four year old woman made shortly following the accident.

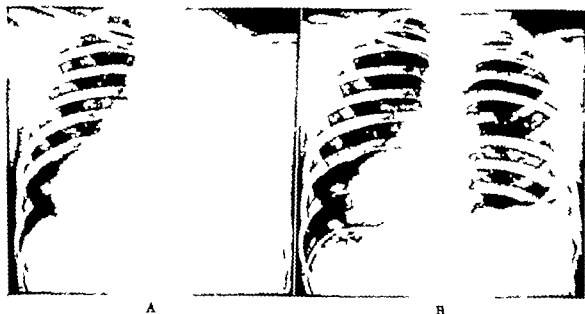


Figure 3 A, X-ray appearance of the chest of a thirty-two year old man six and one half months following an injury which caused a large hemothorax. The blood was not adequately removed. A layer of fibrous tissue "peel" developed over the lung and chest wall pleura with subsequent contraction of the chest wall. Pulmonary capacity as well as ventilatory function was greatly reduced. B, X ray appearance following removal of "peel" covering the lung and chest wall. Note normal re-expansion of the lung. Both pulmonary capacity and ventilation of the lung were improved.

Massive collapse of the lung (atelectasis)

Atelectasis may occur as the result of bronchial obstruction by blood or mucopurulent secretions or from compression of the lung by air or fluid within the thoracic cavity or by a collapsed flail chest resulting from a crushing injury of the chest wall. Since the clinical features of this condition are quite readily recognized, there is usually little difficulty in diagnosis. Treatment consists of eliminating the cause and management of the associated pathologic process as described above.

Hemothorax. Chest injuries may be accompanied by serious hemorrhage into a pleural cavity from one of three locations—an internal mammary artery, an intercostal artery or the lung. Since bleeding within the pleural cavity is "hidden," it may reach considerable proportions before its seriousness is evident. By this time the patient usually presents the usual manifestations of severe shock due to blood loss such as cold clammy skin, pallor, rapid thready pulse and lowered blood pressure. In addition to the usual symptoms caused by severe hemorrhage, local findings over the involved chest lead to the establishment of a correct diagnosis.

Treatment consists of replacement of blood loss, oxygen and antibiotic therapy and removal by aspiration of blood from the pleural cavity. If the blood pressure level

continues to drop after replacement of blood loss by transfusion, continued bleeding from a systemic artery is most likely present. Exploration of the wound or intrathoracic structures with control of bleeding is then indicated. If bleeding has stopped after replacement of blood loss, removal of all blood from the pleural cavity by aspiration should be completed within twenty-four hours. In the event that clotting of blood has occurred, one of two types of management should be followed: open thoracotomy with removal of clots and re-expansion of the lung or drainage of the pleural cavity through a catheter placed in the chest wall and the local use of enzymes for liquefaction of the clotted blood. If trauma is extensive, exploratory thoracotomy is to be preferred, since traumatized lung can then be removed or repaired. When a hemothorax persists any considerable time, a fibrous tissue "peel" forms over both the parietal and visceral pleurae. After this has occurred, an open operation and removal of the "peel" along with the remaining clotted blood and serum are necessary for re-expansion of the lung (Fig. 3).

Chest Injuries in Which Infection Is Likely to Occur Contaminated and lacerated wounds. Therapy in these patients consists of careful débridement combined with the administration of antibiotics and sulfonamides. When the pleura is involved,

open thoracotomy is indicated and closure is made, allowing for continuous suction drainage of the pleural cavity by catheter.

Foreign bodies. Since foreign bodies are apt to carry particles of clothing or other foreign material into the chest, infection of the pleura or other structures is apt to occur. Therapy will include measures used in contaminated wounds. In addition the size and location of the foreign body as well as the presence of infection will determine whether the object should be removed.

Lower chest injuries involving the upper abdomen. Since the large bowel and stom-

ach are above the lowermost level of the lung on the left side penetrating wounds in this region are apt to be accompanied by sepsis, as well as blood loss from injury to the spleen. Exploration of the upper abdomen and lower thorax is indicated and the damaged organs and tissues are treated as required.

Chest Injuries Not Usually Demanding Surgery. *Subcutaneous and mediastinal emphysema.* The seriousness of this condition is usually more apparent than real. The emphysema may result from penetrating, non-penetrating or crushing injuries of the chest.

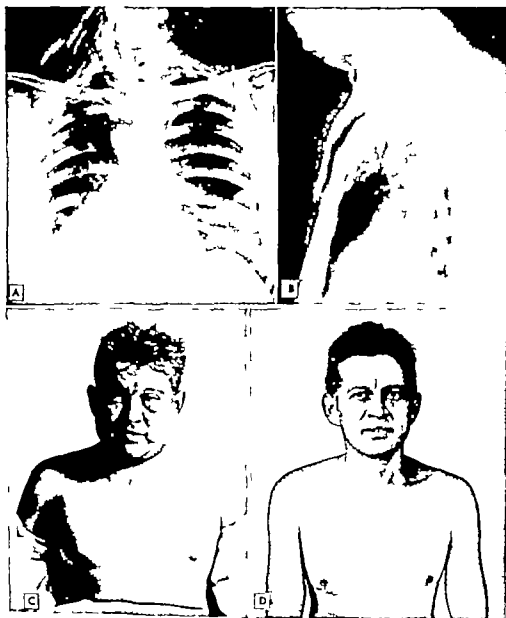


Figure 4. Subcutaneous and mediastinal emphysema. A and B, X-ray appearance of the chest, front and side views, of a forty-four year old man following the development of mediastinal and subcutaneous emphysema. This resulted from elevated pressure within the chest produced by repeated attacks of severe coughing caused by bronchial pneumonia. C and D Photographs of patient during the presence of emphysema and after it had disappeared. Dissection by the air through the subcutaneous tissues and along fascial planes extended up to the scalp and down to the pelvis. No treatment was required other than that directed toward control of the cough and the management of the pulmonary infection.

Air from an injured lung may dissect through the damaged chest wall to all layers of the soft tissues. In crushing injuries, air may escape along the bronchovascular roots of the lung to the mediastinum and pass up into the base of the neck and thence over the chest wall and up over the face and scalp (Fig. 4). Unless the air within the mediastinum tends to compress the large veins such as the superior vena cava, this condition is usually not serious and requires only control of the etiologic factor. The condition is usually easily recognizable since palpation demonstrates a crackling sensation due to the presence of air beneath the skin.

Traumatic asphyxia. This condition is caused by sudden compression of the thorax with the resultant forcing of venous blood back into the capillaries of the upper extremities, head and neck. This produces a bluish purple discoloration of the involved area. Although the appearance suggests an alarming condition, actually little or no alteration of cardiopulmonary function occurs. Proper management includes rest and the treatment of associated lesions.

Blast injury of the lung. This injury usually results from sudden severe compression of the chest by rapid elevation of atmospheric pressure about an individual in a confined area, such as a tunnel or a narrow street bordered by tall buildings. Cough with mild hemoptysis and symptoms and findings of mild shock and cerebral concussion are commonly present. Recovery usually occurs following a regimen of rest, oxygen therapy and adequate sedation. Death has

occurred, however, possibly from coronary air embolism due to air being forced into the coronary vessels.

Closed pneumothorax. When this condition is due to a penetrating or crushing wound of the chest, no symptoms may occur unless the trauma has been of such a severe degree as to produce collapse of the major portion of an entire lung. On physical examination the diagnosis is obvious and re-expansion of the lung may be accomplished by aspiration of the air. If the lesion recurs because of the presence of a bronchial fistula, continuous suction through an intercostal catheter usually produces permanent re-expansion of the lung.

CONGENITAL ANOMALIES OF THE THORAX

Malformations of the thoracic cage are not uncommon, however the majority are of no clinical significance. Occasionally anomalies are incompatible with life, such as absence of the diaphragm, an extremely high eventration of the diaphragm or partial absence of the thoracic wall which may be associated with ectopia cordis. Anomalies which have no particular clinical importance include forking of a rib anteriorly, fusion of two ribs and absence of a portion of a rib. Anomalies which may be of surgical significance are pectus excavatum (trichter breast, funnel chest), cervical rib and hernia of the lung.

Pectus excavatum. This anomaly consists of a congenital deformity of the sternum and adjacent cartilages and anterior segments of ribs. At times, there is associated a

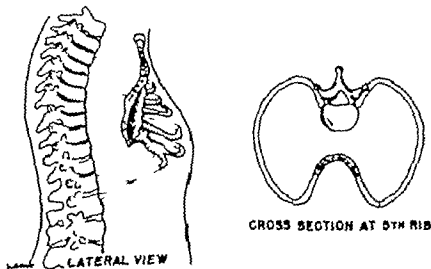


Figure 5. Pectus excavatum. Drawing of lateral (sagittal) and cross-sectional view of deformity of chest wall showing marked encroachment upon area (space) normally occupied by the heart. The heart is rotated farther into the left side of the chest as the deformity increases.



Figure 6 A and B Front and side x-ray appearance of a nineteen-year-old male with pectus excavatum. Note marked reduction of anteroposterior dimension of the mediastinum to less than 2 inches and some deviation of cardiac shadow to the left. C and D Postoperative appearance following surgical correction of defect. Note increase in A P dimension of mediastinum. Also note brace placed over the chest anteriorly to aid in maintaining elevation of sternum. E, Photograph showing scar and normal contour of chest wall following surgery

varying degree of scoliosis. The deformity usually begins at the junction of the first and second portion of the sternum and extends downward to and including the xiphoid process which is at the greatest backward extension of the deformity (Fig. 5) The significance of this deformity may be because of its psychologic effect, or its interference with cardiopulmonary function. In severe cases, the distance between the anterior surface of the thoracic vertebrae and the posterior surface of the sternum may not be more than 2 or 3 cm. (Fig. 6) Thus, the heart is rotated to the left and at times its activity is affected.

Treatment of this condition by a plastic operation for elevation of the sternum and adjacent involved cartilages and ribs is justified when the deformity either interferes with cardiopulmonary activity or is likely to interfere later if the degree of deformity is increasing. The operation is also indicated when the deformity is a psychologic handicap to the individual. The outlook following surgery is favorable.

Cervical Rib. Although in a high percentage of its subjects this deformity is of no clinical significance, at times it gives rise to a condition known as scalene syndrome. The anomaly usually occurs bilaterally but clinical

manifestations may involve only one side. These consist of numbness and tingling and a decrease in the normal temperature of the hand and forearm of the affected side. The clinical picture is caused by an interference with the blood supply to the arm resulting from compression of the subclavian artery and a portion of the brachial plexus between the scalenus anticus muscle and the cervical rib. Treatment consists of liberation of the constriction by surgical division of the scalenus anticus muscle. Favorable results following the operation usually can be anticipated.

Hernia of the Lung. This is a very uncommon condition. It may be congenital or may develop following trauma to the chest wall. The congenital type is a true hernia, with a sac of parietal pleura into which the lung protrudes, it occurs most commonly near the sternum or vertebrae through an intercostal space. The acquired type may follow an injury to any part of the chest wall, the lung prolapsing through a defect produced by the trauma.

The usual symptoms are a bulging mass, pain over the local area and cough. The size of the mass increases on coughing or straining. The cough is usually nonproductive but may bring forth bloody sputum. Incarcerated

tion and strangulation of these hernias have been reported.

Physical examination reveals a soft, crepitant, bulging mass which suddenly increases in size on coughing. A bony or fibrous ring may be felt through which a finger may be inserted into the chest cavity. The diagnosis is easily made.

Since these hernias rarely heal spontaneously treatment consists of freeing the contents of the hernial sac and closure of the defect in the chest wall by a plastic operation.

INFLAMMATIONS AND TUMORS OF THE CHEST WALL AND PLEURA

Inflammations of the Chest Wall. Subpectoral abscess. A subpectoral abscess or subpectoral phlegmon, is an uncommon although serious lesion and because of its location beneath heavy bellied muscles it is frequently misdiagnosed in its early development. The infection begins in the subclavicular glands at the extreme apex of the axilla and is usually caused by a hemolytic streptococcus secondary to an infection or injury of the hand, arm, axilla or breast. It also may follow an injury of the chest wall or of the supraclavicular fossa.

The subclavicular glands lie adjacent to the axillary vein high in the axilla and just beneath the costocoracoid membrane. When the infection extends outside of these glands it spreads downward and outward beneath this fascia and at times may extend posteriorly around the chest wall to the subscapular region.

Since the infection lies deeply hidden from the surface local symptoms and findings are insignificant in the early stage and are usually preceded by fever, chills and general malaise. Pain in the shoulder and arm, especially on adduction or rotation, rigidity of the pectoralis major muscle and a sensation of tenderness or pain near the middle of the clavicle are the first local manifestations. Examination reveals no early local abnormalities, but later swelling and a fullness near the outer inferior border of the pectoral muscle with tenderness over this area are noted. Much of the swelling may be due to edema and the lack of fluctuation is misleading, often delaying the diagnosis.

Treatment consists of incision and adequate drainage along the outer inferior margin of the pectoralis major muscle. Additional drainage by division of the muscle fibers beneath the clavicle may be necessary.

Sulfonamides, penicillin and streptomycin are helpful in controlling the spread of the infection but should not be relied upon as a substitute for surgical drainage. Because of the location of the abscess pyemia and septicemia are not uncommon. The condition is always serious and the prognosis must be guarded.

Subscapular abscess. A subscapular abscess is much less common than a subpectoral abscess and usually produces few local or systemic symptoms. Although caused by pyogenic organisms it may present the appearance of a cold abscess. At times it is differentiated from tumors only by the presence of fluctuation. Treatment consists of adequate drainage after determination of the etiologic organism by aspiration.

Acute osteomyelitis. Acute osteomyelitis of a rib may result from drainage of an empyema due to trauma or general staphylococcal infections. With the exception of the last mentioned, the systemic involvement is much less common than that produced by infection in other bones. Involvement of the cut end of the ribs following rib resection for draining an empyema is uncommon and produces no systemic reaction. Occasionally an entire rib may become involved and sequestrate. Surgical removal of the sequestrum is followed by complete healing. Osteomyelitis following trauma to a rib produces no special problem and is treated in the usual manner for this lesion. Osteomyelitis of the sternum is much less common but when present carries a high mortality.

Typhoid osteomyelitis of a rib is rarely seen, since the incidence of typhoid fever has been reduced. The local condition may not appear for months or years after an attack of typhoid fever, however a history of an attack is usually present. Locally a fluctuant swelling develops but signs of an acute inflammation are lacking. Its recognition is important in order to prevent spread of the organisms before the diagnosis is made. Typhoid osteomyelitis must be differentiated from tuberculosis, syphilis, sarcoma and chronic pyogenic infections of the rib. Aspiration and culture along with a history of typhoid fever are usually sufficient for establishing the diagnosis. Treatment consists of surgical excision of the involved bone.

Tuberculous Tuberculosis of the sternum, ribs and cartilages is seldom seen since pulmonary tuberculosis has been brought under better control. It may develop by one of two methods—either by direct extension

from tuberculous glands lying just outside the parietal pleura, usually near the sternum or by hematogenous spread from an active tuberculous lesion elsewhere in the body. The former route is probably the much more common, since in many of the patients the tuberculous infection is around the rib or cartilage, with no evidence of infection within these structures.

Although this infection may occur at any age, it is commonest during middle life. The lesion presents local manifestations similar to those produced by cold abscesses elsewhere. There is little evidence of local inflammation, a fluctuant nontender swelling indicating its presence. If it is allowed to progress, necrosis of the skin occurs, with sinus formation. The infection may however extend quite some distance along a rib or cartilage before sinuses develop. Aspiration of the swelling reveals thick, curdy pus. If a sinus is present, injection of a radiopaque material and x-ray examination of the chest will aid in determining the limits of the involvement.

Treatment consists of wide surgical excision of the involved rib or of the entire cartilage, if the latter is involved. Streptomycin has been found effective in the treatment of tuberculous sinuses when no foreign body is present. In the absence of sinuses or secondary infection, closure of the wound without drainage is indicated. Supportive treatment as for tuberculosis elsewhere is helpful.

Actinomycosis. Actinomycosis of the chest wall is practically always secondary to actinomycosis of the lung or intestine. In the former the process extends outward through the pleural cavity and in the latter extension is up through the diaphragm. Since this infection respects no tissue boundaries, it involves all structures of the chest wall and slowly produces a brawny induration in the soft tissues, with considerable swelling and edema. A productive cough, usually with hemoptysis, and local pain and tenderness are experienced along with the constitutional symptoms of fever, weakness, sweats, anorexia and loss of weight. If the disease process is allowed to continue, discharging sinuses are produced.

This condition may be mistaken for tuberculosis of the chest wall or tumor of the lung with secondary invasion of the chest wall. The diagnosis is established by the finding of sulfur granules in the exudate and the ray fungus on staining material removed by aspiration or biopsy technique.

Treatment consists of wide excision of the involved parts usually performed best in several stages. The use of thymol and potassium iodide by mouth and thymol locally has been of definite benefit in some subjects. More recently penicillin has been found to be much more effective in the majority of patients. The prognosis must be guarded in advanced lesions; however the use of adequate amounts of penicillin has greatly improved the outlook for these patients.

Inflammation of the Pleura. Acute empyema. Inflammations of the pleura are all ways secondary to inflammation of the lung and parietes of the thorax or to trauma. With the advent of chemotherapy for the treatment or prevention of pneumonia, pyogenic empyema as a complication of pneumonia is seldom seen. Likewise, the clinical picture is no longer characteristic as was the case before specific therapy was available. This condition is seen more commonly today as a complication of subphrenic abscess, lung abscess or bronchiectasis or following trauma. The causative organism in empyema as a complication of pneumonia is usually a pneumococcus or a streptococcus. With another etiologic basis, it may be one of the two above or one of a fairly large variety of either aerobic or anaerobic or ganglions.

In the early stage of this infection, the pleural exudate is serous in nature and may occupy the entire pleural cavity or only a limited space (encapsulated empyema). More frequently it occupies the lower portion of the pleural cavity, the apex of the lung becoming attached to the upper chest wall. As fibrin deposition occurs and inflammatory cells, such as lymphocytes, leukocytes and endothelial cells increase, the exudate thickens. This occurs much more rapidly with pneumococcal than with streptococcal empyema. As this process continues the exudate becomes frank pus and in reality an abscess of the pleural cavity occurs. With this development, the lung becomes adherent to the chest wall at the edges of the involved pleural space. In pneumococcal inflammation, this process requires five to seven days to develop whereas in the presence of streptococcal infection or other aerobic and anaerobic organisms, it may require two or three weeks or longer.

The clinical manifestations of empyema thoracis are those of an inflammatory process in addition to those of a partially collapsed lung. Fever and leukocytosis with some pain in the involved area are characteristic. Be-

cause of the presence of pain or the large size of the pleural space involved, dyspnea and elevation in pulse and respiration occur in varying degrees. Examination of the chest reveals decreased motion as well as decreased to absent breath sounds on the involved side. The percussion note is dull to flat and cardiac dullness may be shifted to the opposite side. Tubular breathing and rales are not infrequently present adjacent to the area of involvement.

In view of the usual presence of an etiologic factor the diagnosis of empyema thoracis is not difficult. An x ray film of the chest, exposed with the patient either sitting or standing, will reveal an opacity in the region of involvement. If this is in the lower portion of the pleural cavity, the opacity will extend outward and upward as a curved shadow along the chest wall. When the empyema is encapsulated, the shape and size of the opacity are quite variable and this lesion may be confused with lung abscess or a tumor with secondary infection. Fortunately the encapsulated type of involvement is seldom seen and thus this difficulty in diagnosis is not often confronted.

When an empyema is suspected, thoracentesis should be performed to confirm the diagnosis. If the empyema cavity extends to the base of the pleural space aspiration is made through the seventh intercostal space in the postaxillary line. If during the procedure, a cough is incited or exacerbated, the procedure should be discontinued until the cough is controlled by sedation.

Once the diagnosis of empyema is established, a knowledge of its extent and location of involvement is desirable. For this purpose a diagnostic pneumothorax is induced by introducing air into the empyema cavity following the removal of exudate. In order to determine the causative organism, smears and cultures of the removed exudate should be made in order to plan proper therapy.

The treatment of acute empyema will vary somewhat according to the etiologic basis and the causative organism. In addition to chemotherapy methods of management include aspiration, closed catheter drainage and open drainage. Chemotherapy according to the sensitivity of the organisms, should be instituted immediately if it is not already in use. If the empyema has already reached the abscess stage when diagnosis is made, adequate open dependent drainage is the treatment of choice. This can be readily obtained by the resection of a

segment of rib in the most dependent portion of the cavity with the patient under local anesthesia. An adequate opening must be maintained for the free drainage of exudate until the cavity is obliterated. The principles of therapy at this stage of the disease are the same as those for a pyogenic abscess elsewhere in the body. Prior to the formation of frank pus repeated aspiration by means of thoracentesis or closed suction drainage through the use of an intercostal catheter introduced into the empyema cavity should be employed. In this way deleterious effects due to an open pneumothorax, with perhaps deviation of the mediastinum toward the opposite side are avoided during the stage when severe toxic manifestations from the primary cause of infection are present. If treatment is started by aspiration or the closed type of drainage, this should be changed to open drainage when the exudate becomes purulent. When repeated thoracentesis or closed drainage by catheter is utilized, introduction of the needle or trocar through the chest wall should be made at the upper edge of a rib in a dependent location. In this way intercostal vessels and nerves will not be traumatized. This procedure can be carried out with the patient under local anesthesia and in a semisitting position in bed or on an operating room table. Various irrigating solutions have been of benefit in some patients; however the principal object is to obtain adequate drainage. In addition to chemotherapy and drainage of the empyema space the use of blood transfusions, oxygen therapy and improvement of the nutritional state are important adjuncts to proper management.

When a bronchopleural fistula develops during the acute stage of empyema, serious disturbance of cardiorespiratory function may occur because of tension pneumothorax as seen in Figure 7. Continuous suction drainage through an intercostal tube or open drainage as indicated will give immediate relief.

The prognosis of empyema will depend largely upon the etiologic factor if adequate and prompt therapy is administered. When this condition occurs as a complication of pneumonia, the prospect of a cure is excellent. Empyema as a complication of lung abscess or bronchiectasis may take on a serious aspect and prognosis must be guarded.

Staphylococcus empyema. *Staphylococcus aureus* is the only organism involving this region which is not controlled by the most

commonly used antibiotics and, therefore more attention is being focused upon it. Staphylococcus empyema occurs much more frequently in young children and in elderly people and may be unilateral or bilateral. The disease is characterized by rapidly developing toxicity with chills, fever and dyspnea and at times cyanosis. Being bronchial pneumonia in type, early pleural involvement is the rule. The rapid progressive development of pleural effusion unresponsive to the usual antibiotics is presumptive evidence of a staphylococcus infection. However culture of aspirated pleural fluid is necessary for a definite diagnosis. Because of the rapid necrotizing effect on the lung, early effective management is mandatory. Multiple lung ab-

cesses frequently with bronchopleural fistula and resultant pyopneumothorax, are commonly seen in inadequately managed cases. Multiple, smaller lung abscesses may coalesce leading to pneumatocele formation.

Treatment consists of chemotherapy, evacuation of pleural exudate and supportive measures. Although a number of antibiotics have been used alone or in combination for this infection, the most effective have been Chloromycetin with erythromycin and Chloromycetin with novobiocin. These drugs should be used in full therapeutic amounts and continued for two weeks following recovery. Repeated removal of pleural exudate by thoracentesis frequently leads to pocket formation, inadequate evacuation and com-

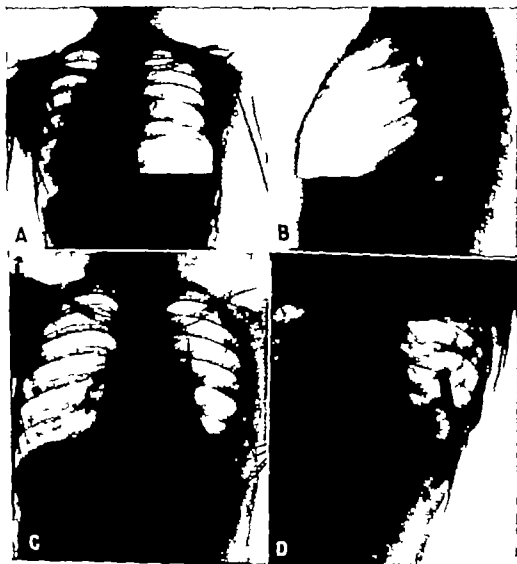


Figure 7. A and B, Front and side x ray appearance of an eight-year-old girl with pyopneumothorax under considerable tension due to the presence of a bronchopleural fistula. This patient experienced marked respiratory distress due to the tension pneumothorax as well as presenting manifestations of toxicity. C and D. X-ray appearance of chest five and fourteen weeks, respectively following open drainage of the empyema. Note progressive re-expansion of the lung with only a small sinus tract filled with radiopaque material remaining. The patient made an excellent recovery.

plications. Thus, unless satisfactory results are obtained with one or two pleural taps early intercostal tube drainage by suction as soon as the diagnosis has been made is the procedure of choice. Supportive measures include the judicious use of oxygen, salicylates and sedatives and maintenance of electrolytic and water balance. With prompt adequate management, the prognosis is good. If treatment is delayed or inadequate, open thoracotomy to remove fibrous tissue peel or to obtain adequate drainage may be necessary and with this is an associated increase in morbidity and mortality.

Figure 8 shows x-rays of the chest of an infant of ten months following admission to the hospital with fever, dyspnea and cough of three or four days duration. The patient had been given penicillin. The patient was found to have a temperature of 40° C., was dyspneic and presented crepitant rales in the midzone region of the left lung poste-

riorly. There was a hemoglobin of 12 gm. and a white blood count of 14,800. A diagnosis of pneumonia of the left lung was made and the patient was immediately started on a regimen of Chloromycetin, 250 mg. every six hours, erythromycin, 30 mg. every eight hours and aspirin. A water mattress was to be used for temperature elevation above 39° C. A positive growth of hemolytic *Staphylococcus aureus* was found on throat culture the following day. An admission x-ray of the chest showed infiltration in the midzone of the left lung (Fig. 8A). There was some elevation of the left leaf of the diaphragm. The patient ran a spiking fever with a temperature up to 40° C. during the next three or four days. An x-ray of the chest made six days later revealed sparse areas within the left lung, as well as pneumothorax. There was some increased elevation of the left leaf of the diaphragm with mild deviation of the mediastinum to the

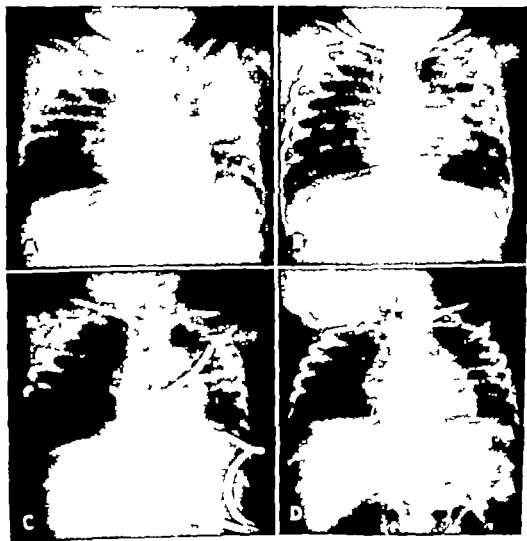


Figure 8. Intercostal drainage in staphylococcus empyema (see text for explanation)

right (Fig. 8B) The patient's condition became somewhat improved. However closed drainage with two intercostal tube drains was instituted which completely expanded the lung with subsequent disappearance of the cavities (pneumatoceles within the lung) (Fig. 8C) The patient's course subsequently was smooth and uneventful. The temperature returned to normal and all evidence of previous involvement within the lung disappeared (Fig. 8D)

Chronic empyema The most common cause of chronic empyema is inadequate drainage of an acute empyema. This may be due to lack of dependent drainage, inadequate drainage opening or cessation of drainage before closure of the cavity. Lack of dependent drainage is the most common causative factor. Occasionally chronic empyema occurs as a result of a foreign body in the cavity such as a sequestrum from an osteolytic rib following rib resection or from drainage material. Drainage of tuberculous empyema has at times been instituted before sufficient information as to the causative organism was secured, thus resulting in chronic empyema.

In chronic pyogenic empyema, a small pleurocutaneous sinus with intermittent drainage of small amounts of exudates is not infrequently present. Likewise, a communication between the empyema cavity and the bronchial tree (bronchopleural fistula) is frequently seen. The clinical features in the chronic form of empyema include a pale undemourished individual showing various degrees of anemia and low-grade leukocytosis. The chest wall on the affected side is shrunken and immobile. Breath sounds over the involved area are absent and the percussion note is dull to flat.

Usually, on the basis of etiologic factors alone, a diagnosis of chronic empyema may be readily suspected. An x ray examination reveals the area of involvement to be opaque, with or without a fluid level in the cavity. If a bronchopleural fistula or a draining sinus of sufficient size for the admission of air into the space is present, a fluid level will be seen.

After the diagnosis has been established, adequate open drainage, usually by the removal of segments of two ribs in a dependent location, should be made. Improvement in the patient's general condition should also be brought about through the use of transfusions, chemotherapy, vitamins and a nourishing diet. Inspection of the cavity

should be made for the presence of foreign material and the cavity cleaned by irrigation.

Within a period of two or three months through open drainage alone the cavity may be reduced considerably in size. In addition and of even greater importance in the preparation for more adequate surgery the patient's general condition will be materially improved. In most instances dependent open drainage in itself will be inadequate for complete obliteration of the cavity. Although, in the past, emphasis has been placed on obliteration of the residual cavity by collapsing the overlying chest wall, at the present time the principle of therapy is based upon mobilization of the empyema wall and surrounding lung, removal of both visceral and parietal portions of the fibrous empyema wall and sufficient reinflation of the underlying lung for obliteration of the pleural space. If the lung tissue adjacent to the area of the empyema is adequately mobilized, the object of the operation will be accomplished. Adequate replacement of blood loss is of paramount importance in this procedure. Closed drainage of the pleural space by use of an intercostal catheter following closure of the chest wall should be rigidly maintained during the postoperative period.

Tuberculous empyema. This condition is almost always secondary to tuberculosis of the lung. Tuberculous empyema is seldom seen at the present time, since the use of pneumothorax therapy for tuberculosis has been largely abandoned, or is not pushed beyond its limitations and effective chemotherapy has become available for the treatment of pulmonary tuberculosis. In tuberculous empyema, the exudate in the pleural space may vary greatly in density from an extremely thin serous-like material to one fairly purulent in character. In the presence of a bronchopleural fistula, the pleural space may become secondarily infected by pyogenic organisms, in which case the condition is termed "mixed tuberculous empyema." This form of infection carries greater clinical significance and therapy must be based on different principles than in the presence of a pure tuberculous empyema. In the pure infection, symptomatology will vary little from that of the primary condition within the lung. Physical findings are those characteristic of the presence of fluid within the pleural space. In mixed tuberculous empyema, symptomatology will include greater

elevation in temperature and leukocytosis. Likewise, systemic manifestations of a pyogenic infection may be present.

Whereas therapeutic measures for purely tuberculous empyema may vary little from those for the original condition within the lung, therapy for mixed tuberculous empyema will include adequate dependent surgical drainage and chemotherapy for control of the pyogenic organism. If the pulmonary lesion can be adequately controlled, some consideration should be given to an open operation for removal of the localized pulmonary infection by resection, in addition to management of the empyema cavity. Chemotherapy, the use of blood transfusions and attention to the nutritional state of the patient are important aspects of the over-all management of the mixed tuberculous type of infection.

Tumors of the Chest Wall and Pleura. Various tumors of connective tissue origin, such as lipoma, lymphangioma, hemangioma, osteoma and chondroma, may develop in the

chest wall. The most common is chondroma or osteochondroma, which arises from a rib, cartilage or the sternum. Many of these tumors are malignant, especially those of a cartilaginous nature. Because most of them grow toward the pleural cavity they may reach a considerable size before producing symptoms or before their presence is discovered. The tumor frequently arises at a costochondral junction and, owing to its slow growth, symptoms may be entirely lacking until adjacent structures are affected by pressure. All structures of the chest wall are usually involved by the growth, which is more or less spherical in shape, hard and nodular with areas of softening due to degeneration. Since metastases are late, the tumor is apt to be considered benign and differentiation is frequently difficult or impossible even on microscopic examination. Of the 213 tumors of the chest wall analyzed by Hedblom 61.4 per cent were sarcomas. In the absence of metastases, differentiation between the benign and the



Figure 9 Chondroma (see explanation in text)

malignant tumor is impossible before operation. These tumors may also be confused with a dermoid cyst, cold abscess, aneurysm and exostosis.

Figure 9 illustrates the case of a thirty-five-year-old white female who had noted the presence of a tumor of the chest for the preceding seven years. She had experienced fleeting pains beneath the right breast during that time. The tumor had increased slowly during that period, but no symptoms were present on admission to the hospital. Physical findings were normal except for a tumor mass which was firm, nodular and nontender and projected out from the left chest wall for a distance of 2 to 3 cm. over an area from the third to the sixth ribs and from the right border of the sternum to the postaxillary line, being approximately 13 cm. in diameter (Fig. 9A). The tumor was removed through an incision over the fifth right interspace from the sternum to the spine. This also included removal of portions of the fourth, fifth and sixth ribs anterolaterally as well as other elements of the chest wall (Fig. 9B). The breast was elevated during this procedure and was then used to aid in filling the defect. The patient had an uneventful convalescence. An x-ray of the chest following surgery showed only mild reduction in the size of the pleural space on the operated side (Fig. 9C). The tumor mass was spherical in shape, projecting almost entirely into the pleural cavity. It was firm in consistency and quite nodular throughout and measured approximately 14 cm. in diameter. Macroscopic examination showed this to be a chondroma (Fig. 9D).

Treatment consists in wide surgical excision. When the tumor is large, no attempt should be made to preserve the underlying parietal pleura. Positive pressure anesthesia must be used for dealing with the surgical pneumothorax produced. If a large defect in the chest wall remains following removal of the tumor it can frequently be filled in by the breast or scapula. Plates of Lucite, secured to adjacent ribs or cartilages, have been successfully employed in bridging large defects. Good results may be expected in a benign lesion, but recurrence is the rule following extirpation of a sarcomatous tumor.

A common malignant tumor described by Pancoast arises in the superior pulmonary sulcus. Although the etiologic source has been questioned, it is epithelial in nature and is

now classified as bronchogenic in origin. This tumor gives rise to a characteristic clinical picture of pain in the shoulder, wasting of the muscles of the hand and Horner's syndrome. Roentgenograms reveal a sharply defined opacity in the extreme apex of the pleural cavity with destruction of one or more of the first three ribs and at times the adjacent vertebrae. X-ray therapy may give symptomatic relief and prolong life in some of the patients. There is no other known effective therapy.

Other malignant tumors frequently seen in the ribs are metastatic carcinoma from the breast and multiple myelomas. The latter are more easily detected here than in other bones.

Tumors of the pleura may be primary or metastatic. The latter are seen much more commonly than the former and are usually secondary to tumors of the breast or lung. A serous or blood-tinged fluid is formed and is usually the first sign of the tumor's presence. Pain of a variable nature is usually experienced. Large quantities of fluid may form, with as much as several hundred cubic centimeters being present and recurring over a period. Examination of the fluid by centrifuging, fixing and staining will usually reveal the presence of malignant cells, especially when the fluid is bloody in character. X-ray examination of the chest will show the presence of fluid and frequently also an eroded rib.

Primary tumors of the pleura include endothelioma, mesothelioma and sarcoma. These are rarely seen and do not present a characteristic clinical picture. Pleural effusion may also accompany these lesions, although it occurs less frequently than in the presence of metastatic tumor. Treatment is by excision if the growth has not extended beyond the limits of surgery.

READING REFERENCES

- Allen, D. S. The Treatment for Penetrating Wounds of the Pleural Cavity. *Arch. Surg.* 21:1161, 1930.
 Blades, B. Penicillin as an Adjunct to the Surgical Treatment of Acute and Chronic Empyema. *Ann. Surg.* 121:672, 1945.
 Blades, B., and Paul, J. S. Chest Wall Tumors. *Ann. Surg.* 131:976, 1950.
 Burford, T. H., Parker, E. F., and Sampson, P. C. Early Pulmonary Decortication in the Treatment of Posttraumatic Empyema. *Ann. Surg.* 122:163, 1945.
 Craig, W. M., and Knepper, P. A. Cervical rib and the Scalene Anticus Syndrome. *Ann. Surg.* 105:556, 1937.

- Graham, E. A. A Reconsideration of the Question of the Effects of an Open Pneumothorax. *Arch. Surg.* 8:345, 1924.
- Graham, E. A. Some Fundamental Considerations in the Treatment of Empyema Thoracis. St. Louis, C. V. Mosby Company, 1925.
- Haight, C. Intratracheal Suction in the Management of Postoperative Pulmonary Complications. *Ann. Surg.* 107:218, 1938.
- Hedblom, C. A. Tumors of the Bony Chest Wall. *Ann. Surg.* 98:528, 1933.
- Johnson, J. Battle Wounds of the Thoracic Cavity. *Ann. Surg.* 123:321, 1946.
- Langston, H. T. and Tuttle, W. M. The Pathology of Chronic Traumatic Hemothorax. *J. Thoracic Surg.* 16:99, 1947.
- Ravitch, M. M. The Operative Treatment of Pectus Excavatum. *Ann. Surg.* 129:423, 1949.
- Samson, P. C., and Burford, T. H. Total Pulmonary Decortication. *J. Thoracic Surg.* 16:127, 1947.
- Snyder, H. E. The Management of Intrathoracic and Thoracoabdominal Wounds in the Combat Zone. *Ann. Surg.* 122:333, 1945.
- Thomas, G. I., and Jarvis, F. J.: Decortication in Primary Tuberculous Pleuritis and Empyema with a Study of Functional Recovery. *J. Thoracic Surg.* 32:178, 1958.
- Watkins, D. H. Surgical Complications in the Thorax of Staphylococcal Pneumonitis. *Arch. Surg.* 77:508, 1938.
- Watkins, E. Jr. and Hering, A. C.: The Management of Staphylococcal Tension Pneumatoceles by Intracavitary Suction Tube Drainage. *J. Thoracic Surg.* 36:642, 1958.
- Zuckerman, S. Experimental Study of Blast Injuries to the Lungs. *Lancet* 2:219, 1940.

Diseases and Tumors of the Lungs and Bronchi

By BRIAN BLADES M.D.
OWEN GWATHMEY M.D. and PAUL C. ADKINS M.D.

BRIAN BLADES the son of a Kansas doctor, was educated at the University of Kansas and Washington University Medical School. He received his training in thoracic surgery at the hands of Everts Graham and was one of the early disciples who established and extended the principles upon which this specialty rests. Following a distinguished service in thoracic surgery during World War II, he was appointed to the chair of surgery at George Washington University School of Medicine in 1946.

OWEN GWATHMEY is the son of a practicing country physician in King William County, Virginia. Like his father, he was educated in Virginia and graduated from the Medical College of Virginia. He was trained in surgery at George Washington University and is now in practice in Richmond, Virginia.

PAUL CHARLES ADKINS was born in Massachusetts and received his college and medical education at The Johns Hopkins University. He was trained in surgery at The Johns Hopkins Hospital and the hospital of The George Washington University, where he is now Assistant Professor of Surgery.

A thorough knowledge of the anatomy of the trachea and lungs is essential in the evaluation and treatment of pulmonary disease.

The trachea is approximately 11 cm. in length. It contains cartilaginous rings for

support, which circumvent the organ except posteriorly. The trachea divides at the carina into the right and left main stem bronchi. The right main stem bronchus angulates slightly at the bifurcation. It gives off bronchi to the right upper lobe, right

middle lobe and right lower lobe. The left main bronchus is longer than the right and has a sharper angulation at the carina. The left upper lobe arises at a greater distance from the carina than its counterpart on the right. The bronchus continues as a left lower lobe bronchus and on both sides the lobar bronchi divide into tertiary bronchi to form the segments of the lung. On the left, the lingula of the left upper lobe arises from the left upper lobe bronchus and is a counterpart of the right middle lobe. The segmental bronchi divide into ramifications forming bronchioles, alveolar ducts and sacs.

The latter contain no cartilage and are therefore, spongy in nature. Each segment is separate and usually communicates with the adjacent segments, allowing collateral ventilation. This phenomenon is known as air drift. The segments are conical in shape with the apex toward the hilum (Fig. 10).

The main pulmonary artery divides into the right and left pulmonary arteries in the mediastinum. The right pulmonary artery crosses the right main stem bronchus on its anterior aspect and courses downward. It gives off two or three large branches to the apical and the posterior apical segments. It

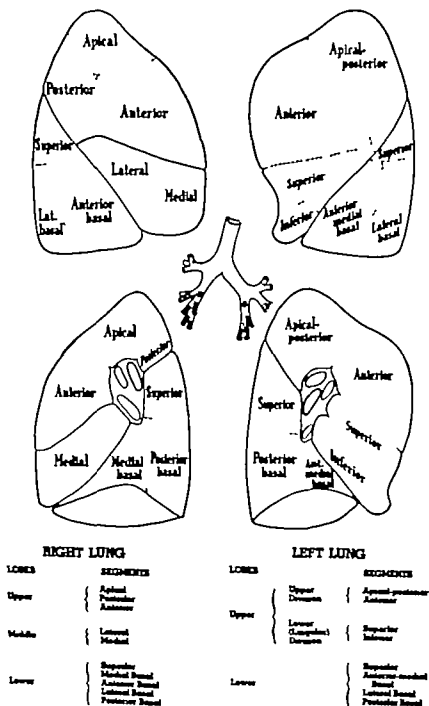


Figure 10. The bronchopulmonary segments. (From Jackson and Huber.)

descends posteriorly beneath the superior pulmonary vein and gives off a branch to the anterior segment of the upper lobe. This branch lies in the fissure between the superior and middle lobes and gives off two branches to the middle lobe. It continues in the fissure as a lower lobe artery and gives off branches to the corresponding bronchial segments.

The left pulmonary artery arches over the left main stem bronchus to descend in the fissure between the left upper and lower lobes. It has a longer main trunk than the right. It gives off branches to the apical and then to the apical posterior segments as it arches around the main stem bronchus. Continuing in the fissure are two to three segmental branches to the anterior segments. Just below this one or two branches are given off to the lingular segment of the left upper lobe and the apical segmental artery to the apex of the lower lobe. At this level, the vessel becomes the lower lobe artery and continues as a common trunk for 2 or 3 cm. where it divides into the basilar segmental arteries.

The segmental veins of the right upper lobe and the right middle lobe converge to form the right superior pulmonary vein. The vein crosses in front of the lower lobe artery and enters the left atrium. The inferior pulmonary vein is formed by the segmental veins from the right lower lobe and enters the left atrium with the right superior vein.

The segmental veins of the left upper lobe and lingula form the left superior pulmonary vein which crosses in front of the bronchus just inferior to the left pulmonary artery and enters the left atrium. The left inferior pulmonary vein is formed from the segmental veins of the lower lobe. It courses directly to the mediastinum and enters the left atrium with the other veins.

The bronchial arteries arise from the undersurface of the distal aortic arch. They pass to the anterior surface of the bronchi near the lateral margin. Their distribution is segmental. The bronchial veins drain segmentally and enter the azygos and hemi azygos veins.

The lymphatic drainage of the lungs is divided into superficial and deep plexuses. The superficial ones are just beneath the pleura while the deep accompany the bronchi, pulmonary arteries and pulmonary veins. Both communicate freely and drain into the lymph nodes of the pulmonary hilum and mediastinum. For the most part,

these channels drain superiorly although they may drain inferiorly toward the abdomen. There are cross-anastomoses from one hilum to the other.

The primary function of the lung is the transfer of oxygen into the arterial blood and the removal of carbon dioxide from the venous blood. This is accomplished by two important pulmonary functions, ventilatory and respiratory.

Ventilatory function is the transport of gases through the respiratory passages to and from the alveoli. Respiratory function is the absorption of oxygen and the elimination of carbon dioxide from the blood stream. These two functions may be altered alone or together by disease processes.

The surgeon should learn in advance the status of the ventilatory and respiratory functions before surgery of the lungs is considered, since decrease in either function may result in a cardiorespiratory cripple after an operation affecting the lung.

The mechanism of normal ventilatory function depends upon certain factors: a mobile and intact thoracic cage; negative intrathoracic pressure; unobstructed larynx and tracheobronchial tree; elasticity of the lung and properly functioning diaphragms. On inspiration, the intrapleural pressures become more negative as a result of expansion of the thoracic cage and descent of the diaphragms. The lungs expand, the bronchi and bronchioles dilate and elongate and air is sucked into the lungs. Upon expiration the reverse occurs and the lungs decrease in size.

Ventilation can be affected by diseases of the larynx and the trachea or bronchi, lung parenchyma, chest wall or diaphragm.

Respiratory function depends upon the permeability of the partition between the alveolar sacs and capillary lumen. Diseases of the lung parenchyma may injure the alveolar capillary membrane and impair oxygen absorption and carbon dioxide elimination. The exchange of gases through the alveolar capillary membrane depends upon the difference in the partial pressures (gradient) of the gases in the alveolar sacs and the arterial and venous capillaries. Normally the partial pressure of carbon dioxide in the alveoli is between 35 and 45 mm. Hg, whereas in mixed venous blood, it averages 45 mm. Hg. The partial pressure of oxygen in the alveoli varies from 100 to 120 mm. Hg and in mixed blood it varies from 30 to 40 mm. Hg. Hence the diffusion of oxygen will be from the alveolar spaces to the blood and

carbon dioxide from the blood into the alveoli spaces. Even though ventilation is normal parenchymal disease can alter greatly respiratory function causing an increase of carbon dioxide accumulation in the blood and decrease in oxygen consumption.

Lung volume measurements and determination of oxygen and carbon dioxide transport across the alveolar capillary membrane may be necessary in evaluating patients for resectional surgery. However in patients who give no history of pulmonary insufficiency examination of the respiratory rate and pulse rate before and after exercise is a valuable pulmonary function test. The condition of the patient after walking rapidly up a flight of stairs also gives a reliable impression of pulmonary reserve.

The efficiency of the bellows action of the chest and lungs may be obtained from spirometric tracings and from the residual air capacity measurements. The timed vital capacity is of more importance in determining the efficiency of ventilation than is the vital capacity alone. The vital capacity is the volume of air which is collected between maximum inflation and maximum deflation of the lungs. The timed vital capacity is the volume of air which is rapidly exhaled from maximum inflation of the lungs and collected over a three-second period. It should equal the predicted vital capacity. If it is less, pulmonary insufficiency is present and ventilation is impaired.

The maximum breathing capacity (MBC) is the volume of air which is moved in and out of the lungs over a given period, while the patient is breathing as deeply and rapidly as possible. Normal for man is 120 liters per minute, if the maximum breathing capacity is 40 liters or less then a significant degree of insufficiency is present. Normal measurements of the three-second timed vital capacity and maximum breathing capacity indicate that there is normal lung elasticity no obstruction of the airways and good movement of the diaphragm.

Residual air studies are helpful in determining the degree of pulmonary insufficiency. Normally residual air occupies about 25 per cent of the total lung capacity and is the volume of air present in the lung following maximum expiration. A volume of 40 per cent or over indicates pulmonary insufficiency.

Bronchspirometric studies determining the amount of oxygen consumption and ventilation of each lung may be helpful in patients with bilateral disease.

The determination of oxygen and carbon dioxide tension in the arterial blood is important in determining the transport of oxygen and carbon dioxide across the alveolar capillary membrane. These measurements should be obtained both at rest and following exercise. Oxygen consumption minute ventilation tidal volume arterial oxygen saturation, the arterial blood p_{H_2} , the arterial carbon dioxide content and the carbon dioxide combining power are necessary to establish these values.

PULMONARY TUBERCULOSIS

It is important to emphasize that pulmonary tuberculosis is a chronic systemic disease. Surgical treatment, therefore affects only the predominant manifestation usually the organ or part of an organ most seriously involved. The discovery and widespread use of the antituberculosis drugs have furnished a method to help control the systemic aspects of tuberculosis, which has not only made earlier and successful operations possible but has sharply decreased morbidity following all types of surgical intervention.

Early clinical diagnosis of minimal tuberculosis is difficult. The disease may mock the common cold, grippé influenza and, if severe and fulminating, pneumonia. Physical signs cannot be relied upon to establish the diagnosis but may be suggestive. Rales in the upper lung fields diminished breath sounds and other physical signs have been described as typical of tuberculosis. Regardless of physical signs, roentgen ray examination of the chest is essential. Emaciation hemoptysis and other evidences of terminal tuberculosis are now rarely seen as presenting signs and symptoms.

The nationwide availability of chest x ray surveys has resulted in the detection of many early cases of tuberculosis. Everyone, young and old, should have a roentgenogram of the chest made at least once a year not only for tuberculosis detection but for the detection of other diseases of the lungs.

The final and absolute diagnosis of pulmonary tuberculosis cannot be made until tubercle bacilli are found in the sputum. A single positive sputum examination is not reliable and must be duplicated and, preferably confirmed by culture. Failure to observe these precautions has resulted in the hospitalization of patients for long periods with an erroneous diagnosis of tuberculosis.

Until effectual chemotherapeutic agents were available in the treatment of tuberculosis surgical therapy was largely confined

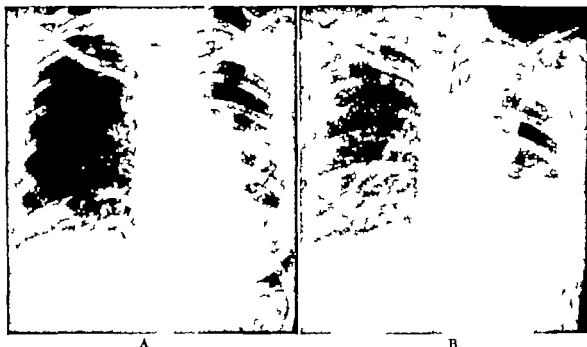


Figure 11 A, Roentgenogram of chest showing destruction of the left lung. There was stenosis of the left main bronchus. B Appearance of the chest following pneumonectomy. Many surgeons would perform a thoracoplasty at the time of pneumonectomy or later to prevent overdistention of the remaining lung.

to operations designed to collapse or partially collapse the diseased lung to put the organ at rest. If bed rest did not result in healing of the lesion, artificial pneumothorax sometimes combined with internal pneumonolysis, phrenic nerve paralysis, pneumoperitoneum and, finally collapse of the chest wall by thoracoplasty were accepted procedures.

A great variety of operations have been suggested for tuberculosis: division of the scalene muscles to relax the apex of the lung, extrapleural pneumonolysis, cavity drainage oleothorax, paraffin plompage and extrapleural pneumothorax. Except under unusual circumstances these operations are obsolete.

Tuberculosis is a multiform disease and there has always been some disagreement, even among the most skilled, in the management of each specific problem. Even with the most recent advances each case requires individualization in the decision for the exact type of surgical approach and the timing of the operation.

Artificial Pneumothorax. Collapse of the lung by introduction of air into the pleural cavity is now rarely undertaken but was a very popular method of treatment in the past. Although the procedure is easily performed, the complications of artificial pneumothorax may be serious, including development of serous fluid, empyema, bronchopleural fistula or an obliterative pleuritis

with associated loss of pulmonary function. The practice of always attempting artificial pneumothorax before recommending more radical surgical treatment has been discontinued.

Pneumoperitoneum. Elevation of the diaphragms by the introduction of air into the peritoneal cavity has been more popular in the past decade. The principal use for the procedure is in those patients who do not respond to chemotherapy alone or whose condition is too serious for major surgical intervention. There are sharp differences of opinion concerning the value of pneumoperitoneum, but many believe the incidence of sputum conversion and cavity closure with combined use of chemotherapy and pneumoperitoneum is significant. Contra-indications include chronic fibroid disease with thick walled cavities, caseous foci and endobronchial tuberculosis. A history of abdominal disease with subsequent operation is a positive contraindication to the introduction of air into the peritoneal cavity.

Thoracoplasty Thoracoplasty was reserved until other operations had failed in the early days of the surgical treatment of tuberculosis. Later cases were selected on the basis of extent and location of disease and permanent collapse of the diseased lung tissue became a popular and valuable operation. With the increasing popularity of pulmonary resection for tuberculosis, however the number of thoracoplasties performed is

diminishing. The undesirable features include leaving diseased tissues in the body and depending upon relaxation and collapse to cause permanent healing. Moreover thoracoplasty may cause varying degrees of deformity. It appears from early results that excision of the diseased lung tissue, combined with chemotherapy will give better results than collapse therapy. At the present time, the principal value of thoracoplasty usually a limited one is to decrease the size of the pleural space after the removal of a lobe or a lung. This procedure will help prevent overdistention of the remaining lung tissue and will decrease the incidence of bronchopleural fistula. Depending upon the patient's condition and the circumstances of the case, the thoracoplasty can be performed at the time of the lobectomy or pneumonectomy or may be delayed until a later date. Some surgeons believe that thoracoplasty is particularly important following pneumonectomy to prevent overexpansion and shift of the remaining lung, which in turn might result in reactivation of tuberculous foci in the apparently sound lung.

Pulmonary Resections for Pulmonary Tuberculosis. The majority of patients with predominantly unilateral disease, in whom response to chemotherapy and bed rest is not satisfactory are now treated by removal of the diseased portion of the lung. Depending

upon the extent of the disease the operation may be pneumonectomy (Fig. 11), lobectomy (Fig. 12) segmental resection of a lobe (Fig. 13) or in some instances a simple wedge resection of the involved tissues. This approach to the surgical treatment of pulmonary tuberculosis has been made possible by the discovery and use of the chemotherapeutic agents effective against the disease. Moreover during the past two decades there have been perfections of techniques for pulmonary resection methods for the evaluation of pulmonary function and tremendous advances in anesthesia. The concept of removing the diseased lung tissue in patients having pulmonary tuberculosis is more than a half-century old but could not be accomplished with safety until rather recently.

The present tendency is to recommend removal of tuberculous foci much earlier than would have been acceptable a few years ago. The work of Medlar and his associates has strongly influenced this trend. They contend that necrotic, caseous tuberculous foci are important in instances of clinical recurrence after apparent arrest. They caution against operations if tubercle bacilli can be found in the sputum and depend upon the antituberculosis drugs to control this factor. And finally they justify operations on small lesions to conserve func-

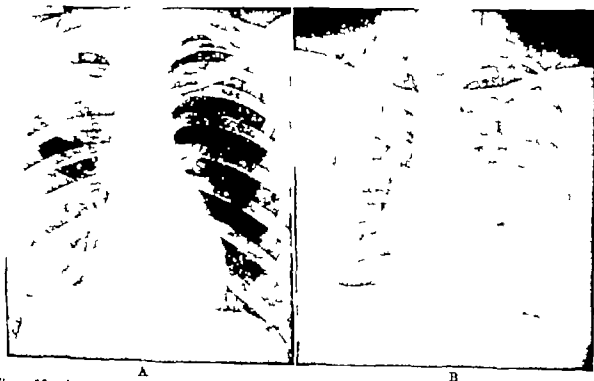


Figure 12. A, Roentgen ray appearance of disease of the right upper lobe. B, Postoperative roentgenogram after upper lobe lobectomy and limited thoracoplasty.

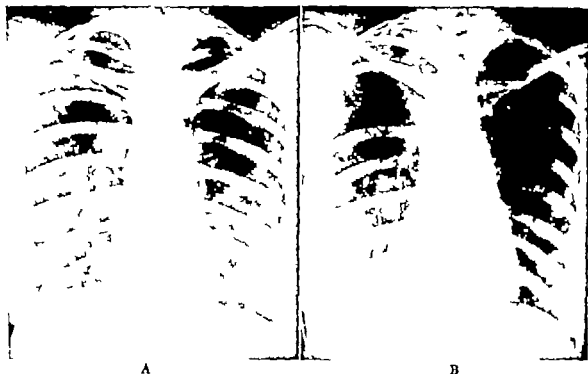


Figure 13. A, Minimal lesion located in the right upper lobe. B Postoperative x-ray appearance of the lung after removal of the posterior and apical segments of the right upper lobe.

tion of the lung tissue and point out that the most dangerous pulmonary disease is located in the superior and posterior portions of the pulmonary lobes. Modern surgeons, therefore rely upon the antituberculous drugs to control the systemic phase of the disease and often excise the predominant, demonstrable foci as delineated on the x ray film. Opportunities to operate upon earlier lesions have been made possible by the widespread use of x ray surveys for the detection of the disease.

The first infection of the lung by the tubercle bacilli usually is followed by rapid healing and the formation of a Ghon's complex. If the host's resistance to tuberculosis is low there may be a rapid spread with caseation and exudation with little fibrosis. Scarring and fibrous encapsulation may come later as resistance increases. There is usually marked enlargement of the hilar lymph nodes associated with the initial pulmonary lesion if it is progressive.

Reinfection tuberculosis is the type which usually is of interest to the surgeon. These lesions are characterized by marked fibrous reaction and little tendency to further involve local lymph nodes. The degree and seriousness of reinfection tuberculosis depend principally on the host's resistance. The late manifestations of reinfection are scars, usually in the upper lobe and often in the apical or posterior segments of the up-

per lobe. These lesions may or may not be associated with caseation. The mechanism which accounts for localization in the posterior portions of the upper lobes is not understood.

The allergic reaction to the tubercle bacilli has some bearing on the types of disease. Caseation and abundant exudation may be present if the allergic factor is predominant. In instances where encapsulation of the disease is extreme, a small, round focus may present and produce the formation of a tuberculoma. If liquidation and necrosis continue, there will be cavity formation with communications with one or more bronchi. In patients with good resistance, the surrounding fibrous connective tissue will be laid down in an effort to check the disease. These lesions are often termed "fibrous tuberculosis." Surgical extirpation combined with antibiotic therapy is usually required to obtain a satisfactory result when the disease has reached this stage.

The diversified nature of tuberculosis results in a wide variety of pathologic lesions. Moreover there are sharp differences of opinion concerning the mechanisms involved.

There is practically complete agreement that if a thoracoplasty has failed to arrest the disease, pulmonary resection will be necessary. The presence of tuberculous bronchostenosis or bronchiectasis constitutes an

other positive indication for excisional surgery as do instances of extensive lung destruction. In patients who have had thoracoplasty without conversion of sputum, lobectomy or pneumonectomy will be necessary. Giant cavities and lower lobe disease are also considered indications for extirpation of the involved lobe or lobes.

The presence of a suspected tuberculoma (Fig. 14) usually demands excision of the involved tissue. The most important reason for excision of this lesion is to differentiate it from a bronchogenic carcinoma, since the roentgen ray appearance may be identical and, secondly, tuberculomas of any size may undergo cavitation and result in active disease.

Chemotherapy The discovery of streptomycin by Schatz and Waksman, in 1944 was followed by Hinshaw's demonstration, in 1945 that the drug was effective in clinical tuberculosis. After a trial-and-error period, it was found that the combination of para-aminosalicylic acid (PAS) and streptomycin reduced the hazard of bacterial resistance to streptomycin used alone. Both in medical treatment and in preparation for any type of surgery chemotherapy is uniformly employed. In addition, following operation, drug therapy is of utmost importance to prevent spread of disease and to aid in healing, particularly of the divided bronchus. Drug therapy is continued after operation as protection against late complications or reac-

tivation of disease. The length of time the antituberculosis agents are employed depends upon the needs of each individual patient.

The isonicotinic acid derivatives, particularly isoniazid or isonicotinic acid hydrazide introduced a few years ago are now used in combination with both streptomycin and PAS depending upon the strains of tubercle bacilli resistance to the various drugs. New antimicrobial agents are constantly being discovered and the exact pattern of drug therapy will probably change as time goes on.

LUNG ABSCESS

A lung abscess is a circumscribed collection of pus within the pulmonary parenchyma. Pulmonary gangrene is a massive destruction of lung tissue exceeding that commonly seen with the ordinary lung abscess. These conditions, therefore are similar only varying in extent. The widespread use of the various antibiotics has made lung abscess a rare disease.

Single or multiple lung abscesses can result from a variety of causes. Probably the most common is aspiration of infected material during operations on the upper respiratory passages, the mouth and nose. Unconsciousness for any reason increases the chance of aspiration of foreign material. Infected emboli in the blood stream may lodge in the lung and result in abscess formation. Extension of bronchiectasis into the lung parenchyma or infections distal to obstructing carcinomas may produce the condition.



Figure 14. A tuberculoma demonstrated by x-ray film. The differential diagnosis between bronchogenic carcinoma and tuberculosis could not be made until the tissue was excised.



Figure 15. A large lung abscess caused by bronchogenic carcinoma. This possibility must always be considered, particularly in older patients.

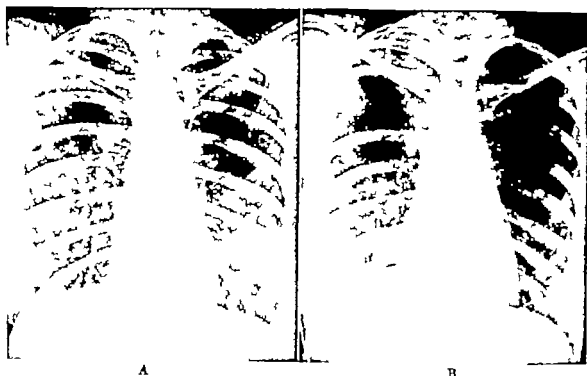


Figure 13. A, Minimal lesion located in the right upper lobe. B Postoperative x ray appearance of the lung after removal of the posterior and apical segments of the right upper lobe.

tion of the lung tissue and point out that the most dangerous pulmonary disease is located in the superior and posterior portions of the pulmonary lobes. Modern surgeons therefore, rely upon the antituberculous drugs to control the systemic phase of the disease and often excise the predominant, demonstrable foci as delineated on the x-ray film. Opportunities to operate upon earlier lesions have been made possible by the widespread use of x ray surveys for the detection of the disease.

The first infection of the lung by the tubercle bacilli usually is followed by rapid healing and the formation of a Ghon's complex. If the host's resistance to tuberculosis is low there may be a rapid spread with caseation and exudation with little fibrosis. Scarring and fibrous encapsulation may come later as resistance increases. There is usually marked enlargement of the hilar lymph nodes associated with the initial pulmonary lesion if it is progressive.

Reinfection tuberculosis is the type which usually is of interest to the surgeon. These lesions are characterized by marked fibrous reaction and little tendency to further involve local lymph nodes. The degree and seriousness of reinfection tuberculosis depend principally on the host's resistance. The late manifestations of reinfection are scars, usually in the upper lobe and often in the apical or posterior segments of the up-

per lobe. These lesions may or may not be associated with caseation. The mechanism which accounts for localization in the posterior portions of the upper lobes is not understood.

The allergic reaction to the tubercle bacilli has some bearing on the types of disease. Caseation and abundant exudation may be present if the allergic factor is predominant. In instances where encapsulation of the disease is extreme, a small, round focus may present and produce the formation of a tuberculoma. If liquidation and necrosis continue, there will be cavity formation with communications with one or more bronchi. In patients with good resistance, the surrounding fibrous connective tissue will be laid down in an effort to check the disease. These lesions are often termed "fibrocaseous tuberculosis." Surgical extirpation combined with antibiotic therapy is usually required to obtain a satisfactory result when the disease has reached this stage.

The diversiform nature of tuberculosis results in a wide variety of pathologic lesions. Moreover there are sharp differences of opinion concerning the mechanisms involved.

There is practically complete agreement that if a thoracoplasty has failed to arrest the disease, pulmonary resection will be necessary. The presence of tuberculous bronchostenosis or bronchiectasis constitutes an



Figure 16. Roentgenograms showing the appearance of an atelectatic middle lobe typical of a middle lobe syndrome

diseased lobe. At operation enlarged, firm lymph nodes are present which compress the proximal portion of the middle lobe bronchus. This is particularly striking on the inferior surface of the bronchus.

The middle lobe is atelectatic with varying degrees of fibrosis and associated bronchiectasis and multiple small abscesses.

The same clinical and roentgenologic findings may result from occlusion of the bronchus by bronchogenic carcinoma. This possibility must always be considered in cases of the middle lobe syndrome.

CHRONIC SUPPURATIVE PNEUMONITIS

The definite etiologic agent for chronic suppurative pneumonitis is unknown. The condition also has been referred to as non putrid pulmonary suppuration and chronic sclerosing pneumonitis.

This nonspecific disease produces signs, symptoms and pathologic changes which are fairly consistent and justify its description as a separate disease entity.

The onset is usually insidious without a history of an acute respiratory illness. The most common symptoms are chronic cough, chest pain and, later hemoptysis. Symptoms are usually of weeks or months duration with gradual increase in severity. There is usually little or no weight loss. Fever if present, is low grade.

The physical findings are vague with some increased dullness over the involved lung and frequently coarse rales.

X rays of the chest will show diffuse infiltration and sometimes atelectasis. Bronchoscopic examination will usually reveal in



Figure 17. The roentgenographic appearance of chronic suppurative pneumonitis. An identical roentgenogram may be seen in cases of bronchogenic carcinoma.

In a patient in the older age group with a lung abscess, the possibility of a carcinoma should always be considered seriously (Fig. 15). Trauma is a possible but unusual, etiological factor.

Pyogenic organisms of any type may be found in lung abscesses, including streptococci, pneumococci, staphylococci and fusiform bacilli. If the pus in the abscess is foul smelling, both aerobic and anaerobic organisms are often present. The putrid lung abscess, common before antibiotics were available, is now a rare lesion. Apparently the general use of chemotherapy destroys some of the bacteria and, particularly the symbiosis between anaerobic and aerobic organisms which produces the necrotizing, foul-smelling infections.

Before penicillin, streptomycin and the more recently discovered antibiotics were available lung abscesses constituted a very serious disease and fulminating pulmonary gangrene was usually fatal.

If the abscess did not empty spontaneously by coughing, or sometimes with the aid of bronchoscopy drainage operations were necessary. The operation was dangerous and the convalescence long. If the abscess became chronic the prognosis was even more serious.

In the formative stage before liquefaction of the lung tissue has produced a visible cavity or copious amounts of foul-smelling sputum, it is impossible to make a differential diagnosis between pulmonary abscess and pneumonia, consequently, almost all patients receive antibiotic therapy immediately. It is most likely that a certain number of persons with lung abscess recover from a pulmonary infection without the lesion progressing to abscess formation or the diagnosis becoming apparent. Most lung abscesses can be aborted by antibiotic therapy unless a cavity develops. Even under these circumstances, postural drainage, bronchoscopic aspiration and antibiotic therapy may cause healing of the lesion. Even if the abscess does heal promptly via utilization of the bronchial tree with Lipiodol at a later date may show residual bronchiectasis.

If the lung abscess does not respond to conservative therapy most surgeons now prefer to excise the lesion rather than to drain it, since the incidence of residual bronchiectasis after drainage of lung abscesses is high. The amount of lung tissue removed will depend upon the extent and location of the abscess. Occasionally in de-

bilitated and poor risk patients, external drainage is safer. This must be done through an adherent pleura and it is often necessary to excise the lobe involved at a later date to obtain an entirely satisfactory result.

FRIEDLANDER'S PNEUMONIA

Pulmonary infections caused by Friedländer's bacillus (*pneumobacillus Bacillus mucosus capsulatus* or *Klebsiella pneumoniae*) may produce pneumonic consolidation which is usually lobar in type associated with multiple abscesses and minute necrotic foci. As the disease progresses the necrotic foci may liquefy and form large cavities. If the lesions become chronic, organization and fibrosis of the diseased tissue produce a dense, heavy granulation tissue.

If the disease does not respond to medical therapy usually with Achromycin, streptomycin or other antibiotics, depending upon sensitivity studies of the bacteria, extirpation of the involved lung parenchyma should be recommended. In Friedländer's bacilli infections this usually involves lobectomy and, since these infections do not follow segmental boundaries, removal of the entire lobe or lobes involved is necessary. Antibiotic therapy must be continued during and after the operation. The prognosis in persistent cases of Friedländer's bacillus disease of the lung is poor because even after operation the disease may appear in the same or contralateral lung.

MIDDLE LOBE SYNDROME

The right middle lobe is particularly vulnerable to atelectasis with associated suppurative changes. This probably results from compression of the middle lobe bronchus by lobar lymph nodes. The middle lobe bronchus is at the apex of the lymphatic channels to both the upper and lower lobes and is, therefore, particularly subject to impingement of lymphadenopathy.

The symptoms are commonly chronic cough with or without productive sputum, recurrent attacks of pneumonia with fever and hemoptysis. Any or all of the symptoms may be present.

Physical signs of consolidation of the middle lobe are usually present. X-rays will show atelectasis of the middle lobe and if bronchograms are made a block of the middle lobe bronchus is usually demonstrated. On bronchoscopy the middle lobe bronchus is markedly stenotic with associated edema and inflammation (Fig. 16).

The proper treatment is extirpation of the

have had heavy sedation and depression of respiration. Pain alone may restrict portions of the chest wall with resulting accumulations of viscid material. Endobronchial tumors, aspirated foreign bodies and bronchial stenosis have the same effects (Fig. 19).

Prevention consists of careful nursing care, frequent change of position and encouraging the patient to cough and breathe deeply particularly after having a general anesthetic. If atelectasis should develop, it should be relieved at once either by coughing or by endotracheal aspiration with a catheter or by bronchoscopy if necessary. Chemotherapeutic agents should be employed prophylactically to prevent residual infections even after the collapse has been relieved.

CONGENITAL ANOMALIES OF THE LUNG

Occasionally there may be congenital absence of an entire lung. More frequently however one lung has remained in a fetal state and never expanded. Tremendous variations may be found in the locations of interlobar fissures and not infrequently in the number of lobes in the lung. A fairly common and sometimes troublesome anomaly is an aberrant bronchial artery rising directly from the aorta and entering the lung. These vessels may be large and, if injured during an operative procedure, may cause serious hemorrhage. If a pulmonary lobe or lung has remained in the fetal state, it should be removed because, in most instances, infection, bronchiectasis and hemorrhage will occur as the patient's age progresses.

Another common congenital anomaly is the accessory lobe of Wrisberg, or azygos lobe. Its chief importance is that it may be confused with other conditions when detected on a roentgenogram of the chest. An azygos lobe is found in less than 1 per cent of persons having x-ray examination.

True congenital cysts of the lung are relatively rare and in the past have been confused with emphysematous blebs or bullae. They are very similar or identical with bronchogenic cysts which may occur in the mediastinum. They may be thin walled, containing clear fluid, or a mixture of solid type of lesion with some fluid. These cysts have the characteristic epithelial lining found in the remainder of the respiratory passages. Pressure on adjacent thoracic structures may cause symptoms. Eventually the cysts become infected and therefore should be removed. Hemoptysis is a rela-



Figure 20. A huge emphysematous bleb displacing the mediastinum to the right. Both on roentgen ray examination and clinically confusion with tension pneumothorax is easy. Removal of the bleb resulted in re-expansion of the left lung.

tively rare complication. The proper treatment is surgical extirpation.

There has been some confusion in the terminology concerning cystic bronchiectasis and congenital cystic disease. If the cysts are multiple, bronchograms may give the impression of cystic bronchiectasis. More often, however the cyst cannot be filled with the radiopaque medium.

Emphysematous blebs and bullae are much more common than true congenital cysts. These lesions are commonly found in patients with pulmonary emphysema and asthma. The exact mechanism in the formation of these lesions is not understood. Loss of elasticity of lung tissue is certainly a prime factor and, particularly in patients with asthma, plugging of the bronchioles with mucus may cause dilatation distal to the obstruction. This situation might occur after any inflammatory disease of the lung.

With the development of giant air cysts, surgical intervention may be necessary. The cysts may be so huge that collapse of adjacent lung tissue will have serious effects on cardiorespiratory function (Fig. 20). In extreme cases, pneumatocele may occupy the entire hemithorax and produce the so-called vanishing lung phenomenon. Under these circumstances, the communication to the air cyst through the bronchus is usually of

flammatory changes in the bronchus to the affected part of the lung and varying amounts of purulent drainage (Fig. 17)

Chronic suppurative pneumonitis usually occurs in the older age group and it is often impossible to differentiate from bronchogenic carcinoma. This is even true when the lung is exposed at operation. The grossly involved lung tissue is in most cases infiltrated and atelectatic. Hard nodules may be present and also small zones of necrotic tissue. Final differentiation may depend upon microscopic sections.

In chronic suppurative pneumonitis there is extensive interlobar fibrosis and thickening of the alveolar walls. There is often considerable lymphoid and plasma cell hyperplasia.

The proper treatment is excision of the diseased lung tissue and a more radical operation if the lesion proves to be secondary to a tumor.

Attenuation of acute pneumonia or other suppurative disease of the lungs by antibiotic therapy may play a role in the production of a slowly progressive chronic lesion.

STAPHYLOCOCCAL PNEUMONIA

Infections with virulent antibiotic resistant staphylococcus appear to be increasing. Involvement of the lungs with staphylococcal

pneumonia may produce conditions which require surgical intervention.

The most common complication is empyema. With or without associated empyema, however there may be serious damage to the lung parenchyma with consolidation and multiple small abscess formation. The abscesses may become confluent and develop cystlike lesions or pneumatoceles. This is particularly true in children. Excision of the irreparably damaged lung tissue may be necessary after the acute pneumonia has subsided (Fig. 18)

BRONCHOLITHIASIS

Broncholiths or lung stones, which usually result from the erosion of a calcified lymph node into the bronchus may cause bronchial obstruction. Infection distal to the obstruction, if not relieved, may destroy the affected portion of the lung. Excision of the diseased lung tissue may be necessary. Broncholithiasis is often associated with severe hemoptysis which will require surgical intervention.

ATELECTASIS

Atelectasis, or imperfect expansion of the lung, may be the result of a variety of mechanisms. The entire lung, a lobe or a segment of the lung may be involved. The most common cause for this condition is the blocking of a bronchus or bronchiole with viscid mucus. The spontaneous removal by coughing, or by endotracheal aspirations, will result in rapid re-expansion of the involved zone. The condition is common following operations, especially in patients who



Figure 18. The roentgenographic appearance of an unresolved staphylococcal pneumonia. The acute phase of the pneumonia was controlled by antibiotics, but the right lower lobe was irreparably damaged. It was necessary to perform a right lower lobe lobectomy before the disease could be cured.



Figure 19. Atelectasis of the right lower lobe as shown by roentgen ray examination.

may not establish either the presence or the extent of bronchiectasis. Precise diagnosis and localization depend upon delineation of the tracheobronchial tree with a radiopaque medium.

It is also important to note that, after any inflammatory process bronchograms may show some cylindrical dilatation of the bronchi. As the inflammation subsides, the bronchi may return to normal. If radiopaque studies are made soon after pneumonia of

any type, a false impression of bronchiectasis may be obtained. This has been termed pseudo or reversible bronchiectasis. Delay of three or four weeks in preparing bronchograms after an acute infection will prevent this error in diagnosis.

Bronchiectasis is most commonly confined to the basal segment of one of the lower lobes associated with disease of the right middle lobe or the lingula of the left upper lobe (Fig. 21). Except in patients in whom

Fig. 21



Fig. 22



Figure 21. Bronchogram showing bronchiectasis of the left lower lobe and lingula of the left upper lobe.

Figure 22. Bronchiectasis of the right upper and middle lobes. Location of the disease in the upper lobes suggests a tuberculous origin.



Fig. 23

Figure 23. Bilateral bronchiectasis involving both lower lobes, the right middle lobe and lingula of the left upper lobe. In young patients in good general condition, bilateral lobectomy is feasible. In the older age group, conservative management is usually preferable.

the ball valve type with air entering the cavity but not escaping in sufficient quantity. In addition to the mechanical factors, therefore there is created an unusually large dead air space which will dilute tidal air requirements. In instances of single large, giant bullae excision of the lesions is usually followed by gratifying improvement.

Single or multiple bullae on the surface of the lung may occur in otherwise healthy young people and rupture of one or more of the lesions produces a sudden and sometimes severe pneumothorax. Interstitial rupture of a bleb with dissection of the air along the line of cleavage of the bronchi and blood vessels may produce mediastinal emphysema with progression to the neck and face. These lesions may be unilateral or bilateral. Spontaneous pneumothorax may occur on one side or both.

In a patient with a small pneumothorax without severe symptoms aspiration with a needle may result in re-expansion of the lung and formation of pleural adhesions with no further difficulty. If at the time of the first pneumothorax, aspiration fails to control the intrapleural pressures and a leak continues, catheter drainage with water seal may be necessary as an emergency measure. Most patients with this severe condition will require surgical intervention. If two or more episodes of spontaneous pneumothorax have occurred, surgical treatment should be recommended.

In the past, various sclerosing agents have been injected into the pleura to produce adhesions and prevent further leaks either through the thoracoscope or with needle and syringe. Modern treatment of spontaneous pneumothorax, however consists of thoracotomy excision of visible blebs and sterile talc poudrage of the pleura to promote strong adhesions. Water seal drainages should be maintained after the operation until the lung is completely re-expanded. Before operation is performed upon patients for spontaneous pneumothorax, the possibility of tuberculosis must be considered and ruled out.

BRONCHIECTASIS

The term "bronchiectasis" means dilatation and thickening of the wall of the bronchi or bronchioles. Almost always a chronic inflammation will occur in the dilated bronchioles which becomes permanent in nature. As the disease progresses there is thickening of the walls of the bronchi and bronchioles and a loss of elasticity. Accumu-

lations of infected material lead to a cough productive of thick sputum in the untreated patient.

Occasionally the first manifestations of the lesion will be bleeding from erosion in the walls of the affected portion of the lung. Hemoptysis from bronchiectasis may be severe and not infrequently results from erosion of a bronchial artery which is enlarged as a consequence of inflammatory changes accompanying the disease.

Usually two types of the disease are considered—congenital and acquired.

The congenital type is extremely rare. When it does occur it is due to developmental defects of a lobe or lung which become infected and produce the clinical manifestations of bronchiectasis usually at an early age. There is considerable controversy on the precise differentiation of cystic disease of the lung and congenital bronchiectasis.

The acquired variety is much more common and usually follows pneumonia, whooping cough, measles or inflammations of the lung from any cause. A persistent atelectasis may produce residual bronchiectasis. Obstruction of the bronchi by foreign bodies or tumors may result in bronchiectasis distal to the obstruction. The reduction in the incidence of cases of bronchiectasis since antibiotics are available indicates clearly the predominance of the acquired type of disease.

The classical picture of bronchiectasis including chronic cough, foul-smelling sputum and, in the late stages, hemoptysis and clubbing of the fingers, is now rarely encountered. The patient is more apt to complain of easy fatigue and some chronic cough, which usually is not strikingly productive. These changes in the clinical appearances result from the frequent and early use of antibiotics. There is a history of frequent colds, grippe or pneumonia like attacks and sometimes associated nasal sinusitis. Bronchiectasis is an insidious slowly progressive disease and the patient may not be aware how handicapped he is until the condition is relieved. Pleuritic pain is a common and often ignored early indication of disease in patients with little cough and sputum. In the far-advanced stages, there are fever, chills and progressive emaciation.

Physical signs may be absent or consist of impaired resonance, distant breath sounds or extensive coarse bubbling rales.

It is important to emphasize the fact that the conventional roentgenogram of the chest

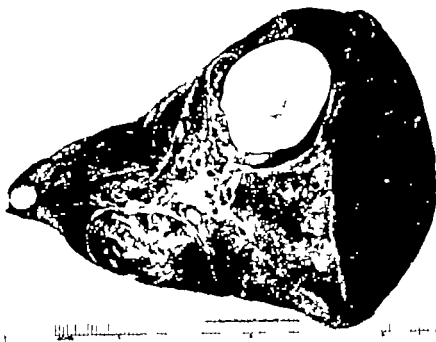


Figure 24 Echinococcus cysts involving the left lower lobe. The wall of the cyst in the superior division of the lobe has been removed and the fibrous tissue reaction of the bed is apparent. The second intact cyst is seen in the basilar division.

lobectomy and segmental resection constitute desirable methods of treatment. Care should be taken during operation to remove the cyst intact to prevent spillage and anaphylactic reactions.

FUNGUS INFECTIONS

Histoplasmosis. Asymptomatic histoplasmosis may cause granulomatous deposits in lung tissue. Differentiation from early bronchogenic carcinoma and tuberculoma may be difficult. Positive reactions to skin tests and complement fixation tests however may be helpful. Some authors believe that tuberculous lesions in the form of so-called tuberculomas have been confused with histoplasmosis even after the lesions have been excised.

Blastomycosis. Blastomycosis may occur in the lung, but it is rare and usually the skin manifestations and other stigmata of the disease are more prominent.

Coccidioidomycosis. Coccidioidal granuloma resulting from infection with *Coccidioides immitis* appears commonly in endemic areas in California, Texas and Arizona. More recently transmission has occurred from dust in airplanes and the disease should be considered even if the patient has not been in the endemic area. Early manifestations include symptoms of bronchopneumonia with chills and fever. Residual lesions may occur in the lungs and cavitate. Probably the only positive indication for surgical intervention with excision of the

lesion is hemoptysis caused from the cavitation.

Pulmonary Actinomycosis. Actinomycosis of the lung may be confused with ordinary pyogenic infections or with bronchogenic carcinoma. When the infection does occur the tissues become edematous and fixed. Pus which is found within the lesion will often have the characteristic sulfur granules suggesting the diagnosis.

The onset of the disease may be insidious and mock lung cancer or pulmonary tuberculosis. Pain is usually absent. The positive diagnosis can only be made when the causative organism is located in the tissue or the pus.

Before chemotherapeutic agents were available, the prognosis of the patient having pulmonary actinomycosis was extremely poor. Multiple incisions and drainages of abscesses of the lung often resulted in chronic draining sinuses and eventually metastasis of the disease to the brain, liver or elsewhere, with fatal termination. Modern treatment consists of excision of the diseased lung tissue combined with drug treatment, usually consisting of sulfadiazine and penicillin.

LIPIOD GRANULOMA

Lipoid granuloma of the lung may result from inhalation of various oils, usually employed in nasal sprays or from aspiration of mineral oil.

The disease is usually bilateral and is most

scarring of tuberculosis has caused bronchiectasis the disease is uncommon in the upper lobes (Fig. 22) Bilateral bronchiectasis (Fig. 23) is present in 40 to 50 per cent of the subjects.

The type of dilatation, as visualized by radiopaque studies may be cylindrical, fusiform or saccular Symptoms are apt to be more severe with the saccular type but there is not complete correlation between x ray appearance and the severity of the disease.

Unless the patient's general condition or the extent of the disease precludes excision of the diseased lung tissue, surgical intervention should be undertaken. The choice of lobectomy segmental resection or pneumonectomy is made after visualization of the dilated bronchi with bronchograms and exact localization of the disease. A three-dimensional concept of the bronchi is essential for identification of the various lobes and segments. Both frontal and oblique projections on roentgen ray examination are necessary.

Inhalation therapy with a bronchodilator combined with one of the detergents and antibiotic therapy are useful in preoperative preparation Postural drainage combined with bronchoscopic aspiration may be necessary in advanced cases In patients with extensive disease precluding surgical treatment, these measures are helpful in controlling the infection.

The modern operations of lobectomy, pneumonectomy and segmental resection have been sufficiently perfected so that both mortality and morbidity are very low Even in instances of bilateral disease successful operations are not uncommon. Both lower lobes the right middle lobe and the lingula of the left upper lobe have been removed successfully.

The antibiotics particularly penicillin, have played an important role in reducing both mortality and morbidity Patients with severe infection can be practically freed of purulent secretions and cough before operation and protected during the postoperative period.

ECHINOCOCCUS CYST

Pulmonary echinococcus cysts or hydatid cysts, may be present in the lung as a result of invasion of the tapeworm, *Echinococcus granulosus*.

Echinococcus disease is rare in the United States but is common in other parts of the world, particularly in South America, Italy

Australia and New Zealand. Increase in world wide travel incident to two world wars has made the disease more common.

The adult stage of the disease is found in the dog (definite host) which transmits the ova in the feces The ova may then be ingested by intermediate hosts either animals or human beings. The outer coat of the ova is digested in the stomach and the embryo burrows through the mucosa, passing into the portal blood stream, and usually lodges in the liver in about 10 per cent of the subjects there is transmission to the lung.

The developing embryo expands to form a cyst with two layers The internal wall usually contains the active parasites. Daughter cysts may be formed from the germinal layer or from the developing blood capsules. In pulmonary echinococcus cysts, compression of the lung causes a layer of fibrous material to form about the cysts (Fig. 24). These lesions grow slowly but steadily and may reach enormous sizes Serious complications include erosion of the cyst into a bronchus or into the pleural cavity.

Echinococcus cysts of the lung are usually asymptomatic unless complications occur or until a cyst attains enormous size. Travel at high altitudes in an airplane may cause expansion of a cyst producing symptoms which have not been noted before.

Chronic cough, mild chest pain and dyspnea may result from pressure. Rupture of the cyst into a bronchus may be followed by hemoptysis or the sudden expectoration of large amounts of clear fluid.

Anaphylaxis may be dangerous if absorption of the hydatid antigen occurs in a patient who has already been sensitized to the development of the cyst. Anaphylactic reactions are characterized by vomiting, diarrhea, a skin rash and asthmatic like attacks. In patients having extreme cases, cyanosis, tachycardia and even delirium and coma may be noted.

Physical signs are variable and not dependable Cysts are easily visualized in roentgenograms and are spherical or ovoid, discrete and homogeneous.

The history of exposure or possible exposure, combined with roentgen ray findings may suggest the true condition and confirmation can usually be made by the high eosinophilic count and positive reactions to the Casoni intradermal test, complement fixation and precipitin test.

In the past, symptomatic echinococcus cysts of the lung were manipulated, but, since the perfection of their techniques,

States 20 per cent of cancer deaths among males between the ages of forty and seventy years. In 1935 there were 27,000 recorded deaths from bronchogenic carcinoma.

Carcinoma of the lung is primarily a disease of older age. There is little doubt that the increase in life expectancy plays a role in the increased incidence of the disease. Nevertheless, isolated cases have been reported in young adults and even in children.

Studies by the United States Public Health Service and others indicate that there is an actual increase in the frequency of bronchogenic carcinoma. Some of the increase, however, may be more apparent than real since there is greater knowledge and interest in the problem.

The increase in the frequency of pulmonary cancer appears to be uniform throughout the world. There is, however, a relationship between the prevalence of this tumor and the diagnostic facilities available. In locations where facilities for chest x-rays, bronchoscopy and thoracotomy are sparse, the number of deaths ascribed to lung cancer has remained relatively low. Almost invariably, however, when these facilities become available the incidence of bronchogenic carcinoma rises.

Etiology. Voluminous and controversial reports on the etiology of bronchogenic carcinoma featured in newspapers and magazines have frightened and bewildered the public. Moreover, there are sharp differences of opinion among experts in the medical profession concerning this question.

A considerable portion of these reports has been concerned with the relationship of cigarette smoking and lung cancer. On the basis of studies in the United States and in England, there appears to be a statistical relationship between the use of cigarettes and the frequency of bronchogenic carcinoma. In addition, the production of skin cancer in mice by tobacco tars by Graham and Wynder has added support to the theory of a cause-and-effect relationship between tobacco and cancer. Nevertheless, there is still room for doubt concerning a direct relationship between cigarette smoking and malignant disease. Other surveys have shown that a statistical affinity can be demonstrated between bronchogenic carcinoma and environmental factors such as air pollution and smoke.

It appears that the effects of radioactive ore may be a valid factor in the development of malignant lung tumors. Approximately 50 per cent of the deaths among

workers in the cobalt mines of Schneeberg and Joachimsthal were due to lung cancer.

Symptoms. The location of a malignant tumor in the bronchopulmonary tree is extremely variable and may occur in the main stem bronchus or originate peripherally in a terminal bronchiole. The presence or absence of early symptoms is usually dependent upon the location of the tumor. A centrally located tumor may cause bronchial obstruction with the accompanying symptoms. Conversely, a peripheral tumor may remain asymptomatic until an advanced stage. Symptoms associated with peripheral lesions usually result from involvement of adjacent structures and the tumor is frequently inoperable when such symptoms appear. The most common symptoms of bronchogenic carcinoma are cough, hemoptysis, chest pain, dyspnea and fever.

Cough. Cough is the most common presenting symptom of bronchogenic carcinoma. The cough is usually the result of stimulation of the cough mechanism by the tumor or the cellular debris of a tumor. The alert physician should consider the possibility of a malignant tumor in any adult patient with a persistent cough or in a patient who does not respond to the usual antitussive medications. Many persons who smoke have a dry "smokers" cough. Any change in the character of the cough bears investigation. A cough accompanied by wheezing, especially of a unilateral nature, may be indicative of partial bronchial obstruction due to neoplasm.

Hemoptysis. Hemoptysis is not a common initial symptom in patients with bronchogenic carcinoma. Ariel and co-workers report an incidence of 5.6 per cent in an analysis of 1109 cases. In far advanced cases hemoptysis is more common.

Every patient with hemoptysis should be thoroughly investigated. Necessary diagnostic studies include roentgenograms of the chest, bronchoscopy and bronchography.

Pain. Chest pain associated with bronchogenic carcinoma may be due to involvement of the parietal pleura and chest wall by tumor. In more advanced lesions intercostal nerve pain is characteristic. Some patients complain of vague discomfort or feeling of fullness in the chest on the side of the tumor. The exact etiology of this sensation is unknown, but it is not necessarily a sign of chest wall involvement or inoperability.

Dyspnea. Shortness of breath associated with a bronchogenic carcinoma is usually of grave significance. The dyspnea may be due

frequently located in the lower lobes because the oil gravitates to those organs. The right lower lobe is often more involved than the left. The lesions are firm and may be either diffuse or nodular.

Infants and older people are particularly vulnerable to the aspiration of foreign material. Accordingly lipid granulomas are more apt to be encountered in individuals of these age groups.

Many patients with lipid granulomas of the lung have no symptoms. If symptoms are present, they usually consist of cough, sometimes chest pain, and malnutrition. In rare instances hemoptysis may occur.

History of excessive use of oil in the form of nose drops, sprays or laxatives will suggest the diagnosis and sometimes sputum examinations will reveal oil droplets.

The principal importance of lipid granulomas to the surgeon is confusion with other diseases which require surgical intervention, particularly bronchogenic carcinoma in persons of the older age group. It is sometimes necessary when all other diagnostic measures have failed, to perform an exploratory thoracotomy to establish the nature of the lesion. In patients with clinical manifestation of lung destruction and distressing symptoms the involved pulmonary tissue should be excised. It is probable that in the past certain inflammatory diseases of the lung have been considered lipid granuloma, or lipid pneumonia, because of cholesterol deposits associated with chronic inflammation of the lung. These fatty like deposits are not the result of ingestion of oil materials but are part of the chronic inflammatory process associated with abscesses, bronchiectasis and other inflammatory diseases of the lung.

BOECK'S SARCOID

Boeck's sarcoid of the lung may suggest other diseases which require surgical intervention. This is particularly true of the roentgen ray appearance of the lesion. When the problem of differentiation arises, a biopsy of the lymph nodes over the anterior scalenus muscle is often useful in establishing the diagnosis. Occasionally it is necessary to perform an exploratory thoracotomy to eliminate the possibility of carcinoma or tuberculosis.

BRONCHOLITHS

Broncholiths or lung stones may result from calcification of inspissated material during chronic lung infections or more commonly from calcified lymph nodes eroding the bronchus. The common form of broncholith is usually the result of old tuberculous infections of hilar lymph nodes.

Broncholiths may cause severe hemoptysis and atelectasis if the lumen of the bronchus is obstructed. Bronchostenosis may result from the damage to the bronchial walls.

Sometimes the lung stone will be freed and expelled by coughing. If not, removal through the bronchoscope will be necessary. Irreparable lung damage distal to the obstruction or intractable hemorrhage may require excision of the involved portion of bronchus and lung distal to the lesion.

SYPHILIS

Syphilis of the lung may occur but is extremely rare. From the standpoint of surgical treatment it is of importance in the differential diagnosis.

Tumors

During the past decade few neoplasms have received as much attention in publications of the lay and medical press as have lung cancers. Concern regarding bronchogenic carcinomas is justifiable since the disease is common and appears to be increasing. The problems of diagnosis and treatment are therefore of utmost importance.

Benign lung tumors are uncommon. They are of interest, however, not only because of

their rarity but because they may simulate malignant tumors.

BRONCHOGENIC CARCINOMA

Incidence. Bronchogenic carcinoma is probably the leading cause of death from visceral cancer in males. Although the lesion is not rare in females it is six to eight times more common in males. It accounts for 10 per cent of total deaths and in the United

tasis, depending upon the degree of blockage (Fig. 27). Since the bronchi are expansile, a tumor mass may partially obstruct the bronchus during inspiration and completely obstruct it during expiration. The lung distal to the tumor may become emphysematous or even have the appearance of a large cyst or bleb. The presence of localized emphysema on a roentgenogram should not distract attention away from the underlying cause.

Complete obstruction of a bronchus resulting in atelectasis may cause sufficient symptoms to give fair warning of the presence of a tumor. This may not be true, however, when segmental or subsegmental atelectasis occurs. Atelectasis in a retrocardiac position may be difficult to detect radiologically especially on a frontal projection. Both anteroposterior and lateral full size roentgenograms must be employed to satisfactorily visualize these lesions.

The differentiation between tuberculosis and atelectasis associated with bronchogenic carcinoma may be very difficult in some instances. The absence of tubercle bacilli in the sputum or bronchial washings should focus suspicion on the possibility of neoplasm. Failure of lobar or segmental atelectasis to improve in cases of tuberculosis which have received adequate antituberculous therapy suggests the possibility that both tuberculosis and carcinoma are present. This is particularly true in older patients.

Pneumonia and lung abscess. Bronchogenic carcinoma may commonly simulate the clinical and roentgenologic picture of viral pneumonia. These patients are often subjected to a long and ill-advised course of antibiotic therapy before the true nature of their difficulty is recognized. In any case of pneumonia which does not show signs of clearing with adequate medication, bronchogenic carcinoma should be suspected. Similarly the radiologic appearance of pneumonia which is out of proportion to the clinical manifestations should be highly suggestive of malignancy especially in patients over forty years of age (Fig. 28).

Symptomatic improvement and partial resolution of the pneumonia like shadow on the x ray film may result from antibiotic therapy. This further complicates the diagnostic problem and may give the physician a false sense of security. Pessimistic suspicion of all cases of unresolved pneumonia may circumvent a diagnostic delusion.

Bronchogenic carcinomas may produce clinical and radiologic manifestations identical with lung abscesses. Obstruction of a bronchus by tumor resultant pneumonia and subsequent abscess formation of the involved lung may occur. Central necrosis of a large tumor mass may also closely simulate the radiologic picture of a lung abscess. The absence of an antecedent cause for pyogenic lung abscess or the location of an abscess in the upper or anterior portion of the lung

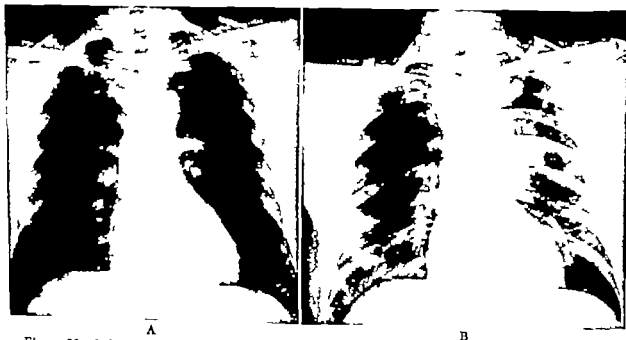


Figure 28 Hilar enlargement. A, A roentgenogram which appears to be almost normal except for a suggestion of enlargement of the left pulmonary hilum. B The roentgenographic appearance of the left hilum five weeks later. The lesion was a highly undifferentiated carcinoma.

to a spread of malignancy throughout both lung fields or may be due to an obstructing tumor of the main bronchus or the carina. Dyspnea may also be due to a pre-existing disease such as pulmonary emphysema or heart disease and merely accentuated by the presence of the tumor.

Fever. Obstruction of a segmental or lobar bronchus by carcinoma will result in atelectasis or pneumonia of that portion of the lung. The initial symptoms may be fever, cough and chest discomfort secondary to pneumonitis or atelectasis.

Physical Findings. The physical findings in patients with bronchogenic carcinoma are extremely variable. They are dependent upon the extent of the tumor, the degree of obstruction and the presence or absence of an accompanying pleural effusion. Should a lobar or segmental bronchus be partially blocked by tumor, a persistent localized wheeze is often present and may indeed be the only positive finding to localize a tumor. When complete obstruction of a bronchus occurs the physical findings are usually those of atelectasis or pneumonia of the involved segment or lobe. Tumors located in the periphery of the lung may be asymptomatic for many months. Physical examination of the chest may be completely negative until the tumor reaches a large size or until pleural effusion occurs.

Examination of patients with suspected lung tumor should always include a diligent search of the supraclavicular and axillary areas for metastatic lymph nodes.

Röntgenologic Diagnosis. The roentgenologic manifestations of a bronchogenic carcinoma usually fall into four patterns: (1) a nodular lesion of the lung, the so-called coin lesion; (2) hilar enlargement; (3) atelectasis; or (4) a shadow suggesting pneumonia or lung abscess.

Nodular lesion. Bronchogenic carcinoma may appear as a round shadow in the lung field (Fig. 25). There are often no associated symptoms and the lesion is discovered on a routine x-ray examination. The differential diagnosis of such a solitary round lesion usually lies between tuberculosis, a granuloma due to histoplasmosis or other fungus and bronchogenic carcinoma. Skin tests and sputum examination for acid-fast organisms or fungi may be helpful but often fail to establish the diagnosis. Microscopic examination of the nodule is the only totally reliable method of reaching the correct diagnosis.



Figure 25. Nodular peripheral bronchogenic carcinoma demonstrated on a tomogram of the chest. The lesion was an adenocarcinoma.

Small solitary nodules in which there are concentric rings of calcification generally may be assumed to be granulomas due to histoplasmosis or tuberculosis and removal for diagnostic or therapeutic purposes is not mandatory. Conversely nodular or coin lesions without calcification or with eccentric flecks of calcium visible by x-ray are not necessarily benign lesions. In such instances, removal of the lesion is strongly advisable.

Hilar enlargement. Radiologic evidence of enlargement of the pulmonary hilus (Fig. 26) may be caused by a tumor mass, lymph nodes or a large pulmonary artery. In this circumstance, it may be helpful to compare the x-rays with earlier chest films. Unfortunately such films are often not available. Other diagnostic procedures of value are body section x-rays (tomograms, planigrams) and in some instances angiocardiography. Exploratory thoracotomy is often necessary to establish the correct diagnosis.

Some bronchogenic neoplasms, particularly the anaplastic carcinoma, may appear radiologically to originate in the mediastinum. Not infrequently they may be mistaken for mediastinal tumors. Superior vena caval obstruction may occur when the neoplasm is in this location.

Atelectasis. Obstruction of a main lobar or segmental bronchus by a tumor mass may produce obstructive emphysema or atelec-

Anaplastic carcinoma. This group includes a number of different cellular types including the oat cell carcinoma and the more poorly differentiated forms of adenocarcinoma and epidermoid carcinoma. In general anaplastic tumors appear to grow more rapidly than the more differentiated types and metastasize earlier both by way of regional nodes and by the blood stream.

Bronchiolar carcinoma (pulmonary adenomatosis, alveolar cell carcinoma) This form of bronchogenic carcinoma is relatively uncommon. It was formerly believed that this type of neoplasm arose primarily in the alveolar epithelium. However more recent studies indicate the site of origin probably to be in the terminal bronchioles with subsequent involvement of the alveoli. Similar pathologic lesions called jagzkiekte have been observed in the lungs of sheep horses and other animals. There is some evidence that the disease in animals is caused by a virus. This tumor has a tendency to occur in multiple sites in the lungs. Such multiple foci of tumor may be due to multicentricity of origin although some observers believe that these represent secondary implants from a single primary lesion.

Emphasis has been placed on a correlation of the cellular type of bronchogenic carcinoma with the clinical course of the disease. Some pathologists, however have denied that the cell type has an important role in determining prognosis in survival. It is becoming increasingly evident that there are many interrelated and interdependent factors, rather than any single one, which are important in determining the ultimate course of the disease. Among these factors are histologic type, location and size of the primary tumor, presence or absence of vascular and lymphatic invasion and the status of the margins of resection if surgical removal is possible. In addition, there is a group of poorly defined "host factors" which undoubtedly play some part in influencing the operability, survival and cure rate for carcinoma of the lung.

Another concept which has recently interested many surgeons is the multicentric origin of pulmonary carcinoma. Numerous demonstrations of *in situ* carcinoma of the bronchus, adjacent to and away from well defined malignant lesions have strengthened the theory of multiple sites of origin. This is particularly true in bronchiolar carcinoma but may be applied to other cellular types.

Recent analyses of patients with bronchogenic carcinomas establish that more than half have inoperable lesions when first seen by the surgeon. Of the remaining patients subjected to exploratory thoracotomy only 40 to 50 per cent have lesions which can be extirpated. It becomes apparent that about three fourths of all pulmonary neoplasms are advanced beyond the stages of surgical removal when the diagnosis is made.

Diagnosis. The rapid progression of most cases of bronchogenic carcinoma requires an early and aggressive approach to establishing a diagnosis. Rarely if ever can a policy of "watchful waiting" be condoned in the management of an unexplained pulmonary shadow.

Bronchoscopy. Bronchoscopic examination of the tracheobronchial tree may yield much valuable information concerning the extent of malignancy as well as establishing a definite histologic diagnosis.

Unfortunately only about 30 per cent of pulmonary carcinomas can be seen through a bronchoscope. The remainder are located too far peripherally in the bronchopulmonary tree to be visualized or for biopsy.

Exfoliative cytology. The microscopic examination of sputum or bronchial washings may also be helpful in making a histologic diagnosis. The technique is not infallible, however and requires the services of a skilled and experienced pathologist to have practical value.

Bronchography. The instillation of a radiopaque dye into the tracheobronchial tree may be of aid in visualizing an obstructing lesion in one of the more peripheral bronchi. This technique is of no value in establishing a definite diagnosis of tumor but merely serves to localize accurately the level of obstruction.

Scalene node biopsy. Carcinoma of the lung may metastasize through carinal and peritracheal lymphatics to the anterior surface of the scalenus anterior muscle. Excision of the prescalene nodes for diagnostic purposes was first popularized by Daniel. The percentage of positive biopsies will vary greatly depending upon the selection of patients for the procedure. If weight loss, pain or other findings suggest far advanced disease, study of the prescalene nodes is indicated.

Failure to find metastatic carcinoma in the prescalene nodes obviously does not exclude the diagnosis of bronchogenic carcinoma.



Figure 27. A roentgenogram showing atelectasis of the right upper lobe caused by obstruction of the right upper lobe bronchus by epidermoid carcinoma.



Figure 28. A pneumonia-like shadow on a roentgenogram of the chest which was caused by bronchogenic carcinoma. Not only was the roentgenologic evidence suggestive of pneumonia, but the original clinical course was compatible with inflammatory disease.

should make one suspicious of neoplasm.

Pathology Bronchogenic carcinoma may originate at any level in the bronchial tree. Clinical manifestations and cell types have some, but not absolute, correlation with the location of the tumor. Well-differentiated epidermoid carcinomas are more common in the larger bronchi and produce symptoms of obstruction. Peripheral tumors are usually silent until there is metastasis or invasion of contiguous structures; they are often histologically anaplastic or adenocarcinomas.

Bronchogenic carcinoma is slightly more common in the right lung than in the left, about 55 per cent of the tumors being found in the right main stem bronchus or right lung. The upper lobes are slightly more frequently involved than the lower lobes. The middle lobe is a relatively infrequent site of bronchogenic carcinoma, only about 6 per cent of all primary tumors being located in this area. Tumors arising peripherally are somewhat less common than those arising in the main or lobar bronchus levels. In many instances of more advanced tumors it is impossible to state with any certainty the exact anatomic site of origin.

Numerous classifications of bronchogenic carcinoma have been based on histologic appearance. The most practical, however, is that which divides the tumors into four major histologic categories. These categories and their relative frequency are as follows:

Epidermoid	50 per cent
Anaplastic carcinoma	32 per cent
Adenocarcinoma	15 per cent
Bronchiolar carcinoma	3 per cent

There is some variation in the relative incidence of the above histologic categories as a result of minor differences in classification by individual pathologists.

Epidermoid (squamous) carcinoma. This is the most common type of bronchogenic carcinoma. The tumor is seen predominantly in males, in a ratio of eight to one. Although it is generally stated that squamous carcinoma is relatively slow growing, there are a great number of individual factors which influence the rate of growth and the course of the disease. Metastases of epidermoid carcinomas of the lung are usually to regional lymph nodes in the hilum and peritracheal region. Direct invasion of the blood vessels, however, may occur.

Adenocarcinoma. This form of neoplasm is more common in females, comprising about half the cases of bronchogenic carcinoma seen in women. There is no statistical relationship to smoking habits. Adenocarcinoma of the lung has a tendency to occur originally as a peripheral tumor more commonly than epidermoid carcinoma. Early invasion of the blood stream with dissemination is more characteristic of this form of cancer than with the squamous carcinoma.

reveals the carcinoma has extended past the carina to the main stem bronchus of the opposite side or involves the trachea, the lesion is inoperable.

Chest wall involvement. Gross extension of the tumor into the chest wall generally precludes satisfactory surgical resection. Under extremely favorable circumstances of localized chest wall involvement, bloc removal of the lung and involved area of the chest wall may be undertaken. The ultimate prognosis in such cases is poor.

Treatment. Although a few isolated cases of spontaneous regression of pulmonary carcinoma have been reported, surgical extirpation of the tumor offers the best hope of cure at the present time. Roentgen therapy and chemotherapeutic agents, such as nitrogen mustard, offer some possibility of palliation in patients who are past the stage of operability.

Pneumonectomy and lobectomy. Total pneumonectomy with removal of the hilar lymph nodes is the surgical procedure most frequently employed for bronchogenic carcinoma. Some surgeons advocate the so-called radical pneumonectomy which includes not only removal of the lung and hilar lymphatics but also the lymphatic chain along the trachea and the pericardium on the involved side. The operative mortality from pneumonectomy including the first thirty days after operation, is generally 10 per cent or less. The mortality associated with resection of a tumor when the lesion has produced invasion of the surrounding structures is considerably higher.

Under the following circumstances lobectomy is considered preferable to pneumonectomy in the treatment of bronchogenic carcinoma. (1) if the primary lesion is small and peripheral and is located well away from the interlobar fissure or adjacent lobes, (2) if there is no evidence of extension to the mediastinal lymphatics and (3) if the pulmonary reserve of the patient is such that total pneumonectomy would not be tolerated or would render him a respiratory cripple. Under these circumstances, the results from lobectomy are approximately equal to those following pneumonectomy.

Results. Analysis of large numbers of survivors after operations for bronchogenic carcinoma indicates that in patients with a localized tumor and without evidence of vascular or lymphatic invasion, 35 per cent or more may survive five years. If, however, vascular or lymphatic invasion was demon-

strated or the tumor had extended outside the lung, five year survival statistics are generally 5 per cent or less.

The over-all five year survival of all patients with bronchogenic carcinoma is less than 10 per cent. This includes those patients who were inoperable when first seen or who were found to be inoperable at the time of exploration.

BRONCHIAL ADENOMA

Adenoma of the bronchus is a slow growing sessile, usually circumscribed tumor growing in the submucosal tissue of the proximal bronchi. Although its clinical course and histologic characteristics are different from those of bronchogenic carcinoma, it must be considered a potentially malignant tumor. Numerous examples of metastases from bronchial adenoma have been reported.

Bronchial adenomas are generally firm, smooth, rounded, or wormlike projections lying beneath the mucous membrane of the main stem or lobar bronchi. The tumors may be divided into two main types histologically: carcinoid and cylindroid.

The carcinoid type comprises approximately 85 per cent of all bronchial adenomas. Grossly on cut sections, the tumor is pinkish or tan. There is usually a capsule of connective tissue around the tumor but in long-standing lesions, invasion of the capsule or the bronchus may occur. Histologically the carcinoid is composed of uniform, small, acidophilic cells with finely stippled nuclei. The cells resemble somewhat the carcinoid tumors of the gastrointestinal tract. Metastases from such tumors have been reported in the liver, kidneys and vertebral column.

The cylindromatous type of bronchial adenoma is less common than the carcinoid. Histologically the cells of the cylindromatous type are less uniform, smaller and with more basophilic cytoplasm. This tumor shows a more marked tendency to invade the capsule than does the carcinoid variety. Metastases are more frequent in this type of bronchial adenoma.

Clinically bronchial adenoma produces symptoms of bleeding or of obstruction of the bronchi. In some instances bleeding may be exceedingly severe because of the extreme vascularity of the tumor. Tissue for diagnosis is often obtainable through the bronchoscope because of the central location of the tumor.

Treatment of bronchial adenoma must be

noma. A positive scalene biopsy however establishes that the tumor has passed the stage of surgical curability.

The lymphatic drainage of the lung is such that the entire right lung drains to the right scalene fat pad via the mediastinal and peritracheal nodes. The upper lobe of the left lung drains to the left scalene nodes. The left lower lobe however may drain by way of the carinal nodes to either the right or left scalene areas.

Exploratory thoracotomy. Although the previously mentioned diagnostic studies may be of value, in many cases exploratory thoracotomy may be necessary to establish a definite diagnosis. The risk of exploratory thoracotomy at present is probably no greater than that of exploratory laparotomy. The inconveniences and discomforts of the procedure are of trivial importance when compared to the urgency of diagnosis and possible successful removal of a lung cancer.

Signs of Inoperability. Certain findings are generally considered evidence that a bronchogenic carcinoma has passed the stage of possible cure. In most instances, once the histologic diagnosis has been established and one or more of the signs of inoperability are present, surgical resection is not considered feasible. Under certain circumstances, however, although surgical cure cannot be anticipated, a palliative removal of the primary tumor may be performed to afford symptomatic relief of the complications of the primary tumor such as bleeding, infection or severe cough.

Regional node involvement. Bronchogenic carcinoma metastasizes by way of the lymphatics to the mediastinal lymph nodes and thence to the supraclavicular lymph nodes. The nodes of the axillary region are invaded less frequently and usually later in the disease. Involvement of the prescalene nodes is indicative of metastases which have extended beyond the boundaries of surgical resection.

Nerve paralysis. Extension of a bronchogenic carcinoma into the mediastinum may cause paralysis of the recurrent laryngeal nerve on either side. Because of its anatomic position, the left recurrent laryngeal nerve is more vulnerable than the right. Paralysis of the recurrent laryngeal nerve causes hoarseness due to immobility of the ipsilateral vocal cord. Under such circumstances surgical extirpation of the entire tumor mass is not feasible.

Extension of a bronchogenic carcinoma to involve the phrenic nerve causes paralysis of

the corresponding leaf of the diaphragm. This is generally considered a sign of inoperability although under extraordinary circumstances it may be feasible to resect the pericardium and the involved nerve.

Pleural effusion. A pleural effusion associated with bronchogenic carcinoma may rarely be due to irritation of the pleura as a result of a peripheral tumor but is usually the result of tumor implants on the visceral or parietal pleura. In the latter case the effusion is frequently bloody. The presence of a serosanguineous effusion and demonstration of malignant cells in the effusion generally indicate that the tumor has passed the stage of resectability. In some instances, however, a small area of the chest wall or parietal pleura may be involved and resection of the lung and a portion of the pleura or chest wall may be technically feasible.

Pericardial effusion. Involvement of the pericardium or myocardium by direct extension of the tumor mass or by metastatic tumor may cause a bloody pericardial effusion, arrhythmias or even manifestations of cardiac tamponade. Under such circumstances, if tumor cells are demonstrated in the pericardial fluid, surgical exploration is not warranted.

Distant metastases. Primary bronchogenic carcinomas commonly metastasize to the adrenal glands, the brain, liver and kidneys. Brain metastases may be suspected when there is mental confusion or other neurologic signs. The electroencephalogram may be helpful in detecting the presence of metastatic lesions in the brain. Metastases to the adrenal gland may be difficult to detect during life. Signs of adrenal insufficiency may appear in the late phases of the disease.

The skull, spine and pelvis are frequent sites of metastases from carcinoma of the lung. Preoperative evaluation of patients with bronchogenic carcinoma should include radiographic studies of these sites in order to rule out bony metastases.

Pancoast syndrome (superior sulcus syndrome) The syndrome first described by Pancoast is usually caused by a carcinoma situated in an apical position of the lung with involvement of the last cervical and first thoracic autonomic ganglia, producing Horner's syndrome. In addition, there is frequent involvement of the brachial plexus, causing pain in the arm and partial paralysis of the ulnar and radial nerves.

Gross extension of tumor to the contralateral side. If bronchoscopic examination



Figure 30. Metastatic melanoma. A, Large circumscribed metastasis from a malignant melanoma is shown on the roentgenogram. B, The appearance of the metastatic melanoma on the lateral projection.

in these cases is poor and the patient's course is usually rapidly downhill.

BENIGN TUMORS OF THE LUNG

Hamartoma. Hamartomas are benign tumors of the lung usually located in the periphery. Usually the tumors are small, contain cartilage with variable amounts of connective tissue and are encapsulated. Occasionally, calcium may be present. The presence of calcium in the tumor may cause it to be confused radiologically with a tuberculoma. It is often impossible to differentiate a hamartoma from bronchogenic carcinoma. Thoracotomy and excision of the tumor are usually necessary to establish the diagnosis.

Other Benign Tumors. Other benign tumors of the bronchus or lung have been reported but are extremely rare. These include fibromas, lipomas and inflammatory polyps. The principal clinical manifestations are due to obstruction of a bronchus or to bleeding. They may simulate bronchogenic carcinoma, excision of the tumor and involved lung is the proper treatment.

READING REFERENCES

- Adams, W. E., and others: The Significance of Pulmonary Hypertension as a Cause of Death Following Pulmonary Resections. *J Thoracic Surg.*, 26: 437, 1953.
- Allsritten, F. F., Jr., and Teasletou, J. Y., III: Treatment of Giant Cysts of the Lung. *J Thoracic Surg.* 20:740, 1950.
- Allison, P. R.: Giant Bullous Cysts of the Lung. *Thorax* 2:169, 1947.
- Anderson, N. L.: The Rationale of Therapeutic Pneumoperitoneum, the Physiological and Mechanical Considerations. *Dis. Chest* 114:732, 1948.
- Auerbach, O.: Pathology of Tuberculosis as Affected by Antibiotics. *Am. J. Surg.* 89:677, 1955.
- Auerbach, O., and Green, H.: The Pathology of Clinically Healed Tuberculosis Cavities. *Am. Rev. Tuberc.* 52:707, 1949.
- Ballou, D. H., Singer, J. J., and Graham, E. A.: Bronchiectasis. *J Thoracic Surg.*, 1:154, 1931.
- Blades, B.: Surgical Management of Tumors of the Lung Discovered in X-ray Surveys. *J.A.M.A.* 154: 190, 1954.
- Blades, B., and Dugan, D. J.: Pseudobronchiectasis. *J Thoracic Surg.* 13:40, 1944.
- Blades, B., and Kent, E. M.: Individual Ligation Technique for Lower Lobe Lobectomy. *J Thoracic Surg.* 10:84, 1940.
- Bradshaw, H. H., Putney, F. J., and Clef, L. H.: The Fate of Patients with Untreated Bronchiectasis. *J.A.M.A.* 116:2581, 1941.
- Brewer, L. A., and Doley, F. S.: The Surgical Management of Chronic Spontaneous Pneumothorax. *J Internat. Coll. Surgeons* 8:42, 1945.
- Brook, R. C., Cann, R. J., and Dickinson, J. R.: Tuberculous Mediastinal Lymphadenitis in Childhood, Secondary Effects on Lungs. *Guy's Hospital Rep.* 87:295, 1937.
- Burford, T. H., Center, S., Ferguson, T. F., and Spjut, H.: Results in the Treatment of Bronchogenic Carcinoma. *J Thoracic Surg.* 36:318, 1958.
- Chamberlain, J. M., and Klopstock, R.: Further Experiences with Segmental Resection in Pulmonary Tuberculosis. *J Thoracic Surg.* 20:845, 1950.
- Chamberlain, J. M., and Ryan, C. H.: Segmental Resection in Pulmonary Diseases. *J Thoracic Surg.* 18:190, 1950.

instituted with the consideration that this is a malignant or potentially malignant lesion. Consequently excisional biopsy through a bronchoscope or fulguration is not adequate. Surgical resection of the involved bronchus and the distal portion of the lung is the only acceptable mode of therapy. Lobectomy is usually necessary with removal of the involved bronchus and the accompanying pulmonary lobe. Occasionally pneumonectomy may be indicated. Sleeve resection and reanastomosis of the bronchus for lesions located in a main bronchus have been reported.

SARCOMA OF THE LUNG

Primary sarcomas of the lung are rare. Histologically the tumor may be a fibrosarcoma or a leiomyosarcoma. The site of origin may be central or peripheral. In the case of the central type the histologic picture may somewhat resemble anaplastic carcinoma.

Generally sarcomas occur in younger age groups than do carcinomas of the lung. Severe bleeding may occur in those tumors arising in the main bronchus. Symptoms are usually absent in the peripheral type until the tumor has metastasized or invaded contiguous structures.

The treatment of sarcoma of the lung is pneumonectomy or lobectomy depending upon its extent. If the lesion is localized, the prognosis is somewhat better than that of malignant epithelial neoplasms.

MALIGNANT TUMORS OF RETICULOENDOTHELIAL ORIGIN

Primary tumors of the lung which arise from the lymphatic or reticuloendothelial system are extremely unusual. Lymphosarcoma, or Hodgkin's disease, may occur in the lungs without other systemic manifestations. The roentgenographic appearance of these tumors is not generally diagnostic and may be confused with pneumonia or bronchogenic carcinoma. Many of these patients are operated upon with a presumptive diagnosis of carcinoma of the lung (Fig. 29).

Surgical resection of the involved lobe or lung appears to offer a fair prognosis. The use of roentgen therapy or chemotherapeutic agents as an adjunct to surgical removal has been advocated and appears to be indicated.

METASTATIC TUMORS

The lungs act as a filtering system for the venous drainage of the body. As a consequence, it is not surprising that the pul-



Figure 29 Roentgenographic appearance of lymphosarcoma of the lung. An identical x-ray shadow could be produced by bronchogenic carcinoma.

monary bed is a frequent site of metastatic implants. Metastases from primary tumors of the gastrointestinal tract, the breast, the prostate, the kidney and the adrenals are common. In addition, metastases from peripheral tumors such as melanomas or sarcomas of bone often appear in the lungs (Fig. 30).

Commonly such blood borne metastases appear as single or multiple rounded peripheral densities seen in chest roentgenograms. The presence of multiple densities appearing in an x-ray should immediately bring up the possibility that these are metastatic lesions rather than primary bronchogenic carcinoma. Pulmonary metastases may appear years after extirpation of the primary lesion.

The presence of multiple nodules in the lung should stimulate a search for a primary lesion as well as investigation of other organs for possible sites of metastasis. If a solitary metastatic lesion is present in the lung or metastases are localized to one lobe, removal of the involved lung should be considered. Numerous cases of survival for years after resection of such localized metastases have been recorded.

Lymphogenous spread of carcinoma may occur in the lungs either from primary bronchogenic carcinoma or from distant sites such as breast, prostate or stomach. Under these circumstances there is usually a marked increase in the perivascular markings of the lung which represents involvement of lymphatic channels. The prognosis

- tion for Chronic Abscess of the Lung. *J. Thoracic Surg.* 17:514 1948.
- Smith, R. R., Knudsen, A. P., and Watson, W. L.: Terminal Bronchiolar or "Alveolar Cell" Cancer of the Lung: A Report of Twenty Cases. *Cancer* 2: 873, 1949.
- Sussman, M. L.: Non-putrid Pulmonary Suppuration, *Am. J. Roentgenol.*, 40:22, 1938.
- Sussman, M. L.: The Roentgen Aspects of Nonputrid Pulmonary Suppuration. *Am. J. Roentgenol.* 44: 345 1940.
- Sweet, R. H.: An Analysis of the Massachusetts General Hospital Cases of Lung Abscess from 1938 through 1942. *Surg. Gynec. & Obst.* 80:568, 1945.
- Symposium on End Results in Treatment of Cancer: Proceedings of Third National Cancer Conference. Philadelphia, J. B. Lippincott Company 1937 pp. 833-953.
- Thomas, C. P.: Benign Tumors of the Lung. *Lancet* I 1, 1954.
- Thompson, L. W., and Janes, R. M.: Collapse Therapy in Pulmonary Tuberculosis. *Am. Rev. Tuberc.* 45:357 1942.
- Touff, A. S. W., Nabatoff R. A., and Neuhof H.: Acute Putrid Abscess of the Lung. VI. The Late Results of Surgical Treatment. *J. Thoracic Surg.* 20:266, 1950.
- Transactions of the Thirteenth Conference on the Chemotherapy of Tuberculosis. Veterans Administration, Washington, D. C., Feb. 1954.
- Umiker, W.: Cytology in Bronchogenic Carcinoma. *Am. J. Clin. Path.* 22:558 1952.
- Walter J. B., and Pryce, D. M.: The Site of Origin of Lung Cancer and Its Relation to Histologic Type. *Thorax* 10:117 1955.
- Warring, F. C., Jr., and Lindskog, G. E.: Surgical Management of Giant Air Cysts of the Lung: Physiologic Improvement after Resection. *Am. Rev. Tuberc.* 63:579 1951.
- Watson, W. L.: Radical Surgery for Lung Cancer. *S. Clin. North America*, 33:401 1953.
- Watts, C. F., Clagett, O. T., and McDonald, J. R.: Lipoma of the Bronchus: Discussion of Benign Neoplasms and Report of Case of Endobronchial Lipoma. *J. Thoracic Surg.* 15:132, 1946.
- Weissler H. and Rabin, C. B.: Benign Tumors of the Bronchus. *Am. J. M. Sc.* 183:164 1932.
- Womack, N.A., and Graham, E. A.: Mixed Tumors of the Lung, So-Called Bronchial or Pulmonary Adenoma. *Arch. Path.* 26:165 1938.
- Wynder E. L., and Graham, E. A.: Tobacco Smoking as a Possible Etiologic Factor in Bronchogenic Carcinoma: A Study of 684 Proved Cases. *J.A.M.A.* 143:329 1950.

- Churchill, E. D.: Lobectomy and Pneumonectomy in Bronchiectasis and Cystic Disease. *J Thoracic Surg.* 6:286 1937
- Churchill, E. D.: Extrapleural Pneumothorax. *Am. Rev. Tuberc.* 41:423 1940.
- Churchill, E. D., Sweet, R. H., Soutter L., and Scannell, J. G.: The Surgical Management of Carcinoma of the Lung. *J Thoracic Surg.* 20:949 1950.
- DeBailey, M. E., Ochsmen, A., and DeCamp, P. T.: Primary Carcinoma of the Lung. *Surgery* 32:577 1952.
- Effler, D. B., Blakes, B. and Marks, E.: The Problem of the Solitary Lung Tumor. *Surgery* 24:917 1948.
- Gibson, J. H., Jr., Allbritten, F. F., Templeton, J. Y., and Newlon, T. F.: Cancer of the Lung. An Analysis of 532 Consecutive Cases. *Ann. Surg.* 138:489 1953.
- Gibson, J. H., Jr., Stokes, T. L., and McKeown, J. J., Jr.: The Surgical Treatment of Carcinoma of the Lung. *Am. J. Surg.* 89:484, 1955
- Graham, E. A., and Singer, J. J.: Successful Removal of an Entire Lung for Carcinoma of the Bronchus. *J.A.M.A.* 101:137 1933.
- Haight, C.: Total Removal of Left Lung for Bronchiectasis. *Surg. Gynec. & Obst.* 58:768, 1934.
- Hood, R. T., Good, C. A., Clagett, O. T., and McDonald, J. R.: Solitary Circumscribed Lesions of the Lung. *J.A.M.A.* 152:1185 1953.
- Jenisk, R. J. and VanHise, W.: The Surgical Treatment of Metastatic Pulmonary Lesions. *Surgery* 43:100, 1958.
- Johnson, J., Kirby, C. K., and Blakemore, W. S.: Should We Insist on Radical Pneumonectomy as a Routine Procedure in the Treatment of Carcinoma of the Lung? *J Thoracic Surg.* 36:900 1958
- Jones, J. C., Robinson, J. L., and Meyer, R. W.: Primary Bronchogenic Carcinoma of the Lung. Statistical Study of 704 Private Patients. *Arch. Surg.* 70:288 1955.
- Karnofsky, D. A., Myers, W. P. L., and Phillips, R.: Treatment of Inoperable Pulmonary Cancer: Primary and Metastatic. *Am. J. Surg.* 89:529 1955
- Kent, E. M., and Ashburn, P. S.: Pulmonary Reaction for Chronic Lung Abscesses. *J Thoracic Surg.* 17:523, 1948.
- Kernher, R. D. and Adams, W. E.: Chronic Non-specific Suppurative Pneumonitis. *J Thoracic Surg.* 17:495, 1948.
- Kirney, W. M.: Bronchiectasis: A Neglected Disease. *Dis. Chest* 13:93, 1947
- Kirklin, J. W., and others: Bronchogenic Carcinoma: Cell Type and Other Factors Relating to Prognosis. *Surg. Gynec. & Obst.* 100:429, 1955
- Leach, J. E.: Pneumothorax in Young Males. *Arch. Int. Med.* 76:204, 1945.
- Liebow, A. A., Hales, M. R., and Lindskog, G. E.: Enlargement of the Bronchial Arteries and Their Anastomoses with the Pulmonary Arteries in Bronchiectasis. *Am. J. Path.* 25:311, 1949
- Lindskog, G. E.: Surgical Treatment of Chronic Pulmonary Abscess. *Surgery* 15:783 1944.
- Lindskog, G. E., and Liebow, A. A.: Thoracic Surgery and Related Pathology. New York, Appleton-Century-Crofts, 1953.
- Lindskog, G. E., Liebow, A. A., and Hales, M. R.: Bulobectomy—Surgical and Anatomical Considerations in Resection of Right Middle and Lower Lobes through the Intermediate Bronchus. *J. Thoracic Surg.* 18:618, 1949.
- Mayer, E., and Maier, H. C.: Pulmonary Carcinoma, Pathogenesis, Diagnosis and Treatment. Philadelphia, J. B. Lippincott Company 1958.
- Moore, R. H.: Palliative Resection for Cancer of the Lung. Proceedings of the Second National Cancer Conference. New York, American Cancer Society 1952, vol. 2, part 4, 935-937
- Modler, E. M., Bornstein, S., and Steward, D. M.: A Bacteriologic Study of Resected Tuberculous Lesions. *Am. Rev. Tuberc.* 66:36, 1952.
- Meyer, A., Mosson, O., Brunel, M., and Pesle, G.: Segmental Resection of Lung. *Bull. et mém. Soc. méd. hôp. Paris* 67:262, 1951
- Moersch, H. J. and Clagett, O. T.: Pulmonary Cyst. *J Thoracic Surg.* 16:179 1947
- Moyer, R. E.: Pneumoperitoneum and Pleurodesis in the Treatment of Pulmonary Tuberculosis. *Dis. Chest* 15:43, 1949
- Neuhof, H.: Acute Putrid Abscess of the Lung. *Surg. Gynec. & Obst.* 80:351 1945.
- Neuhof, H., and Touroff, A. S. W.: Acute Putrid Abscess of the Lung. *J Thoracic Surg.* 9:439 1939
- Neuhof, H., Touroff, A. S. W., and Aufses, A. H.: The Surgical Treatment, by Drainage, of Subacute and Chronic Putrid Abscess of the Lung. *Ann. Surg.* 113:209 1941.
- O'Brien, E. J., and others: The Present Chaos Regarding Resection of Residual Caseous Lesions in Pulmonary Tuberculosis. *J Thoracic Surg.* 26:441, 1953
- Ochsmen, A., DeCamp, P. T., DeBailey, M. E., and Ray, C. J.: Bronchogenic Carcinoma. *J.A.M.A.* 143: 981, 1952.
- Overholt, R. H., and Bongas, J. A.: Common Factors in Lung Cancer Survivors. *J Thoracic Surg.* 32:508 1956.
- Overholt, R. H. and Langer, L. A.: A New Technique for Pulmonary Segmental Resection. Its Application in the Treatment of Bronchiectasis. *Surg. Gynec. & Obst.* 84:257 1947
- Overholt, R. H. and Schmidt, I. C.: Silent Phase of Cancer of the Lung. *J.A.M.A.* 141:817 1949
- Overholt, R. H., Woods, F. M. and Wilson, N. J.: Segmental Resection in Tuberculosis. *Dis. Chest* 23:255 1953.
- Pancost, H. K.: Superior Pulmonary Sulcus Tumor. *J.A.M.A.* 90:1391, 1932.
- Progress Report on Therapeutic and Toxic Effects of Combinations of Isoniazid, Streptomycin, and Para-aminosalicylic Acid. United States Public Health Service Cooperative Investigation of Antimicrobial Therapy of Tuberculosis. *Am. Rev. Tuberc.* 69:1, 1954.
- Robinson, S.: The Surgery of Bronchiectasis. Including a Report of Five Complete Resections of the Lower Lobe of the Lung with One Death. *Surg. Gynec. & Obst.* 24:194, 1917
- Ryan, B. J., Medlar, E. M., and Welles, E. S.: Simple Excision in the Treatment of Pulmonary Tuberculosis. *J Thoracic Surg.* 23:327 1952.
- Scott, S. M.: A Critical Review of One Hundred and Sixty Consecutive Scalenic Node Biopsies. *Am. Rev. Tuberc.* 70:1002, 1957
- Seiler, H. H., Clagett, O. T., and McDonald, J. R.: Pulmonary Resection for Metastatic Malignant Lesions. *J Thoracic Surg.* 19:855 1950.
- Shaw, R. R., and Paulson, D. L.: Pulmonary Resec-

heart disease there coexists with the primary mechanical lesion an inability of the myocardium to maintain a normal propulsive force, and in the evaluation of all patients for cardiac surgery this must be constantly borne in mind.

EXTRACORPOREAL CIRCULATION

In the present era of cardiovascular surgery an understanding of the principles of cardiopulmonary by pass and hypothermia is a basic requirement to a discussion of the treatment of such lesions. While these methods are not used in the correction of all major cardiac defects, their fields of application are rapidly widening. Cardiopulmonary by pass and hypothermia may be used alone or in combination.

The extracorporeal heart lung apparatus has as its prime surgical objective the deviation of the blood from the chambers of the heart or involved great vessels to permit the repair of abnormalities under direct vision in a bloodless field while at the same time maintaining the normal state of the vital functions of the body as a whole. The goal of a relatively bloodless heart for operation is most adequately achieved if the apparatus employed can satisfactorily substitute for the pumping and oxygenating functions of the heart and lungs for prolonged periods. The artificial heart lung when used for this purpose must remove all of the returning blood from the superior and inferior cavae, oxygenate it and pump it

into the systemic arterial circulation at exactly the same rate at which it returns through the cavae. The period of by pass is usually limited to a period of one to one and one half hours. For most procedures periods of ten to thirty minutes are sufficient. Patients can be maintained satisfactorily with relatively low rates of blood flow at normal temperatures or under conditions of reduced body temperature. Low flow rates are between 35 and 50 ml. per kg. per minute. High flow rates are those between 75 and 120 ml. per kg. per minute. With higher flow rates it would appear that metabolic disturbance resulting from the procedure is less prominent and that these rates are therefore preferable. The optimal flow rate can also be determined by body surface measurement, 2300 cc. per square meter of body surface is an adequate flow rate. If the body temperature is reduced to the range of 30° C. by means of blood temperature control apparatus within the pump oxygenator system, lower rates of flow are desirable than at normal body temperatures.

Figure 1 illustrates diagrammatically the artificial heart lung circuit. Blood removed from the cavae can flow by gravity into the oxygenator which is located 18 to 24 inches below the level of the atrium of the patient. The blood then flows through the oxygenator into the pumping mechanism. The pump returns the blood into a large artery usually the femoral. Incorporated in the apparatus are monitoring instruments which measure

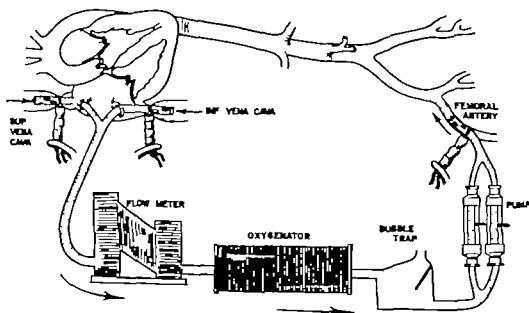


Figure 1 Diagrammatic sketch of pump oxygenator. Blood is withdrawn from cavae through flow meter and passes to oxygenator. Pneumatic pumps then propel blood to patient through a catheter in the femoral artery.

THE HEART AND PERICARDIUM

By CHARLES A. HUFNAGEL, M.D.

CHARLES ANTHONY HUFNAGEL is the son of a Kentucky physician. Educated at the University of Notre Dame and Harvard Medical School, he was trained in surgery at the Peter Bent Brigham Hospital and in the Laboratory of Surgical Research at Harvard. His investigative activities have continued throughout his career and now at Georgetown University's medical school, he has made important contributions to the field of cardiac surgery.

The application of knowledge and techniques acquired through surgical research has played the major role in the present development of the definitive treatment of cardiovascular disease. To recognize the part which surgery properly should play in the correction of organic cardiac lesions some of the fundamental concepts of the heart as a pumping mechanism should be emphasized.

The heart has as its prime objective the unidirectional propulsion of an adequate volume of blood through two systems—the pulmonary, or lesser circuit, and the systemic, or major circuit. The flow through these two systems must be exactly balanced since any inequality must result in fluid disequilibrium or increased cardiac work.

Thus, the heart consists of two synchronous pumping systems, each of which has a collecting chamber an atrium, and a pumping chamber a ventricle. As in all systems for the unidirectional pumping of fluid, there are two major elements—the propulsive force, which in the case of the heart is supplied by the ventricular musculature, and a valvular system, which pro-

tections the inlet and outlet of each of the pumping chambers to maintain the flow of fluid in a single direction.

Organic heart disease is the result of an abnormality of one or more of these major factors—the pumping force, the valvular system, shunts or obstruction to blood flow.

In congenital heart disease, the basic lesions can be divided into those in which there is an obstruction to forward flow those in which there is an abnormal shunting of blood and those having a combination of obstruction with abnormal shunting from one chamber to another or from one circulation to another.

In acquired heart disease the lesions can be classified as valvular obstruction, valvular regurgitation, a combination of these, impairment of the blood supply to the myocardium, metabolic derangement, infection or neoplasm.

If this simplified mechanistic concept of organic heart disease is kept in mind, it removes some of the confusion which is so often associated with the classification of cardiac lesions. It must always be remembered, however that with certain forms of

pass are rare. Those complications which do arise tend to be secondary to the disease process for which cardiopulmonary by pass was undertaken or to a complication of the operative procedure itself rather than to the cardiopulmonary by pass per se.

Partial cardiopulmonary by pass is employed to assist the failing heart in acute medical conditions. The procedure is simplified by using a large peripheral vein as a source of venous blood and returning the blood through a peripheral artery. Such a modified procedure can be accomplished under local anesthesia and may be helpful in support of an inadequate circulation.

Local perfusion of drugs to an isolated part is also possible using a similar method. High concentrations of the agent can be delivered without the systemic effect, if all returning blood is recirculated through the apparatus. This has its greatest application in the treatment of malignant tumors.

Under certain conditions while using the cardiopulmonary by pass it is desirable to induce cardiac arrest. Cardioplegic agents which have been commonly employed are potassium citrate and acetylcholine. These drugs are introduced by injection into the aortic root after the aorta has been clamped above the site of the injection so that the full dosage is delivered into the coronary arteries. The aortic clamp is left in place during the period of the desired arrest. To restart the heart, the aortic clamp is removed and the coronary arteries perfused by the extracorporeal pumping mechanism. This results in resumption of a normal cardiac beat, usually in one to two minutes. Recovery from acetylcholine arrest sometimes occurs even prior to the reintroduction of blood into the coronary system. Anoxic arrest has also been widely employed. When the aorta is clamped the heart gradually assumes a slower rate and then ceases beating. Resuscitation of the heart is accomplished by release of the aortic clamp and restoration of the coronary circulation.

Prolonged periods of cardiac arrest with ventriculotomy appear to be associated with a varying degree of decrease in the myocardial function in the immediate postoperative period. The amount of impairment appears related to the duration of the arrest at normal temperature.

Cardiac arrest can also be accomplished by the use of cold alone. The aorta is clamped just below the innominate artery and Ringer's lactate solution at 4° C. is perfused into the coronary arteries under

systemic pressure. The surface of the heart is simultaneously cooled with Ringer's lactate at the same temperature. This produces cardiac arrest within one to two minutes. Arrest occurs when the temperature of the heart muscle reaches approximately 18° C., but electrical activity continues until below 10° C. The rewarming of the heart can be accomplished by removal of the aortic clamp and the perfusion of the heart with warm blood. Ventricular fibrillation rarely occurs during cardiopulmonary by pass unless cardiac arrest has been used but it is not rare following periods of cardiac arrest for more than ten minutes duration. Fibrillation under these circumstances is readily reversible in almost all instances providing one can maintain an adequate coronary circulation and elevate the cardiac temperature to a satisfactory level.

HYPOTHERMIA

The use of hypothermia paved the way for many important developments in cardiovascular surgery. Although for intracardiac operations it has been largely superseded by the use of the artificial heart lung, it is still an important adjunct to the treatment of certain cardiac problems where it is used alone or with cardiopulmonary by pass. In addition, it is a useful tool in the surgical treatment of other conditions than those of the cardiovascular system.

As early as 1944, Gross and Hufnagel showed the efficiency of body cooling to prevent spinal cord damage during periods of prolonged thoracic aortic occlusion. This work focused attention upon the principle that moderate reduction in body temperature markedly reduces the metabolic need of tissue and that this reduced oxygen need markedly prolongs the period for which the blood supply to an organ may be occluded without permanent damage. The susceptibility to damage at a specific temperature varies from one organ to another, the brain, in general, being the most sensitive. Lowering of the body temperature to levels below 30° C. is associated with increased cardiac irritability which rapidly increases with temperatures below this level. At temperatures of 28° C. and lower the incidence of ventricular fibrillation is quite high, particularly if any manipulation of the heart is carried out in this temperature range. Cessation of an effective cardiac beat usually occurs at 20 to 22° C. Cessation of all electrical cardiac activity however does not necessarily occur until the region of 0 to 10° C. has been

the oxygen tension, carbon dioxide tension, temperature, pH, venous and arterial pressure and the volume of flow

In all pumping oxygenating devices an attempt is made to minimize the turbulence of blood flow clotting and hemolysis. The surfaces with which the blood comes in contact must be free of substances which would have an undesirable physical or chemical effect upon either the formed or liquid elements of the blood and must not liberate toxic or pyrogenic materials. Certain plastics such as polyethylene, methyl methacrylate, polyvinyls or stainless steel with silicone coatings have been found most suitable for blood contact surfaces. These materials also inhibit the coagulation of blood during the period when it is outside the vascular tree.

Three types of pumping mechanism have been widely employed. These are (1) finger pumps, (2) roller pumps and (3) ventricle pumps. The latter is a chamber with a valve at its inlet and outlet which is emptied by external compression, either mechanical, pneumatic or hydraulic. All of these pumping devices when properly designed and employed can give highly satisfactory results.

A simple ventricle type pump using compressed gas as the propelling force is shown in Figure 1. Hemolysis with this type of pump is extremely low.

Oxygenating systems have depended upon the exposure of a large surface of blood to the oxygenating medium. The types usually employed have been (1) bubble type oxygenators, (2) stationary screen oxygenators, (3) rotating disk oxygenators and (4) membrane oxygenators.

Bubble oxygenators introduce small bubbles into a flowing vertical column of blood and depend upon the large surface areas of the blood gas interface to provide adequate oxygenation. Removal of the bubbles is accomplished by exposure of the oxygenated blood to a large surface coated with a defoaming agent. From the defoaming chamber a large helical tube leads the blood downward to a reservoir. During passage of the blood down the helix any small bubbles remaining rise to the surface and break. Bubble oxygenators tend to be relatively limited in oxygenating capacity in relation to their volume and two or more units may be used for patients of large size.

Stationary screen type oxygenators expose large areas of blood to a high oxygen atmosphere by the filming of blood over multiple screens in its passage through the oxy-

genator. Blood is introduced through a manifold at the top so that it will flow uniformly over each of the screens. As it passes downward it films over the large surface of the screens until it also reaches a reservoir at the bottom of the oxygenator.

Rotating disk oxygenators are composed of a series of solid disks within a horizontal glass cylinder. The disks may be corrugated to increase their surface. The lower one-third of the disk is immersed in the blood. As the disks rotate at approximately 100 revolutions per minute a film of blood is carried upward with the rotating disk and exposed to the oxygen in the upper part of the chamber. By the time the blood reaches the outlet it is fully oxygenated. These are highly efficient oxygenators and like the stationary screen oxygenators do not introduce bubbles into the blood.

Membrane type oxygenators depend upon the transmission of oxygen and carbon dioxide through a membrane of Teflon, polyethylene or silicone rubber. These materials have high rates of oxygen transmission and there is never a direct contact between the blood and a gaseous medium. Thin layers of blood are passed between the membranes which are in a high oxygen atmosphere. The volume in these oxygenators tends to remain relatively constant, whereas the amount of blood in the other types of oxygenators may under some conditions vary considerably. The membrane oxygenator is still in the earlier phases of its development, but it appears to have certain theoretical advantages which are particularly helpful. At the present time in experienced hands all of these types of oxygenators are producing satisfactory results.

Disposable oxygenators of several types are being developed to simplify the procedure further. Blood used in the heart-lung apparatus is heparinized when it is secured from donors rather than being taken in an acid citrate dextrose medium.

When the extracorporeal by pass is used, patients are heparinized prior to the introduction of catheters into the cavae and the systemic artery. 2 mg. of heparin per kg. of body weight produces adequate heparinization. At the conclusion of the by pass the anticoagulant effect of the heparin is neutralized by the administration of protamine sulfate. The complications following by pass are hemorrhage, air embolism, acidosis, hemolysis and pulmonary complications. In experienced hands the complications secondary to the use of cardiopulmonary by

pulmonary bed. The aortic pressure gradually rises so that shortly after birth the aortic pressure is slightly higher than the pulmonary. Thus, in neonatal life, flow through the ductus is minimal and, in the normal course of events, anatomic closure of the ductus occurs in the majority of infants between two and six weeks following birth.

Failure of the ductus to become obliterated results in the persistence of the communication between the pulmonary artery just to the left of its bifurcation and the aorta at a point just distal to the left subclavian artery. As the aortic pressure continues to increase, there results a shunt from left to right, that is, from the aorta to the pulmonary artery. The amount of blood which is so shunted will vary with the size and position of the ductus and with the relationship of the pressures in the aorta to the pulmonary artery. In most instances the aortic pressure remains higher than the pulmonary pressure throughout all phases of the cycle, but the greatest flow tends to occur during systole.

In adults, the amount of blood which is shunted from left to right in this way varies from 1 to 10 liters per minute. This lesion therefore results in increased work for the left ventricle since a portion of the blood which is ejected into the aorta is lost in being pumped back again into the pulmonary circuit and is returned to the left side of the heart. There is an increased pulmonary arterial flow, increased left ventricular work, and a decreased effective systemic outflow.

A reversal of shunt sometimes occurs in the presence of a patent ductus and this is characterized by pulmonary hypertension and cyanosis of the lower extremities, because of shunting of blood from the unsaturated pulmonary circuit into the systemic circuit. The upper extremities and head are not cyanotic since all of the blood which flows through the aortic root is fully saturated. In such instances the pulmonary resistance is extremely high.

Patent ductus is frequently associated with other lesions of the heart, particularly with coarctation of the aorta, ventricular septal defect, transposition of the great vessels and truncus arteriosus. The ductus arteriosus also remains patent for a time in association with the tetralogy of Fallot and the symptoms of tetralogy become more marked when the ductus closes.

The symptoms of patent ductus arteriosus vary greatly. There may be no symptoms, but those which do appear are associated

with left ventricular failure, pulmonary engorgement or endarteritis. Heart failure in infancy or childhood should always be investigated for the possible presence of a patent ductus arteriosus. Subacute bacterial endarteritis is a relatively common complication of this lesion and may be associated with chills, fever, malaise and emboli.

Physical examination shows findings which are classically confined to the cardiovascular system. A machinery type of murmur is present over the second left intercostal space in 95 per cent of patients. This murmur is to and fro with a systolic accentuation and is almost pathognomonic, but it is also present in association with aorticopulmonary fistula—a communication between the base of the aorta and the root of the pulmonary artery—and rarely in patients with aortic insufficiency associated with a ventricular septal defect. A venous hum, coronary arteriovenous fistula or pulmonary arteriovenous fistula may produce a similar murmur. In some patients, the murmur may be purely systolic. There may be no murmur heard when the pulmonary and aortic pressures are almost equal.

The heart size tends to be normal or slightly larger than normal. The pulmonary second sound is accentuated if there is pulmonary hypertension. If the ductus is small to average in size, the blood pressure may be entirely within normal limits. Because of the valveless communication between the aorta and pulmonary artery a low diastolic pressure and a wide pulse pressure are frequently noted. When the widening of the pulse pressure is present, the pulse is Corrigan in type.

On x-ray films the cardiac contour is usually normal. The pulmonary markings are slightly accentuated in some cases. Fluoroscopic copy will show pulsations of the secondary pulmonary arteries with "hilar dance." Angiocardiography may reveal the presence of the ductus by direct visualization from the aorta or it may show recanalization of the pulmonary vessels after the blood has reentered the pulmonary circulation from the aorta. Direct aortography may show opacification of the pulmonary artery after direct injection of a contrast medium into the aorta just distal to the left subclavian artery.

Cardiac catheterization should be employed in patients in whom doubt of the correct diagnosis exists. The characteristic finding is increased oxygenation in the pulmonary artery. In general, pulmonary arterial pressures are not usually greatly in

reached. It would appear that in order to lower the body temperature safely below 30 C. cardiopulmonary bypass should be used as an adjunct to maintain effective circulation during these critical periods when ventricular fibrillation and cardiac standstill may occur and to be able to raise satisfactorily the temperature of the body for resuscitation. However it has been possible by external means alone in a few cases to lower the body temperature to the point of cardiac arrest and satisfactorily revive the patient. It would appear probable that in the near future it will be possible to maintain individuals in a prolonged state of suspended animation by the combined use of extracorporeal circulation and control of body temperature.

When hypothermia is used alone body temperature of 30 to 32 C. would appear optimal, combining the maximal benefits with minimal risk. At this temperature the body metabolic requirements are reduced by approximately 50 per cent. Cardiac irritability remains essentially normal. Hypothermia may be induced by external cooling or by cooling of the blood. The external means is most commonly employed. Reduction of body temperature is accomplished by reduction of the environmental temperature under anesthesia. Sufficient anesthesia or other drugs must be used to abolish shivering, since this is a mechanism for heat production. Cooling may be accomplished by immersion of the patient in a bath of cold water with care not to allow contact of ice with the skin. Because of the high heat conductivity of water it is the most effective simple mechanism for the transfer of heat from the body to the bath. Those methods which employ only dry cold are much slower than those in which the body is in direct contact with a water medium. An adult patient can be cooled by external means to a body temperature of 32 C in approximately one hour when an efficient cooling system is used. There is usually some drift of the temperature downward after the initial cooling phase and means must be available to prevent this drift from being excessive. A mattress placed on the operating table through which hot or cold fluid can be circulated is the method usually employed for the final regulation of the temperature during the operative procedure itself. Following completion of that part of the operative procedure for which the lowered temperature is required, the patient can be immediately rewarmed by passing warm

fluid through the mattress. Some surgeons have used a diathermy coil around the pelvis for rapid rewarming in selected cases.

Systems for the cooling of blood have employed the principle of removing blood through an artery or vein, passing it over a large surface in contact with a cold environment, and then returning it to the body by a pumping mechanism. The devices which are used to regulate the body temperature under these circumstances are known as heat exchangers. Most employ the principle of multiple metal tubes of small diameter through which the blood circulates. The tubes are surrounded with a fluid medium which can be heated or cooled to the desired degree. Other heat exchangers pass the blood over a series of tubes through which the cold fluid circulates. In such devices the same principles which apply to the artificial heart lung machine are employed. In such heat exchangers it is possible to lower body temperature at rates of 1 per minute and to rewarm at approximately one-half this rate. These devices give accurate and rapid control of the body temperature at all times.

Drugs may be used to reduce ventricular irritability during intrathoracic operations. Prostigmine, Xylocaine, quinidine and Pronestyl have been advocated for prevention of ventricular fibrillation under hypothermia. Block of the sinoatrial node with procaine has also been employed. While these measures appear helpful, particularly in the temperature range of 28 to 30 C., they are less effective at lower temperatures.

With refinements in the methods of hypothermia and the extracorporeal circulation, it is apparent that many new procedures are now possible which were previously beyond the surgeon's technical ability.

PATENT DUCTUS ARTERIOSUS

The ductus arteriosus is normally patent during intrauterine life and permits a large proportion of the right ventricular blood to pass from the pulmonic circuit into the systemic pathway by way of the descending aorta. The lungs at this period are relatively unexpanded and the volume of blood flow through them is small because of the high pulmonic resistance. At birth, with the expansion of the lungs and the assumption of the oxygenation function by the lungs the resistance in the pulmonary circuit is greatly diminished. With this decreased resistance in the capillary bed of the lungs the pulmonary pressure decreases and the outflow of the right ventricle passes through the

left subclavian artery. This lesion is embryologically related to truncus arteriosus. In the case of aortic septal defects the aortic and pulmonary valves are normal in development in most cases, but there is a partial defect in the septum between the aorta and the pulmonary artery. If the septum between the aorta and the pulmonary artery is completely absent inferiorly to the level of the aortic and pulmonary valves, the lesion is a truncus arteriosus. In the latter case, a single valve may be present at the base of the truncus and the origin of the pulmonary blood supply is highly variable.

The dynamics of aortic septal defect are similar to those of a ductus. Aortic septal defects may be large and when this is the case there may be major elevations of pulmonary pressure.

When pulmonary congestion and hilar dance are present, the heart tends to enlarge gradually to a greater degree than that seen in patent ductus because the communication is usually larger. Retrograde aortography may demonstrate the lesion well. Cardiac catheterization may further delineate the lesion by showing high pressures in the common pulmonary artery and increased oxygenation in the main pulmonary artery in contrast to its branches. Passage of the catheter through the defect into the ascending arch or into the transverse arch may be diagnostic.

A continuous murmur associated with patent ductus is usually heard, but this murmur is heard in a slightly lower position than in patent ductus and tends to radiate to the right.

Treatment of aortic septal defects is closure by suture under direct vision, using extracorporeal circulation. The aorta and pulmonary artery are separated to delineate the communication. By-pass is used and the vessels are divided and the openings remaining at the site of the communication are closed by sutures. The risk of operation is low except in those individuals who have had major degenerative changes in the pulmonary artery associated with a large lesion of long standing.

The prognosis without operation is more serious than in patent ductus. The operative procedure offers total correction of the lesion.

COARCTATION OF THE AORTA

Coarctation of the aorta may be divided into two anatomic varieties—preductal and postductal. Preductal coarctation has been

termed "infantile" and is an obstruction of the aorta proximal to the insertion of the ligamentum or ductus arteriosum. Postductal coarctation ("adult") is associated with obstruction distal to the ligamentum. The confusing designations of "adult" and "infantile" should be abandoned but are mentioned since they are used throughout the early literature and to some extent at present.

The common form of coarctation is postductal in type and consists of a narrowing or complete obstruction of the aorta just distal to the left subclavian artery and distal to the insertion of the ligamentum arteriosum. The ductus arteriosus is usually obliterated. The aorta proximal to the constriction tends to be normal in size and the major proximal branches of the aorta are greatly enlarged as are the intercostal arteries distal to the obstruction. The left subclavian artery may be enlarged to approximately the size of the aorta itself. When the ductus remains patent, it usually communicates with the proximal aortic segment.

The area of aortic obstruction is usually short and the aorta is somewhat kinked at this point. The constriction varies considerably in diameter from complete obliteration to a lumen of 1 to 5 mm. in diameter. Frequently the area of narrowing is long and in adults it may be several centimeters in length. Distal to the obstruction, the aorta may be extremely hypoplastic, normal or dilated. Figure 3 shows some of the common variations of the lesion and their correction.

Aneurysms sometimes occur distal to the obstruction in both children and adults (Fig. 3D). This lesion is most often seen associated with a small lumen in the area of coarctation and is related to the jet effect of blood passing through a very small lumen. The conversion of high forward velocity to lateral pressure distal to the obstruction results in excessive stress on the lateral aortic wall and leads to the development of aneurysm. The intercostal arteries are greatly dilated and tortuous. One or more of these vessels may be aneurysmal at their junction with the aorta.

In addition to the usual site of constriction just below the left subclavian artery cases of coarctation have been reported in the mid and low thoracic aorta and in the abdominal aorta. When they occur below the usual site, the area of constriction tends to be quite long.

The etiologic background of coarctation

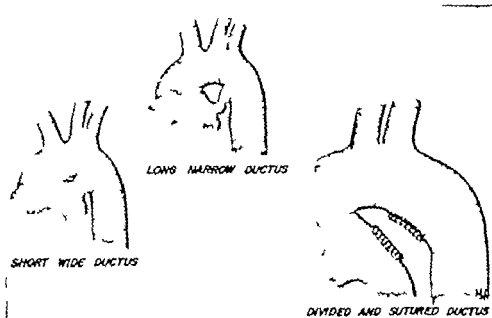


Figure 2. Patent ductus arteriosus. The two principal variations of patent ductus are both corrected by division and suture.

created. The electrocardiogram may show normal tracings or left ventricular preponderance.

Obliteration of the isolated patent ductus is accomplished by division of the ductus and suture of the cut ends (Fig. 2). Multiple ligation of the ductus has also been advocated and is carried out in some clinics. It is preferable to proceed with operation in childhood, but elective operation should be advised whenever the diagnosis is made after the patient is two years old. In the newborn period, the establishment of the diagnosis is difficult and operation is not done unless there is evidence of congestive failure or other complications which endanger the patient's life.

Contraindications to treatment are primarily general diseases which make any surgery hazardous. In the absence of symptoms in the patient over fifty years of age, correction of a ductus is not advised, but if serious signs or symptoms occur operation should be performed in individuals of any age. When the ductus is serving a compensatory function, its closure is obviously indicated only when the associated lesions are also corrected. When bacterial endarteritis is present, attempt at medical cure should be made. If a cure of the subacute endarteritis is obtained by medical therapy elective operation should be carried out in approximately six months. Surgical intervention should be done immediately if there is evidence that antibiotic therapy is ineffective.

Untreated patent ductus arteriosus has as possible complications congestive heart failure, acute bacterial infection in the region of the ductus, development of aneurysm of the ductus, the pulmonary artery or the adjacent aorta, and development of pulmonary hypertension.

Operation in childhood carries a mortality rate of approximately 1 per cent. The operative risk is between 1 and 2 per cent in adult patients who have pulmonary pressures which are normal or only slightly elevated. When pulmonary pressure is normal or moderately elevated, the indication for operation is its presence. When pulmonary pressure is elevated but less than aortic pressure, there is an urgent indication for operation. In some instances there may be transitory reversal of flow from the pulmonary artery to the aorta and operation is also indicated. When permanent reversal of the shunt has occurred, operative risk is markedly increased. This is particularly true in the adult. Opinion is divided as to whether closure of the ductus can produce benefit after permanent reversal of the shunt has occurred. It is the general opinion at the present time that if the pulmonary pressure cannot be lowered to levels less than the aortic pressure, closure is contraindicated.

AORTIC SEPTAL DEFECTS

Aortic septal defects are less common congenital anomalies in which a communication is present in the common pulmonary artery beyond the aortic valve and proximal to the

X-ray examination of the chest demonstrates a normal or enlarged cardiac silhouette. There is frequently a diminished aortic shadow in the region of the aortic arch, but the lack of the normal aortic prominence is variable. Notching of the ribs seldom is noted in childhood, but it is commonly seen after the age of ten. Angiocardiography or direct aortography will demonstrate the zone of constriction.

Electrocardiography usually shows left ventricular hypertrophy, left axis deviation or left ventricular strain.

Demonstration of the anatomic lesion in the patient under forty years of age is an indication for operation. Evidence of continuing cardiac enlargement or signs of congestive failure also are indications for operation. In the older age groups, signs of severe hypertension, cardiac strain or recent bacterial endarteritis are indications for operation.

In the infant, uncontrollable congestive failure sometimes necessitates operation. An attempt should be made to control the failure medically since many infants after their initial bout of failure, can be maintained well on good medical management until late childhood.

The optimum time for operation is when the patient is between six and twelve years of age. Operative mortality and complications are higher in infants and in patients beyond the age of forty years. When severe aortic insufficiency is also present, operation for both lesions should be done simultaneously. In the presence of aneurysm, operation should be performed with resection of both the aneurysm and the coarctation.

Excision of the area of constriction with end-to-end anastomosis is the simplest and most effective form of treatment of coarctation of the aorta. In the usual case, the aorta is freed from its bed through a left thoracotomy. The area of the stricture is isolated and the ligamentum arteriosum is divided. If the ductus is patent, the ends are oversewn in the usual fashion. Noncrushing clamps are applied to the aorta above and below the area of constriction, and, after excision of the constricted segment, an end-to-end suture is carried out. In children under the age of ten years, it is almost always possible to accomplish this type of procedure. Occasionally the presence of an aneurysm may necessitate considerable mobilization of the aorta in order to get the ends together.

In some instances and particularly in

adults, a long constriction or large aneurysm of the aorta is seen and, because of the inelasticity of the aortic wall it is not possible to bring the ends directly together. A homograft or arterial prosthesis can be used to bridge the gap between the aortic ends so that the anastomosis can be made without excessive tension (Fig. 3B to D). Arterial homografts have been used for this purpose but are now only occasionally employed. Flexible cloth prostheses of Dacron or Teflon have been more satisfactory. It is undesirable to use a prosthesis or homograft in infants or young children because of the lack of growth of these replacements. The elasticity of the aorta in the younger age groups is so great that a direct anastomosis can be accomplished in almost all postductal coarctations.

Surgical correction of the preductal type of coarctation may be accomplished by direct anastomosis, utilizing the aortic end of the ductus. The preductal type is frequently associated with a long area of narrowing, involving either the distal transverse aortic arch or the entire isthmus of the aorta. In these instances an additional aortic arch is created by suturing a vascular prosthesis or homograft proximal to the area of obstruction in the ascending arch and making a second end-to-side anastomosis to the aorta beyond the area of coarctation. Those vessels which are supplied by the narrowed, but not completely obstructed, arch continue to carry blood without interruption.

Results of surgery in coarctation of the aorta of the postductal type are excellent. The blood pressure of the upper extremities can be expected to fall to normal limits and the blood pressure in the lower extremities rises. Symptoms of congestive failure are relieved. The mortality for operations of coarctation without complicating features is under 5 per cent and only slightly higher in those associated with long strictures or aneurysms. The major complications are infection of the suture line, renal failure and occasionally neurologic complications associated with interference to the blood supply of the lumbar cord.

VASCULAR RINGS

Anomalous development of the aortic arch and its branches leads to a number of conditions which produce compression of the trachea, esophagus or both. The most common of these conditions are double aortic arch (Fig. 4) origin of the right subclavian artery from the left side of the aortic arch,

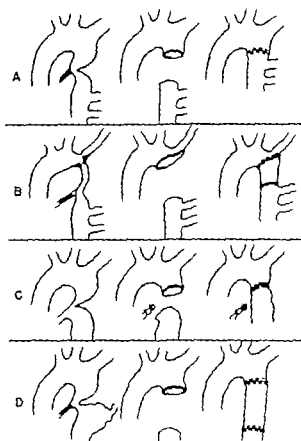


Figure 3 A, The common postductal coarctation is shown before and after direct repair B Combined preductal aortic narrowing with obstruction of the left subclavian artery. Repair with a prosthesis shows removal of subclavian obstruction. C Coarctation with patent ductus arteriosus. The repair utilizes a part of the aortic end of the ductus D Postcoarctation aneurysm and its correction with a prosthesis.

of the aorta is unknown. Many theories have been advanced to relate the presence of coarctation to the insertion of the ductus, but none are generally acceptable.

Collateral circulation in the adult type of coarctation is derived through the subclavian arteries and their branches: the internal mammary artery, the costal cervical trunk, the superior intercostal artery and the thyrocervical trunk, the transverse cervical artery, the transverse scapular artery and the intercostal arteries. The continuation of the internal mammary as the inferior epigastric artery anastomoses with the external iliac artery and the anastomoses between the inferior epigastric arteries and the lumbar arteries form additional channels.

In some patients the left subclavian artery instead of being enlarged, is hypoplastic or is involved in the coarctation. The collateral circulation will be entirely right sided in such subjects. When the coarctation lies in

the low thorax or abdomen, the upper collateral channels are distinctly less well developed than when the coarctation is high. Collateral circulation may be quite well developed at birth, but the size of the collateral channels appears to increase out of proportion to the growth of the child and, therefore becomes much more apparent in adult life.

There may be no symptoms early in life or even well into adulthood. In infants, however if they do appear they are chiefly those of congestive failure or of bacterial infection. Later in life, additional symptoms are associated with cerebral hypertension and peripheral decreased flow. They include dizziness, headaches, syncope, shortness of breath, paralysis or ocular hemorrhage. Coldness and fatigue of the lower extremities and intermittent claudication may also be noted.

Physical examination characteristically reveals elevated blood pressure in the upper extremities and lowered blood pressure in the legs. In a considerable number of patients the blood pressure in the upper extremities will be normal or only slightly above normal, but palpation of the femoral pulses will show them to be greatly diminished or absent and the blood pressures in the legs will be lower than in the upper extremities. If the left subclavian artery is hypoplastic, the blood pressure in the left arm may be greatly lower than that in the right. If the right subclavian artery rises from below the coarctation, the blood pressure in the right arm will be lower than that in the left.

Examination of the chest may show a slight to great enlargement of the heart. There may be no murmurs present, but in most patients a systolic murmur is heard in the aortic area. This murmur is transmitted through to the back and the interscapular region. The murmur is usually maximal near the area of constriction. If the coarctation is in an unusual position, the site of the maximal murmur may give some clue as to whether it is low in the thorax or abdomen. If a diastolic murmur is also present, this is usually associated with a regurgitant bicuspid aortic valve or patent ductus arteriosus. The presence of enlarged collaterals may be noted most easily over the interscapular region in back by palpation and visual examination. Murmurs heard over the back, both on the right and left, may frequently be obliterated by compression of the dilated intercostal arteries.

um in the esophagus and trachea. Angiography may be necessary to elucidate further the more complex lesions by visualization of branches of the aortic arch.

Surgical treatment is directed toward the decompression of the trachea and esophagus. When a double aortic arch is present, one arch is usually larger than the other. By a careful dissection of the two components, one can determine the origin of the blood supply to the head. It is then possible to isolate the smaller branch and, following the application of noncrushing arterial clamps to divide this limb of the arch from the main aortic arch near the point at which the two components unite. This releases the circular constriction and relieves the compression.

When the ligamentum arteriosum is the dominant feature producing the ringlike obstruction, the division of this ligament itself is sufficient to release the compression. Division of an aberrant right subclavian artery will also remove the constriction caused by its posterior course. Reimplantation of the right subclavian artery into the right portion of the aortic arch can also be carried out without difficulty.

Suspension of the aortic arch or one of its branches anteriorly to relieve the compression from its posterior displacement may be necessary in some cases. This is particularly helpful in the lesions in which the carotid arteries arise from the innominate or from a left origin of a right innominate.

Operation must often be carried out in infants whose general condition is extremely poor because of the presence of pulmonary infection from aspiration of esophageal contents. This complication represents one of the serious hazards of the condition which jeopardizes life. If the diagnosis can be made prior to the development of such pulmonary complications, the over-all results are uniformly good and the prognosis thereafter is excellent.

PULMONIC STENOSIS

Pulmonic stenosis is of four general types: valvular, infundibular, pulmonary arterial and combined.

Stenosis of the pulmonary valve associated with another major lesion or lesions is one of the most frequently occurring congenital cardiovascular defects. Isolated pulmonary stenosis and pure pulmonic stenosis are terms which are customarily used to indicate pulmonic stenosis without an interventricular

septal defect. However these terms are also applied to pulmonic stenosis associated with a patent foramen ovale.

If the pulmonic stenosis exists alone cyanosis is not present except as an accompaniment of extreme heart failure. When a patent foramen ovale is present, cyanosis occurs and the amount of shunting from right to left determines the degree of cyanosis. In such patients the patency of the foramen ovale is maintained by a high right atrial pressure and the pulmonic stenosis is usually the major lesion.

In the valvular type the valve cusps are fused to a varying degree, producing all gradations from slight obstruction to almost complete occlusion of the right ventricular outflow tract. The cusps are usually united in a conical or domelike structure with an orifice at the apex. Immediately distal to the valve, the pulmonary artery is dilated in the vast majority of cases. This dilatation distal to the area of obstruction is similar to that which is seen in coarctation of the aorta and is also related to high velocity flow through a narrow orifice into an area of lower pressure.

The second variety is termed infundibular stenosis. In this malformation, a ridge of hypertrophied muscle, the crista supraventricularis or a membranous diaphragm narrows the pulmonary outflow tract (infundibulum) of the right ventricle. The infundibular type of stenosis may be immediately subvalvular or it may be quite low in the ventricle. Between the area of infundibular obstruction and the pulmonary valve is a zone which is sometimes referred to as the third ventricle. In this area the ventricular muscle is usually thin, in contrast to the hypertrophied muscle of the right ventricle proximal to the obstruction.

Obstruction to the right ventricular outflow tract may be caused by a variant of infundibular stenosis in which the valvular structures are normal but in which there is hypertrophy of the muscle of the right ventricle over a long distance below the valve. No zone of localized narrowing is manifest, but marked obstruction to flow exists.

In contrast to this are tetralogy of Fallot and other defects in which there is communication between the right and left sides of the heart with right to-left shunts for in these the systemic blood is highly unsaturated.

In isolated pulmonary stenosis, approxi

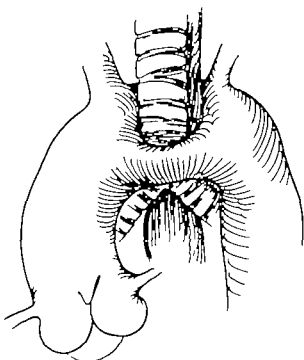


Figure 4 A vascular ring formed by a double aortic arch illustrating encroachment upon the tracheoesophageal space.

origin of the left common carotid from the innominate artery origin of the left common carotid from the right side of the aortic arch and a right aortic arch with a left ligamentum arteriosum.

Compression is not only caused by the vessels themselves but may be associated with fibrous bands or remnants of other vessels which accompany the arteries themselves.

A double aortic arch is produced by the persistence of both right and left primitive arches, the anterior representing the left and the posterior representing the right. The ascending aorta bifurcates and one portion passes behind the esophagus and the other in front of the trachea to reunite to form a single descending aorta at the level of the left subclavian artery. Either the anterior or the posterior branch may be the larger and the major arterial trunks arise from either or both arches.

The right subclavian artery may arise as the last branch of the aortic arch. In this anomaly the right common carotid is the first branch of the arch, the left common carotid is the second, the subclavian artery is the third and the right subclavian artery last. The right subclavian artery then passes to the right and posteriorly behind the esophagus and upward to the arm. This posterior course compresses the esophagus

between the aortic arch and the anomalous vessel.

When the left common carotid artery arises from the innominate artery it passes anterior to the trachea and upward into the neck as the right common carotid artery pursues a similar course and the esophagus and trachea are compressed between these two major vessels. Similarly when the innominate artery rises from the left side of the arch it passes to the right upward into the neck and it compresses the trachea as it passes over it anteriorly.

When a right aortic arch persists, the ligamentum arteriosum has its insertion to the left of the common pulmonary artery in its usual site and as the arch passes behind the trachea and esophagus it is encircled posteriorly by the right aortic arch, anteriorly by the pulmonary artery to the left by the ligamentum arteriosum and behind by the transverse portion of the aortic arch. This gives a complete ring which may obstruct either the trachea or the esophagus.

As a result of the chronic tracheoesophageal compression the symptoms may be either predominantly respiratory or esophageal. Dysphagia is often a prominent symptom and the continued regurgitation of esophageal contents may give rise to considerable pulmonary infection subsequent to its aspiration. Evidence of tracheal obstruction is associated with wheezing, crowing respirations and is most often evident in association with eating. In infancy symptoms may be noted only at the time of ingestion of food and, at these times, cyanosis and extreme respiratory distress may appear. Retraction of the suprasternal and supraclavicular spaces with stridor is common.

A double aortic arch, a right aortic arch with a persistent ligament and an anomalous origin of the right subclavian artery tend to produce severe symptoms. In adults, the symptom of dysphagia tends to be more prominent than do respiratory symptoms.

Instillation of iodized oil into the esophagus shows characteristic defects of esophageal compression on the x ray film. Barium should not be used to demonstrate these defects in infants because of the danger of aspiration. The presence of a right aortic arch can be determined by the side upon which the aortic knob presents its esophageal impression. The presence or absence of constriction of the trachea and esophagus can be shown by the use of a contrast medi-

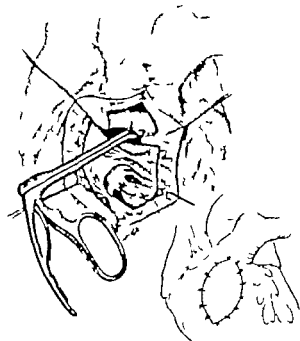


Figure 6 Resection of infundibular pulmonary stenosis through a right ventriculotomy incision.

valve and infundibulum require exposure of the outflow tract of the ventricle and the pulmonary artery. When valvular stenosis is present, a thickened obstructed valve can be palpated through the pulmonary artery and there is usually dilatation just above the obstructed valve. When the heart has been emptied of blood, an incision is made in the pulmonary artery immediately distal to the valve. The valve is seen as a conical volcano-like structure with a small opening in its center. The pulmonary valve is reconstructed by incisions in the valve to open it to the annulus. The heart is then filled with blood. Pressures are recorded in the ventricle and distal to the valve. If no gradient exists, an adequate valvulotomy has been accomplished. If a gradient still persists, the incision in the pulmonary artery must be reopened and the valve further mobilized until the gradient disappears. Infundibular obstruction is also corrected under direct vision. The use of cardiopulmonary by pass is ideal for this purpose. If the obstruction is of the localized type it is removed by excision after ventriculotomy (Fig. 6). When a long area of hypertrophied muscle presents a physiologic obstruction to flow, the outlet tract of the ventricle is incised without cardioplegia. Any localized obstructing hypertrophied muscle is removed by excision and the pulmonary outlet tract is widened

by the use of a ventricular patch. Ovoid patches of Ivalon, Teflon or combinations of these materials are sutured into the longitudinal incision in the ventricle to produce the widening of the pulmonary outlet tract. It is sometimes necessary to carry this through the annulus of the pulmonary valve and into the base of the pulmonary artery. This type of widening is also used in some cases of tetralogy of Fallot. The use of shunting procedures in isolated pulmonic stenosis is contraindicated. The results of direct operations have been excellent and the mortality rate of operation is low.

TETRALOGY OF FALLOT

Tetralogy of Fallot is a complex anomaly in which pulmonary stenosis, a high inter-ventricular septal defect, overriding of the aorta and right ventricular hypertrophy are present. Pulmonic obstruction may be valvular infundibular or in the artery itself. Valvular stenosis is similar to that which is found in pulmonic stenosis. The obstructive component may also be either hypoplasia of the pulmonary artery or a hypertrophied supraventricular crest.

The severity of the lesion depends upon the summation of the factors of the degree of pulmonary obstruction, the size of the interventricular septal defect, the degree of overriding of the aorta and the presence or absence of a patent ductus arteriosus and/or other large bronchial or mediastinal collaterals to the lung.

The degree of peripheral arterial unsaturation is determined by the amount of right ventricular blood which is expelled into the aorta. Arterial oxygen saturation of less than 60 per cent produces severe cyanosis. Polycythemia is usually present and intravascular clotting in the cerebral or pulmonary vessels is not uncommon. The greater the degree of polycythemia the more frequent is the occurrence of this complication. Patients in the older age groups may develop bronchial and other collateral circulations from the chest wall to the lung which form pleomorphic systemic-pulmonic anastomoses. An extremely high hematocrit reading is associated with a diminished fibrinogen factor in the blood and abnormalities of the clotting mechanism.

Thus, under these circumstances intravascular clotting is frequent, but the failure of the formation of a firm clot may lead to considerable bleeding during and after surgery. With an extremely high hemoglobin, the 5 gm. per 100 cc. of reduced hemoglobin necessary to produce cyanosis is more easily

mately 85 per cent of all cases are valvular in type. In tetralogy of Fallot the reverse is true the majority being of the infundibular variety

The symptoms are primarily those of decreased pulmonary flow. The patient has ease of fatigue, dyspnea on mild exertion or syncope. Patients may show some retardation of general development, but this is not always present. Cyanosis is usually absent, but if the foramen ovale remains patent, cyanosis may become marked. If the foramen ovale closes, cyanosis is absent. A history of squatting is seldom obtainable.

Cardiac enlargement of varying degrees may be present. There may be bulging of the precordium due to hypertrophy of the right side of the heart. A systolic murmur of harsh character in the second left intercostal space is characteristic. A systolic thrill is frequent. Radiation of the murmur is to the left and to the back. The pulmonary second sound is usually diminished. Signs of congestive failure occur and, if cyanosis is a dominant feature, clubbing of the fingers may also be present.

Chest films show enlargement of the right atrium and right ventricle. The pulmonary conus is prominent because of enlargement of the main pulmonary artery just distal to its exit from the ventricle. The periphery of the lung fields is relatively avascular. Angiocardiography will show delay in emptying of the right ventricle and a dilated pulmonary artery distal to the valve. Injection of contrast medium directly into the right ventricle may demonstrate either infundibular or valvular type of obstruction. The electrocardiogram shows right axis deviation and right ventricular hypertrophy.

Cardiac catheterization exhibits high right ventricular pressures and a sharp fall as the catheter enters the pulmonary artery. No shunt in the ventricle is demonstrable. If the foramen ovale is patent, the catheter can enter the left atrium or pulmonary veins. Pressures in the right ventricle may be elevated to approach systemic levels. The important diagnostic criterion is the presence of a gradient between the pulmonary artery and the right ventricle. Catheterization studies show that the intensity of the murmur is not directly related to the degree of obstruction. Thus, patients with extremely high right ventricular pressures and very low pulmonary artery pressures may have relatively low intensity murmurs and those who have relatively small degrees of obstruction may have loud murmurs.

The over-all prognosis in pulmonary stenosis is related to the degree of obstruction. Patients who have relatively small degrees of obstruction do well for prolonged periods and may show no disability in spite of the presence of very loud murmurs. Patients with a high degree of obstruction tend to develop congestive failure early and to have severe symptoms. If operation can be advised before the appearance of severe symptoms and congestive failure, the operative risk is extremely low. If congestive failure or a patent foramen ovale is present with severe cyanosis, operative mortality is markedly increased.

The surgical treatment of isolated pulmonary stenosis is directed toward the removal of the obstruction in the valve or in the outlet tract of the right ventricle. Valvular obstruction may be relieved by pulmonary valvulotomy or pulmonary valvuloplasty. Patients with congestive failure, right ventricular pressures of over 70 mm. of mercury or severe symptoms should be selected for operation.

Operative treatment of pure valvular stenosis is best accomplished by direct visualization of the valve (Fig. 3). Hypothermia or cardiopulmonary by pass is used to obtain a dry heart. All operations on the pulmonary

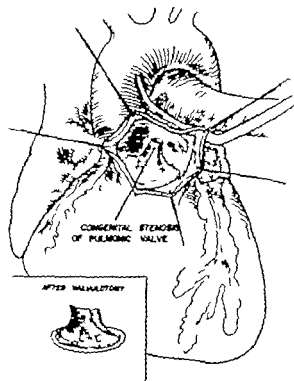


Figure 3. Congenital pulmonary valvular stenosis as seen at operation. Note that the incisions are carried to the edge of the annulus.

ated into the pump oxygenator and a right ventriculotomy is performed. The interventricular septal defect is closed and the overriding corrected by the closure. Patches of Ivalon Teflon or Dacron are used to add substance to the deficient septum. The stenosis of the pulmonary outlet tract is corrected by resection of the infundibulum, widening of the outlet tract or opening of the pulmonary valve as indicated by the particular lesion or combination of lesions. Cardiac arrest may be used in selected instances but is generally avoided. Care is required in closure of the septum to avoid placing stitches in the bundle of His. The results of total correction are excellent and it is anticipated that the problems which arise fol-

lowing the shunting operations will not be seen. Complications of open operation are infections in the suture line failure to obtain complete closure of the septal defect or failure to remove completely the pulmonary obstruction. The risk of such procedures has markedly decreased and approximates that of the indirect operation.

ATRIAL SEPTAL DEFECTS

Atrial septal defects exist in a wide variety of forms which may be grouped into the following classifications: absent septum (single atrium), persistent ostium primum, persistent ostium secundum, atrioventricularis communis. A persistent patent foramen ovale with an incompetent valve or absent

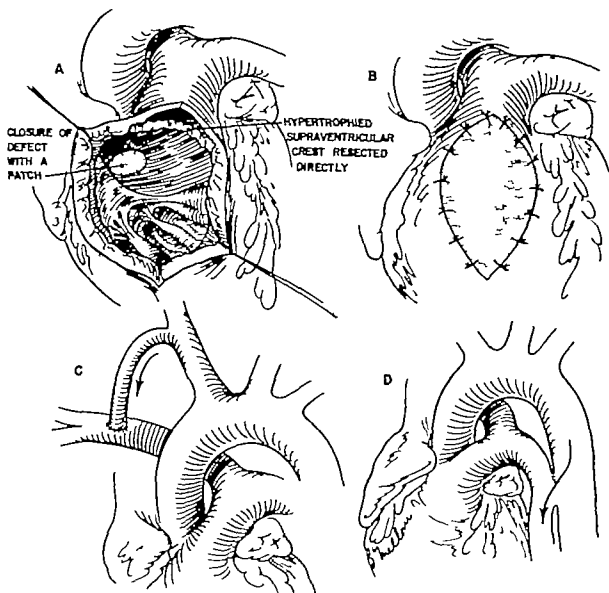


Figure 7 A, Direct operation for tetralogy of Fallot. Closure of the septal defect and resection of infundibular obstruction are shown. B, Widening of the right ventricular outflow tract is achieved by saturating of an oval patch in the linear incision in the right ventricle. C, Subclavian pulmonary anastomosis for tetralogy of Fallot (Blalock). D, Aortopulmonary anastomosis for tetralogy of Fallot (Potts).

accumulated than in the patient with a normal hemoglobin level.

Early in neonatal life the ductus arteriosus tends to remain open and during this period the patient may exhibit relatively few signs and symptoms. Closure of the ductus may be heralded by increasing cyanosis, dyspnea and syncope. Cyanosis is usually noted early in life and is intensified by physical activity, crying or eating. Cyanosis tends to be persistent once it has appeared, although it may fluctuate in intensity. A general retardation of growth is frequently seen. Mental abilities do not appear to be particularly affected. Dyspnea and ease of fatigue are prominent symptoms. The assumption of the squatting posture appears to give these individuals relief and even very small children instinctively assume this position when they have overexerted themselves. Respiratory infections are common as is the occurrence of congestive heart failure.

Syncope with or without convulsions may occur. The presence of syncope tends to be a poor prognostic sign and, indeed, death may occur during such an episode. Because of the mixing of the two circulations, bacteremia and the development of cerebral abscess are also seen.

Physical examination may show cyanosis of the mucous membranes and also clubbing of the fingers or toes. A harsh systolic murmur often associated with the thrill is heard over the pulmonic area. The pulmonary second sound may be absent, diminished or normal.

X-ray examination of the heart shows the organ to be normal in size or slightly enlarged. The cardiac silhouette is classically bootlike in shape, characteristic of an enlarged right ventricle with diminution in the size of the common pulmonary artery as it leaves the right ventricle. Poststenotic dilatation of the pulmonary artery may be present with valvular or high infundibular obstruction. A clear pulmonary window may be observed when the pulmonary artery is small. The lung fields tend to appear quite clear and without the normal prominence of the pulmonary arterial markings. The absence of vascular markings in the outer half of the lung fields is particularly striking. The pulmonary artery and the hilum have decreased or absent pulsations. In adults the pulmonary markings may not show the characteristic avascularity observed in children and infants. The aorta descends on the right in approximately 20 per cent of the subjects and can be demonstrated by barium swallow.

Angiocardiography will show simultaneous opacification of the aorta and right ventricle. The area of pulmonary obstruction and a right to-left shunt may be seen on injection of contrast medium directly into the right ventricle through a catheter or by intravenous angiocardiography.

Cardiac catheterization shows high right ventricular pressures and diminished pressure in the pulmonary artery. The catheter may pass into the aorta through the inter-ventricular septal defect. The right ventricular blood may show an increased oxygenation from the ventricular communication.

Tetralogy of Fallot is quite common and it has been estimated that approximately 70 per cent of children over two years of age who demonstrate a congenital lesion associated with cyanosis have tetralogy of Fallot. The prognosis depends on the relation of the severity of the various defects to the degree of compensatory mechanism present. The vast majority of these patients die in infancy or early childhood with progressive cyanosis, polycythemia and syncope. A few patients will survive to relatively late childhood and a still smaller number will live to adult life. Those who have severe lesions tend to have difficulty in infancy.

The operative risk in such patients is high and the earlier that operation must be performed because of progressive signs or symptoms, the higher is the mortality rate. Operative intervention in patients younger than two years carries an over-all risk of approximately 25 per cent. In patients past the age of three years, the operative mortality rate is under 5 per cent. Successful operation can be expected to eliminate the cyanosis and to permit degrees of activity which were previously not possible.

Early procedures for the amelioration of tetralogy of Fallot were based upon the delivery of increased amounts of unoxygenated blood to the pulmonary artery by the production of a shunt between the aorta or one of its branches and the pulmonary artery. These procedures were the subclavian-to-pulmonary-artery shunt and the direct aortic-pulmonary shunt. These procedures are of historical interest and are still used in selected instances. Total correction of tetralogy of Fallot by open operation is the procedure of choice (Fig. 7).

This can be accomplished by inflow occlusion under hypothermia with localized aortic perfusion with oxygenated blood or by the customary cardiac bypass. By means of the standard method, the inflow is devi-

stress. With the development of high pulmonary arterial pressures over prolonged periods reversal of the shunt may occur and cyanosis may become permanent. The heart is usually enlarged and the characteristic systolic murmur is best heard over the second or third left intercostal space. The murmur of an atrial septal defect is not entirely characteristic and may vary widely in intensity. The pulmonic second sound tends to be loud because of the increased pulmonary flow and increased pulmonary pressure. Fixed splitting of the pulmonic second sound is present in a high percentage of cases. Signs of congestive failure may be present. Electrocardiograms show right ventricular enlargement and/or right ventricular strain. P waves are usually prominent and right bundle branch block of varying degree is the rule in defects of the secundum type. Vectorcardiograms are usually characteristic in primum lesions.

X ray films of the heart show enlargement of the right atrium and right ventricle. The pulmonary artery and pulmonary vascular markings are prominent and pulsations of the secondary vessels are notable. Angiocardiography shows refilling of the right atrium from the left. In the presence of a pure left-to-right shunt, contrast medium cannot be visualized to pass from the right to the left side.

Cardiac catheterization is usually helpful to make certain no additional lesion is present and to determine the volume of the shunt. The catheter may pass directly into the left atrium or left ventricle or a left-to-right shunt may be evident by increased oxygenation in the right atrium. The catheter can also enter the pulmonary veins particularly those on the right. Entrance of the catheter into the pulmonary veins does not necessarily indicate anomalous venous drainage, although abnormalities of the pulmonary venous drainage are commonly associated with the atrial septal defect. By calculation of the right and left ventricular outputs, one may determine the amount of blood which is shunted per minute. In many patients the right ventricular output may be three or more times that of the left. Dye dilution or nitrous oxide techniques permit determination of the site of the shunt and quantitation of the volume of shunt.

Operation is indicated in all patients who exhibit definite signs of progressive cardiac damage, evidence of pulmonary hypertension, signs of congestive failure and those in whom the calculated pulmonary blood flow

is more than twice the systemic flow. It is preferable to advise operation before the development of excessive changes in the pulmonary arterial pressures has taken place.

Patients who develop signs of major cardiac enlargement usually show symptoms of serious difficulty. The large defects may give rise to congestive failure early in life, but the majority of patients develop their symptoms in adult life. If the existing shunt shows a high rate of pulmonary blood flow and relatively normal pulmonary resistance and only moderate pulmonary hypertension, operation is extremely safe. Patients with extremely high pulmonary resistance carry a much more serious prognosis and operation is more hazardous. It is advisable to correct such defects in childhood or early adult life, but infants below the age of one year should be offered operation only when there is evidence of a serious problem. Compensatory interatrial communications should not be closed, except as a part of an over all procedure in which the primary defect is corrected. When patent foramen ovale is associated with pulmonic stenosis, the correction of the latter is usually the only procedure necessary.

Surgical methods for the correction of atrial septal defects are of two types—closed

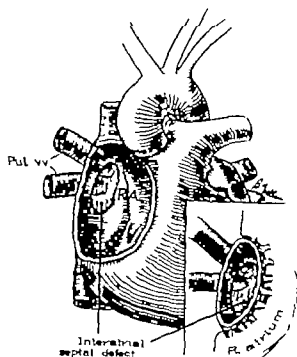


Figure 8. A posterior interatrial septal defect is shown before and after closure by the extracorporeal method. The anterior right atrial wall is approximated to the anterior edge of the defect which deviates the pulmonary venous return entirely into the left atrium.

valve represents another type of atrial septal defect.

Absence of the atrial septum represents failure of development of the primitive Anlage of the atrial septa. The persistence of an ostium primum is associated with the failure of the septum primum to establish fusion with the lowermost portions of the atrium. This defect presents a deficiency of tissue just above the tricuspid and mitral valves. Abnormalities or deficiencies of either or both of these valves occur with this defect. A cleft mitral and/or tricuspid valve is the commonly associated lesion. Atrioventricularis communis represents failure of the fusion of the septum primum to the lower portions of the atrioventricular canal, the atrioventricular cushions. The persistent ostium secundum is the most frequent true septal defect and represents failure of development or fusion of the septum secundum with the septum primum.

During the fourth week of gestation, the separation of the atria and the ventricles begins. The sinus venosus enters into what ultimately becomes the right atrium. The septum primum begins its development as a crescentic ridge on the superior and dorsal portion of the single atrium. It grows downward toward the atrioventricular canal. The common atrioventricular channel is gradually divided into right and left channels by the endocardial cushions. The septum primum in the meantime has gradually grown inward and downward toward the endocardial cushions until it fuses with them. Just prior to the union of the septum primum with the atrioventricular cushions, the uppermost portion of the septum primum becomes resorbed so that the two atria are still in free communication. In the meantime the interventricular septum has gradually become more prominent and finally also fuses with the endocardial cushions at a point which is ultimately just below the annulus of the valve rings. The septum secundum then begins to develop just to the right of the point at which the septum primum had initially originated. It arises as a long ridge along the entire dorsal aspect of the atrium and grows ventrally and downward but does not completely close to form a solid wall. The remaining central opening is the foramen ovale which is covered on its left aspect by the inferior remaining portion of the septum primum. This uppermost remaining segment of the remnant of the septum primum does not fuse with the edges of the septum secundum before birth but

remains as a flap-type valve which permits flow from right to left but does not permit flow from left to right. It is only with the assumption of pulmonary function after birth that cessation of flow from right to left allows the gradual fusion of the uppermost portion of the septum primum with the septum secundum, resulting in complete separation of the two atria. From these considerations it is apparent how the various types of defects occur.

Abnormalities in the development of the septum secundum cause the posterior lying defects to occur. Failure of fusion of the septum primum with the endocardial cushions gives rise to the ostium primum type of defect. Excessive resorption in the septum primum permits the persistence of the foramen ovale. This highly simplified explanation of the developmental aspects of interatrial defects can be expanded to cover the other variations.

In general, the larger the size of the defect the more rapid is the development of symptoms and the greater the amount of the shunt. In the presence of normal pulmonary resistance the shunt is predominantly from left to right. When the defect is large, the shunt may be in both directions and this may result in a predominant left-to-right shunt, but with some degree of peripheral arterial unsaturation. Phenomena which increase the pulmonary pressure may cause a reversal of the left-to-right shunt and thus produce transient cyanosis.

When the atrial defect is small, there may be minimal shunting and no symptoms may exist. There is frequently a history of cyanosis, particularly in infancy and in early neonatal life. Cyanosis may be particularly noted during periods of crying, feeding and respiratory infection but cyanosis tends to be periodic. In a large percentage of patients no history of cyanosis can be obtained even in the presence of a fairly large shunt. Limitation of exercise tolerance or underdevelopment may be the only sign in early life. Large shunts in infants may produce signs of congestive failure. The onset of symptoms in the majority of patients is noted in late childhood or early adult life. Arrhythmias are common and, with the development of the lesion, signs of dyspnea and fatigue appear.

Physical examination frequently shows bulging of the left precordium. Cyanosis is present when there is an associated lesion, particularly pulmonic stenosis, serious anomalous venous drainage or during periods of

stress. With the development of high pulmonary arterial pressures over prolonged periods, reversal of the shunt may occur and cyanosis may become permanent. The heart is usually enlarged and the characteristic systolic murmur is best heard over the second or third left intercostal space. The murmur of an atrial septal defect is not entirely characteristic and may vary widely in intensity. The pulmonic second sound tends to be loud because of the increased pulmonary flow and increased pulmonary pressure. Fixed splitting of the pulmonic second sound is present in a high percentage of cases. Signs of congestive failure may be present. Electrocardiograms show right ventricular enlargement and/or right ventricular strain. P waves are usually prominent and right bundle branch block of varying degree is the rule in defects of the secundum type. Vectorcardiograms are usually characteristic in primum lesions.

X ray films of the heart show enlargement of the right atrium and right ventricle. The pulmonary artery and pulmonary vascular markings are prominent and pulsations of the secondary vessels are notable. Angiocardiography shows refilling of the right atrium from the left. In the presence of a pure left-to-right shunt, contrast medium cannot be visualized to pass from the right to the left side.

Cardiac catheterization is usually helpful to make certain no additional lesion is present and to determine the volume of the shunt. The catheter may pass directly into the left atrium or left ventricle, or a left-to-right shunt may be evident by increased oxygenation in the right atrium. The catheter can also enter the pulmonary veins, particularly those on the right. Entrance of the catheter into the pulmonary veins does not necessarily indicate anomalous venous drainage, although abnormalities of the pulmonary venous drainage are commonly associated with the atrial septal defect. By calculation of the right and left ventricular outputs, one may determine the amount of blood which is shunted per minute. In many patients the right ventricular output may be three or more times that of the left. Dye dilution or nitrous oxide techniques permit determination of the site of the shunt and quantitation of the volume of shunt.

Operation is indicated in all patients who exhibit definite signs of progressive cardiac damage, evidence of pulmonary hypertension, signs of congestive failure and those in whom the calculated pulmonary blood flow

is more than twice the systemic flow. It is preferable to advise operation before the development of excessive changes in the pulmonary arterial pressures has taken place.

Patients who develop signs of major cardiac enlargement usually show symptoms of serious difficulty. The large defects may give rise to congestive failure early in life, but the majority of patients develop their symptoms in adult life. If the existing shunt shows a high rate of pulmonary blood flow and relatively normal pulmonary resistance and only moderate pulmonary hypertension, operation is extremely safe. Patients with extremely high pulmonary resistance carry a much more serious prognosis and operation is more hazardous. It is advisable to correct such defects in childhood or early adult life but infants below the age of one year should be offered operation only when there is evidence of a serious problem. Compensatory interatrial communications should not be closed, except as a part of an over-all procedure in which the primary defect is corrected. When patent foramen ovale is associated with pulmonic stenosis the correction of the latter is usually the only procedure necessary.

Surgical methods for the correction of atrial septal defects are of two types—closed

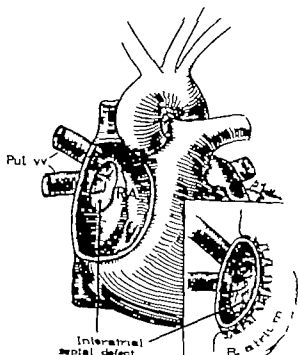


Figure 8 A posterior interatrial septal defect is shown before and after closure by the exclusion method. The anterior right atrial wall is approximated to the anterior edge of the defect which deviates the pulmonary venous return entirely into the left atrium.

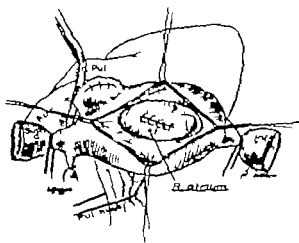


Figure 9 An interatrial septal defect (secundum type) closed by suture under direct vision in a hypothermic patient.

and open. Closed methods have largely been superseded but are sometimes valuable in defects associated with anomalous pulmonary drainage. Figure 8 shows such a method. When this procedure is used, it must be ascertained that no shunt remains

before operation is completed. Atrial defects of the ostium secundum type may be multiple and highly variable in location. Associated anomalies of the pulmonary venous return are common as are hypertrophied valves of the inferior vena cava. By direct visualization of the defects by means of an incision into the right atrium, the defects of the ostium secundum type can be approximated by direct suture (Fig. 9). Very large defects sometimes require patches to avoid undue tension in the closure. When pulmonary venous anomalies are present it may be necessary to use a patch technique to place the pulmonary veins in the left atrium. If pulmonary veins enter the cava itself they must be redirected into the left side, usually by reanastomosis to another pulmonary vein.

Defects of the atrioventricular canal or endocardial cushion defects involve the inferior portion of the interatrial septum and not infrequently the superior portion of the interventricular septum (Fig. 10). There may be associated clefts in the mitral or tricuspid valves. The valvular defects are

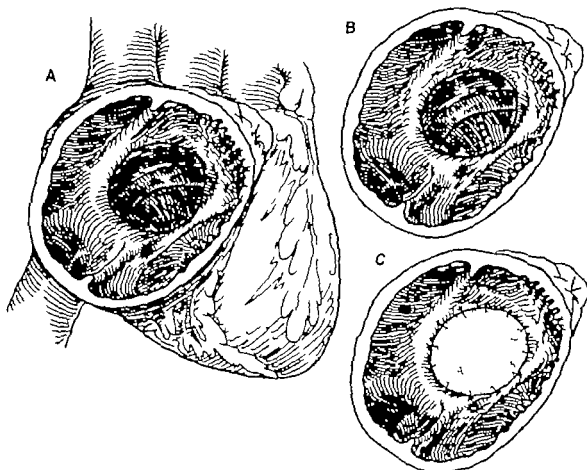


Figure 10 A, Operative view of ostium primum atrial defect (cushion defect) showing a cleft mitral valve seen through the atrial septal defect. B, Closure of mitral cleft by sutures. C, Repair of atrial defect using spongy or plastic cloth.

corrected by approximating the edges of the cleft with fine sutures. A patch must usually be used to close the defect of the atrial septum because of the rigidity of the tissue in the region of the inferior margin of the defect. The repair may have to include approximation of the upper portion of the ventricular septum as well.

Following repair of atrial defects complete closure is the rule. Defects of the secundum variety carry minimal risk. Endocardial cushion defect repair carries increased hazard, but the mortality rate is still low except in the more complex types of atrioventricular canal. Following repair normal hemodynamics are obtained and prognosis is excellent.

INTERVENTRICULAR SEPTAL DEFECTS

Ventricular septal defects may exist alone or in association with other lesions. Such defects may be single or multiple and occupy a position either in the membranous or muscular septum. The high lesions lie immediately beneath that portion of the ventricular septum which joins the aortic root and immediately inferior to the leaflets of the aortic valve. In some cases there is associated aortic insufficiency. When defects of the interventricular septum exist there is considerable variation in the right ventricular pressure, depending upon the size and location of the defect. The lesions can be divided into groups on the basis of the degree of elevation of the pulmonary pressure. In the severe forms the right ventricular and pulmonary pressures approach or exceed the aortic pressure. When the pressures in the right ventricle are only moderately elevated there may be no symptoms or symptoms may develop only later in life. Frequently in the mild forms there is no manifestation of the abnormality except for the murmur and such patients may live to a considerable age without major physical restriction. Patients with moderate to high pulmonary pressures tend to have frequent respiratory difficulty particularly recurrent pulmonary infection and limitation of exercise tolerance. It has been pointed out by Taussig and others that those patients who have only slight elevation of their pulmonary pressure early in life tend to show little change on recatheterization later in life. Those patients who have more severe elevations in pressure early in life tend to have increasing pressures with age.

The principal diagnostic features of the more severe lesions include general underdevelopment, susceptibility to respiratory in-

fections, absence of cyanosis when pulmonary pressure is less than systemic, and symptoms of congestive failure. Physical signs are cardiac enlargement, a harsh systolic murmur which is best heard in the fourth left intercostal space and a loud pulmonary second sound. X-ray examination shows a cardiac silhouette which is globular in shape with enlargement of both ventricles. The pulmonary artery tends to be large and prominent. The lung fields are increased in vascularity. Cardiac catheterization shows an increase in the oxygen tension in the right ventricle and increased pressure in the right ventricle and pulmonary artery. A small gradient may exist between the ventricle and the pulmonary artery without organic obstruction because of the high rate of flow. Nitrous oxide and dye dilution techniques give evidence of a shunt at the ventricular level. Angiocardiography shows refilling of the right ventricle and pulmonary arteries. A filling defect may be present in the right ventricle in some patients. Electrocardiographic changes show biventricular enlargement or right ventricular preponderance.

Interventricular septal defects are corrected under direct vision by open cardiotomy. The right ventricle is incised and the area of the septum is exposed. Cardiac arrest is not necessarily employed. The entire septum must be inspected to be sure multiple defects are not present. Frequently the septal defect lies beneath the leaflet of the tricuspid valve. The papillary muscle may be cut to permit retraction of the tricuspid valve to properly visualize the defect. The bundle of His traverses the medial inferior border of the right side of the defects of the membranous septum. Closure of this portion of the defect is accomplished by placing interrupted sutures in the left side of the septum to avoid the bundle (Fig 11). It may be necessary to use a patch to secure adequate closure without tension. In thinned out areas of the septum the sutures are buttressed by tying them over Teflon felt or cloth. When very high pressures are present in the right ventricle it may be undesirable to completely close the defect primarily. In such instances a fenestrated patch is used to permit readjustment of pressure during the initial postoperative period. The small openings in the patch close over in a period of two to four weeks, allowing for readjustment of dynamics during this interval. A flap valve mechanism can also be employed for this purpose.

Complete heart block has been a compli-

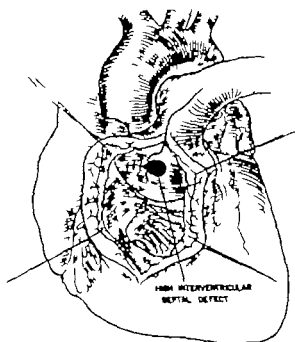


Figure 11 Interventricular septal defect partially closed by sutures under direct vision

cation of the closure of interventricular septal defects and is a serious problem when it occurs. When caused by a suture placed through the bundle of His removal of this suture will usually restore normal rhythm. Following closure of the defect in the membranous septum, an electrode is routinely implanted into the myocardium which will permit the use of an artificial pacemaker if complete heart block occurs. A second electrode is sutured in the chest wall. Both electrodes are insulated, except for the terminal portion, so that stimulation is delivered only to the heart muscle. If asystole or a very slow idioventricular rate supervenes, contraction of the heart may be maintained at an effective and optimal rate by the artificial pacemaker. With increasing experience, the incidence of heart block has markedly decreased. Operation is advised in all patients who show symptoms increasing cardiac size, large shunts or high right ventricular pressures. Operative risk is now low and constantly decreasing, except in patients with very high pulmonary pressures and high pulmonary resistance. Failure to obtain complete closure is not common.

TRANSPOSITION OF THE GREAT VESSELS

Transposition of the great arteries is a common congenital malformation. Relatively few individuals with this anomaly survive to late childhood and a large percentage of such patients die within the first few months

of life. In complete transposition, the aorta rises from the right ventricle and the pulmonary artery from the left ventricle. In order to maintain life, there must necessarily be communication between the two sides of the heart. The usual shunts are maintained through one or more of the following: interatrial septal defects, interventricular septal defects, patent ductus arteriosus or transposition of the pulmonary or systemic veins.

Signs and symptoms of this malformation are usually manifest shortly after birth. Cyanosis, difficulty in respiration, progressive cardiac enlargement and congestive failure are the notable features of this disease. Cyanosis tends to become more intense with increasing age. If oxygenated blood from the pulmonary artery enters the aorta by way of the patent ductus arteriosus, the lower extremities are less cyanotic than the upper. Closure of a patent foramen ovale or patent ductus arteriosus in the neonatal period may produce a sudden deterioration of the patient. The causes of death are cardiac failure and anoxemia.

Physical examination shows increasing heart size beginning shortly after birth. Frequently there is bulging of the precordium. The apex is displaced laterally and there may be systolic or diastolic murmurs which are not characteristic and there may be no murmurs present. Signs of congestive failure may be noted early.

On x ray films the heart is enlarged, although immediately after birth it may be normal in size. The pulmonary conus is small or absent. The shadow at the base of the heart is narrow in the posteroanterior view but in the left anterior oblique position or in the lateral position the shadow is quite wide since the pulmonary artery and the aorta lie over each other. Pulmonary vascular markings are greatly increased. Angiocardiography shows immediate filling of the aorta from the right side, previous to filling of the pulmonary artery and the aorta is anterior in origin.

The electrocardiogram shows right ventricular enlargement, but this is not constant. Cardiac catheterization demonstrates communication of the aorta with the right ventricle.

Complete correction of transposition has not been entirely solved. Partial correction of the defect has given marked benefit in many patients. The most satisfactory technique available with the least risk at the present time would appear to be the transposition of the inferior vena cava to the left

atrium and the anastomosis of the right pulmonary veins to the right atrium. In the second stage the remaining venous return can be transposed and the atrial intercommunication closed. In this way complete transposition of the venous inflow can be accomplished. The right ventricle then functions as the left, serving the systemic circuit. Following the first stage of this procedure improvement has been so striking in many cases that complete correction has been delayed.

Attempts at a one-stage correction of transposition have not yet been successful. Using extracorporeal circulation attempts have been made to transpose the arterial outflow but these have not been satisfactory. A single-stage procedure to bring the caval venous return to the left side and the pulmonary venous return to the right side has been outlined. The interatrial septum can be removed and a patch of saddle-shaped configuration sutured around the orifices of the pulmonary veins and over to the edges of the tricuspid valve. This would bring the pulmonary flow to the right side. Through the remaining orifice of the opened interatrial septum the caval blood could flow to the left side. This is a highly promising procedure which may be successful in the future.

DISEASES OF THE PERICARDIUM

The common diseases of the pericardium may be classified as follows:

- I. Pericardial cyst and diverticulum
- II. Acute pericardial effusion
 - A. Serous or fibrinous
 - B. Hemorrhagic
 - C. Purulent
- III. Chronic pericarditis
 - A. Adhesive pericarditis
 - B. Constrictive pericarditis
- IV. Tumors of pericardium
 - A. Fibroma
 - B. Lipoma
 - C. Mesothelioma
 - D. Sarcoma
 - E. Teratoma

Pericardial cysts are thin walled and contain serous fluid. They are lined by a flattened mesothelium. They are most commonly seen at the pericardiophrenic angles, but may occur at any point in association with the pericardium. A cyst with a direct opening into the pericardium proper is known as a *diverticulum of the pericardium*. The walls of the cyst are fibrous connective tissue and are lined with flattened

mesothelium. Pericardial cysts tend to remain benign. They vary in size from minute structures to cysts which may occupy the major portion of the chest.

Until the cyst becomes extremely large there are no symptoms. Chest pain or pressure may occur. Physical findings may be within normal limits or there may be dullness to percussion and signs of pulmonary compression.

On x ray examination the majority of the pericardial cysts are found to occur on the right side near the junction with the diaphragm. Their borders tend to be rounded and smooth and may change their shape with the position of the patient. When they lie in the region of the aortic arch or root, they are frequently confused with an aneurysm or tumor of the lung.

Therapy consists of excision, even in the absence of symptoms. Surgical treatment is recommended upon the discovery of the lesion because, until exploration is carried out, it cannot be positively identified as a pericardial cyst. No lesion in this position can be considered entirely benign without biopsy since other lesions in this area are malignant. Danger of infection or continued pressure remains until removal is effected.

Acute serous or fibrinous pericardial effusion may be associated with rheumatic or viral agents. It may also be of metabolic and nonsuppurative bacterial origin. Signs and symptoms may be absent but usually include fever, tachycardia, pain and shortness of breath. The symptoms may be confused with those due to pneumonia or acute abdominal states. Pain tends to be periodic, sharp and stabbing in character or pleuritic. As larger amounts of fluid accumulate the pain may disappear whereas if the effusion remains predominantly fibrinous, the pain may persist for considerable periods with remissions and exacerbations. The rapid accumulation of large amounts of fluid may lead to cardiac tamponade (Fig. 12).

Cardiac tamponade gives rise to the signs of acute cardiac compression which are a quiet heart, elevation of the venous pressure and decreasing arterial pressure. The heart sounds are distant. Signs of peripheral vascular collapse and a paradoxical pulse may be manifest. The heart shadow is normal or enlarged on fluoroscopy and shows less than a normal amount of pulsation.

Tamponade is seen frequently in association with penetrating injuries of the heart and accompanies the acute accumulation of blood in the pericardium. Decompression of

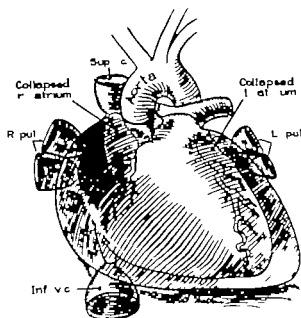


Figure 12. Acute cardiac tamponade showing compression of venae cavae and pulmonary veins. The compression also interferes with diastolic filling of the ventricles.

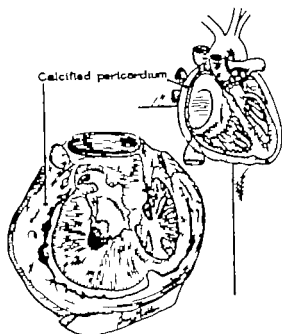


Figure 13. Constrictive pericarditis. The heart and venae cavae are surrounded by dense, thick, fibrotic and calcific pericardium. The ventricular musculature is invaded by fibrous tissue and calcium deposition.

the pericardium is a lifesaving measure. This may be accomplished by needle aspiration or surgical drainage. Aspiration is performed by the paraxiphoid or the parasternal route. Pericardial aspiration always carries the risk of further laceration of the coronary artery or the atrium and further intrapericardial bleeding. The use of intravenous fluids may give clinical improvement in acute cardiac compression and maintain life until pericardial aspiration or drainage can be performed.

Surgical decompression of the pericardium may be accomplished either through a small anterior thoracotomy incision made just to the left of the sternum or through a paraxiphoid extrapleural approach. Incision of the pericardium, with excision of a small window permits external drainage or excision of a portion of the pericardium in its intrapleural portion and institutes drainage into the pleural space.

Conservative aspiration therapy is frequently successful and is particularly useful in serous pericarditis. In tamponade due to trauma, aspiration combined with the use of intravenous fluids and blood replacement will often be associated with spontaneous cessation of the bleeding. If after two attempts at aspiration, the signs of cardiac compression recur or if there is serious bleeding into the pleural space with excessive blood loss, immediate operation should be carried out. The rapid replacement of blood loss by pressure transfusion is ne-

sary in some subjects to gain sufficient time to get the patient to the operating room.

If continued cardiac compression occurs from bleeding, the heart is approached through the anterior fourth intercostal space on the left. The pericardium is opened and the blood removed. The wound in the heart is controlled by pressure and then closed with multiple silk sutures. Excessive delay by continued pericardial aspirations in the patient who shows signs of rapid recurrence of cardiac compression after the first pericardial tap is dangerous. Failure to evacuate all of the blood from the pericardium and to keep the pericardium dry in the post-traumatic period frequently gives rise to signs of chronic cardiac compression. This must be relieved by pericardiectomy at a later time.

Chronic compression is due to dense fibrosis of the pericardium, which prevents adequate venous filling of the heart by constriction of the venous pathways or the ventricular chambers (Fig. 13). The most common cause of constrictive pericarditis is tuberculosis but this affection also is seen after hemopericardium. Chronic cardiac compression is characterized by a quiet heart which, though often normal in size, may be enlarged and by elevated venous pressure and enlargement of the liver associated with ascites. Fluoroscopy shows a heart shadow with little movement or dimin-

ished pulsation. Kymograms confirm the decreased cardiac excursion. Extensive calcification may or may not be present in the pericardium. The presence of calcium in the pericardium is not diagnostic of constriction.

Dyspnea, venous distention, cyanosis, pain in the right upper quadrant and swelling of the abdomen are common symptoms. Heart sounds are distant to normal. Murmurs may be present. A pericardial knock is frequent. The signs of abdominal and pleural fluid and ankle edema are common. The liver is enlarged. Liver function may be markedly impaired, venous pressures are elevated. Cardiac catheterization shows relatively characteristic pulse tracings.

Complete resection of the anterior pericardium with freeing of the superior and inferior cavae, the pulmonary veins, the diaphragmatic surface of the heart and both ventricles can be done in the majority of cases as a single procedure. This may be performed through a midline sternum splitting incision or a transverse bilateral thoracotomy incision. In more severe cases, it may be necessary to do the operation in two stages. The pericardium is densely adherent to the myocardium and calcification may extend through the pericardium into the muscle of the heart itself. The removal of the pericardium always proceeds from the apex to the left ventricle, freeing the entire left ventricle, left atrium, then the right ventricle, right atrium and finally to the cavae. If both right and left ventricles should be badly compressed and one relieves only the right ventricular constriction, fatal acute pulmonary edema may occur.

A high index of suspicion is necessary to make the diagnosis of chronic cardiac compression, which is frequently confused with renal disease or primary liver disease. Swelling of the abdomen and the scrotum with relatively little ankle edema, congestive failure without an adequate explanation, such as valvular disease or coronary artery disease, should make one suspect constrictive pericarditis.

Pericardial biopsy should be performed in all patients in whom there is suspected tuberculous pericarditis or acute pericarditis of unknown origin, in order to establish the cause. If at the time of biopsy there is already evidence of thickening of the pericardium, pericardiectomy can be carried out at this time. If the lesion is tuberculous, specific treatment with streptomycin and other antituberculous drugs is indicated.

Pericardiectomy carries a relatively low

risk. Complete relief of symptoms with disappearance of ascites and return of adequate cardiac function is to be expected. When the constriction has been present for a long time before operation, an interval of three to six months may elapse before the patient manifests his maximal improvement. If pericardiectomy is carried out in the earlier phases of the disease and is adequate in its extent, an immediate return of normal pressures will result.

Suppurative pericarditis is associated with staphylococci, pneumococci, actinomycotic and mixed tuberculous infections.

The symptoms are those of acute pericarditis associated with fever, sweating, shock and sometimes cardiac tamponade. Physical signs are fever, rapid pulse, low systolic and high diastolic pressure and congestive failure. The area of cardiac dullness may be increased and a friction rub may be transiently present. Paradoxical pulse and distention of the neck veins with increased venous pressure are noted if tamponade is present.

X-ray examination usually shows an enlarged cardiac shadow characteristic of fluid in the pericardium. Pericardial aspiration will reveal the presence of purulent exudate which should be cultured aerobically, anaerobically and on media for tuberculosis and fungi.

Purulent pericarditis may be treated by repeated needle aspiration and instillation of the proper antibiotic both locally and systemically. Open drainage of the pericardium is preferable. A window is excised from the pericardium after extrapleural exposure of the pericardium. A Penrose drain is left in the pericardial sac for irrigation with saline and antibiotic solutions. The proper antibiotics are also given systemically.

MITRAL STENOSIS

Mitral stenosis has long been recognized as being primarily associated with rheumatic fever, although it also exists in the congenital form. It is most common in young adults and females are affected more often than males. From the time of the initial attack of rheumatic fever until the development of symptoms of mitral stenosis, there is frequently a lapse of many years. Approximately 50 per cent of the patients who have outspoken mitral stenosis give no history of having had previous rheumatic fever in the classical form. The course of the disease tends to be slowly and steadily progressive, beginning with dyspnea on strenuous exer-

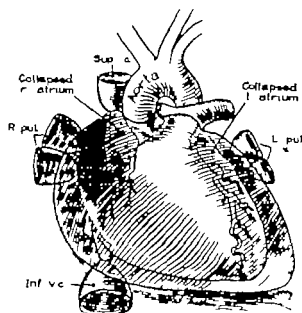


Figure 12. Acute cardiac tamponade showing compression of venae cavae and pulmonary veins. The compression also interferes with diastolic filling of the ventricles.

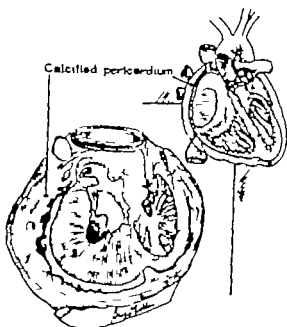


Figure 13. Constrictive pericarditis. The heart and venae cavae are surrounded by dense, thick, fibrotic and calcific pericardium. The ventricular musculature is invaded by fibrous tissue and calcium deposition.

the pericardium is a lifesaving measure. This may be accomplished by needle aspiration or surgical drainage. Aspiration is performed by the paraxiphoid or the parasternal route. Pericardial aspiration always carries the risk of further laceration of the coronary artery or the atrium and further intrapericardial bleeding. The use of intravenous fluids may give clinical improvement in acute cardiac compression and maintain life until pericardial aspiration or drainage can be performed.

Surgical decompression of the pericardium may be accomplished either through a small anterior thoracotomy incision made just to the left of the sternum or through a paraxiphoid extrapleural approach. Incision of the pericardium, with excision of a small window permits external drainage or excision of a portion of the pericardium in its intrapleural portion and institutes drainage into the pleural space.

Conservative aspiration therapy is frequently successful and is particularly useful in serous pericarditis. In tamponade due to trauma, aspiration combined with the use of intravenous fluids and blood replacement will often be associated with spontaneous cessation of the bleeding. If after two attempts at aspiration, the signs of cardiac compression recur or if there is serious bleeding into the pleural space with excessive blood loss, immediate operation should be carried out. The rapid replacement of blood loss by pressure transfusion is neces-

sary in some subjects to gain sufficient time to get the patient to the operating room.

If continued cardiac compression occurs from bleeding, the heart is approached through the anterior fourth intercostal space on the left. The pericardium is opened and the blood removed. The wound in the heart is controlled by pressure and then closed with multiple silk sutures. Excessive delay by continued pericardial aspirations in the patient who shows signs of rapid recurrence of cardiac compression after the first pericardial tap is dangerous. Failure to evacuate all of the blood from the pericardium and to keep the pericardium dry in the post-traumatic period frequently gives rise to signs of chronic cardiac compression. This must be relieved by pericardiectomy at a later time.

Chronic compression is due to dense fibrosis of the pericardium, which prevents adequate venous filling of the heart by constriction of the venous pathways or the ventricular chambers (Fig. 13). The most common cause of constrictive pericarditis is tuberculosis but this affection also is seen after hemopericardium. Chronic cardiac compression is characterized by a quiet heart which, though often normal in size, may be enlarged and by elevated venous pressure and enlargement of the liver associated with ascites. Fluoroscopy shows a heart shadow with little movement or dimin-

normal sinus rhythm should suggest immediate evaluation for operative intervention. Progression of symptoms with increasing dyspnea on exertion and the development of peripheral embolism, major hemoptysis or signs of pulmonary congestion are indications for operation. The presence of acute rheumatic fever, subacute bacterial endocarditis or other diseases with a more unfavorable prognosis than mitral stenosis is a contraindication to operation. Patients with multivalvular disease should be evaluated in relation to the possibility of correcting all valvular lesions simultaneously if possible, or in stages if necessary.

The mitral valve is usually most accessible through the left side of the chest and the

left atrial appendage is used as the site through which the index finger can be introduced into the left atrium to palpate the valve. The valve is then opened along the lines of the commissures either by fracture of the valve with the finger or with a knife which is advanced along the finger to engage the commissure and to incise it. The chordae tendineae below the cut edges of the valve are separated so that the valve leaflets can move freely (Fig. 15). The valve orifice, which is usually smaller than the tip of the index finger, is opened through a width of two to two and one-half fingers. The appendage is amputated to prevent the formation of thrombus. Any clot which may be present in the atrial appendage is re-

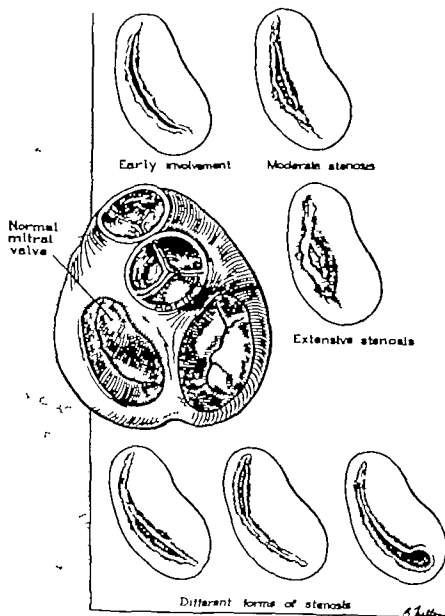


Figure 14. Mitral stenosis. The relation of the normal valves is illustrated. The progressive fusion of the mitral leaflets is shown. The lower figure depicts stenosis with a posterior orifice, an anterior orifice and stenosis combined with a teardrop type of mitral insufficiency.

tion and excessive fatigue gradually increasing to dyspnea on moderate to minimal exertion. Because of the slow and insidious progress of the disease the patient is frequently not fully aware of how limited his activities have become until he is no longer able to carry on his occupation.

Episodes of palpitation pulmonary edema, paroxysmal nocturnal dyspnea and hemoptysis are common symptoms. Liver enlargement and congestion are associated with right upper quadrant discomfort. The occurrence of atrial fibrillation is frequent. In the later stages of the disease, peripheral edema becomes manifest.

The mitral valve is composed of a major anteromedial and a smaller posterolateral leaflet. The valvulitis produced by rheumatic fever tends progressively to narrow the mitral orifice. The sealing together of the two leaflets in a fibrous union begins by the formation of fibrinous adhesions which bridge the commissures at the edges of the leaflets. As vascularization and organization progress, the orifice is gradually narrowed. The area immediately adjacent to the valve orifice is thickened and calcified. The edges of the chordae tendineae also become fused as well as thickened and shortened. In spite of these changes the peripheral zone at the valvular annulus tends to remain quite flexible and it is this factor which makes it possible to restore normal valvular function by operation even though the valve may be stiff, thickened and even calcified toward its central portion. When this hingelike action is preserved peripherally an incision in the commissural area will permit the valve to open widely and to close normally. Incisions at points other than the commissures into either leaflet produce mitral insufficiency of varying degrees. Cutting or fracturing the anterior leaflet produces larger amounts of insufficiency than do lesions of the posterior leaflet because the anterior or aortic leaflet guards the outlet tract of the left ventricle (Fig. 14).

The normal mitral orifice is approximately 4 to 6 sq. cm. in area. When the total area available for flow has been narrowed to less than 2.5 sq. cm., the patient notes progressive symptoms. When it approaches 1 sq. cm., the patient's exercise tolerance is markedly decreased.

Physical examination shows signs which are frequently limited to the heart and lungs. The heart is not greatly enlarged unless other complicating valvular lesions are present. The enlargement of the heart is

predominantly of the right ventricle and left atrium. This may be manifest either as slight enlargement to the right or left side of the chest. As the right ventricle enlarges, it rotates toward the left, moving the apex in that direction. The left atrium enlarges posteriorly and toward the right.

The murmur of mitral stenosis may be extremely difficult to hear. It is characteristically an apical diastolic rumbling murmur with presystolic accentuation. A diastolic thrill sometimes occurs. The first sound is loud and snapping. An opening snap is also audible. If atrial fibrillation is present, presystolic accentuation may not be noted. Systolic murmurs of moderate intensity may also be present even though the lesion may be pure mitral stenosis. In some patients the murmur of mitral stenosis is heard only in the left lateral position. The murmur is accentuated by coughing. The pulmonary second sound is loud. The murmur of pulmonary insufficiency may be present because of dilatation of the pulmonary artery.

The cardiac silhouette may be normal or slightly enlarged. Oblique views will demonstrate enlargement of the right ventricle and left atrium. The left atrial shadow may be prominent on the right border of the heart which shows a double contour. The pulmonary conus is full. The pulmonary markings may be prominent and show pulmonary congestion. Barium swallow usually shows displacement of the esophagus from enlargement of the left atrium.

Cardiac catheterization may show increased pulmonary arterial pressures and high pulmonary capillary pressures. Cardiac output is fixed at relatively low levels. Exercise shows either no increase or a fall in cardiac output since when the heart rate increases the time consumed in systole increases at the expense of diastolic filling time per minute. At a given pressure, the amount of blood that will flow through an opening of fixed size is dependent upon the time of diastole.

The electrocardiogram may show no characteristic changes or only prominent P waves. It may show atrial fibrillation and usually will show some signs of right ventricular hypertrophy.

Patients who have a diagnosis of mitral stenosis should be evaluated to determine whether operation will be beneficial. The presence of previous congestive heart failure has not been a contraindication to operation. The development of atrial fibrillation in a patient who has previously had a

be greatly improved but will have some limitation in extreme exercise and the remaining 2 per cent will show little or no improvement. In this latter group technical difficulties such as contraction of the entire valve annulus the presence of an extremely stiff valve which fails to function properly after being opened, the production of serious mitral insufficiency and the inability to secure adequate separation of the chordae are the principal causes of failure. In the terminal group in which there is failure which is not controllable by medical management, operative mortality is approximately 25 per cent and in this group the over-all good results fall sharply although many patients may obtain considerable improvement even though the ability of the myocardium to regain a normal state appears to be somewhat limited. The operative results may also be compromised by pulmonary changes which are no longer reversible, but this is rare.

MITRAL INSUFFICIENCY

It was formerly taught that some degree of mitral insufficiency was an almost constant accompaniment of mitral stenosis. Exploration of the living heart, however has shown that this is not necessarily correct. Mitral insufficiency produces a decreased cardiac output and increases the work of the left ventricle since a portion of the blood which would be normally ejected through the aorta is lost by backward flow through the incompetent mitral valve. This leads to increased pressure in the left atrium similar to that which occurs in mitral stenosis. Small degrees of mitral insufficiency appear to be consistent with a long life expectancy. Mitral insufficiency may be primary or secondary to another disease which produces major left ventricular enlargement, such as aortic insufficiency.

The symptoms are similar to those of mitral stenosis. Bacterial endocarditis is a more common complication of mitral insufficiency than of stenosis. Atrial fibrillation is frequent.

The physical signs associated with mitral insufficiency are a systolic murmur at the apex which may be accompanied by a thrill. There is cardiac ventricular enlargement and a left ventricular lift. The murmur radiates to the axilla and to the back. The pulmonary second sound is loud. Pulmonic insufficiency may be present.

The x ray shows left ventricular and left atrial enlargement. The left atrium may be gigantic in size and extends far to the right. The pulmonary artery is prominent and there may be pulmonary congestion.

Left heart catheterization may show high left atrial pressures with a large V wave. Regurgitation may be demonstrated by injection of contrast medium into the left ventricle.

Acute dilatation of the mitral annulus may be also associated with acute rheumatic myocarditis and dilatation of the left ventricle. Insufficiency is also caused by deformity of the valve cusps or in the presence of relatively normal valve cusps by extreme shortening of the chordae tendineae. Mitral stenosis and insufficiency may coexist. This is characteristically seen in patients in whom there is fusion of one commissure associated with a major deformity of the aortic cusp of the mitral valve in the area of the other commissure. Insufficiency may be particularly severe in this instance if the chordae are shortened.

Many methods for the correction of mitral insufficiency have been devised. Open operation under direct vision without interruption of the cardiac beat is the procedure of choice. The details of the procedure must be altered to suit the particular circumstances which exist in the individual pa-

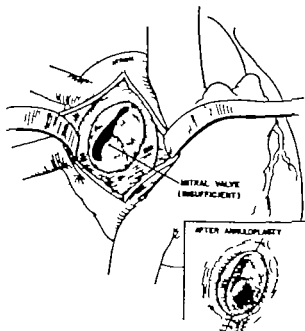


Figure 16. Mitral insufficiency with dilated annulus. Valve made competent by sutures in annulus.

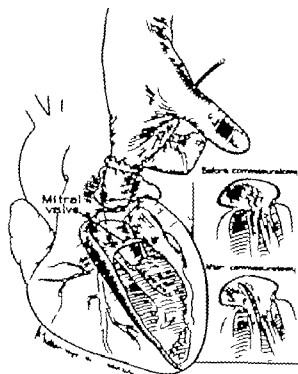


Figure 15 Mitral stenosis. The anterior commissure is being incised by the knife held on the index finger. The insets show the stenotic valve with fusion of the chordae before operation and the separation of the leaflets and chordae.

moved prior to insertion of the finger. It is sometimes necessary to enter the heart through one of the pulmonary veins in order to avoid a clot which cannot be removed from the atrial appendage itself.

In patients in whom difficulty is experienced in opening the valve satisfactorily a combined approach may be used. The index finger is inserted in the atrium in the usual way. A dilating instrument is then introduced into the ventricle and guided from above into the mitral orifice by the intracardiac finger. The instrument permits forcible dilatation of the valve orifice under guidance of the finger.

A right-sided approach is desirable in some patients. After a right thoracotomy the groove between the superior vena cava and the right pulmonary vein is dissected. A finger is introduced into the left atrium just behind the superior vena cava. Since in patients with mitral stenosis the left atrium tends to enlarge far to the right, this gives a good route of access to the mitral orifice.

For these patients with combined lesions of mitral insufficiency and mitral stenosis and those who have had previous operations for mitral stenosis which have not been successful, a direct operation using the heart

lung apparatus is now advocated. The approach through the right thorax is highly satisfactory; the left atrium can be entered just anterior to the pulmonary veins or through the interatrial septum. Incision of the commissures and separation of the chordae can then be carried out under direct vision. The mitral insufficiency which is present can be corrected.

The postoperative complications associated with mitral stenosis can be divided into those which are primarily associated with surgery of the mitral valve and those which are associated with a major thoracotomy and an operation upon the heart in the presence of cardiac disease. Complications most commonly encountered primarily related to the operation for mitral stenosis are dislodgment of clots from the left atrium with peripheral or cerebral embolization, inadvertent production of excessive mitral insufficiency with cardiac failure and reactivation of the rheumatic process common to all operations for rheumatic heart disease.

Certain complications associated with cardiac and thoracic procedures in general are: hemorrhage, cardiac arrhythmias, atelectasis associated with either retention of bronchial secretions or aspiration, bacterial endocarditis in procedures in which a valve, major artery or myocardium has been incised, pneumothorax due to laceration of the lung during operation or to rupture of an emphysematous bleb, electrolyte imbalance, pleural effusion, acute pulmonary edema, pericardial effusion and tamponade, pulmonary embolism, and renal complications, particularly lower nephron syndrome with oliguria or anuria usually associated with transfusion reaction and hypotension.

The best results are obtained in patients who have pure mitral stenosis as an isolated lesion and who are under fifty years of age at the time of operation. Patients in the older age groups are not excluded from operation but should be very carefully screened. If there has not been prolonged intractable congestive failure and if there is no arrhythmia other than atrial fibrillation, the over all mortality rate from operation is less than 4 per cent. Fatalities during or following operation result from technical difficulties, such as hemorrhage during operation, embolic phenomena or from pulmonary complications. Of the surviving patients, approximately 80 per cent will have an excellent result and will be able to return to normal activities. Another 15 per cent will

the cusps similar to that in pulmonary stenosis. Aortic insufficiency and stenosis may coexist although free aortic insufficiency is not common in the presence of severe degrees of aortic stenosis.

Subaortic stenosis is of congenital origin and may be manifest anatomically by a fibrous diaphragm or muscular ridge at a variable distance below the aortic valve. Generalized hypertrophy of the left ventricular musculature in the subvalvular region has also been described as a cause of obstruction to the outlet tract of the ventricle. This appears to be similar in type to that seen in the right ventricle, proximal to the pulmonary valve.

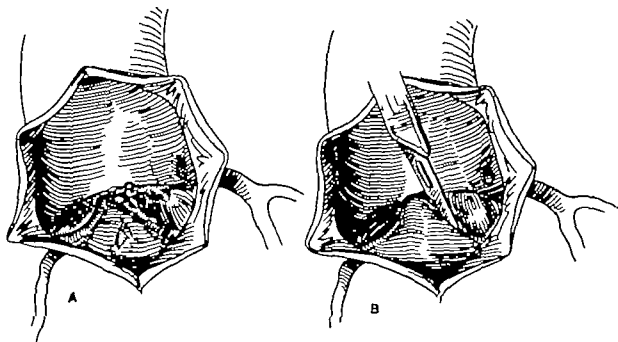
In rheumatic aortic valvulitis fusion of the commissures begins peripherally and extends centrally gradually narrowing the orifice (Fig. 18). Calcification is a common accompaniment of the process even in its earlier phases before it has produced severe symptoms. Calcification of the entire aortic ring in the region of the aortic valve may also occur early in the process. Fusion of the valve cusps occurs by a similar process to that of other valvular fusions that is, with edema, inflammatory exudate, fibrin deposits, gradual replacement and organization of fibrous tissue. This later becomes calcified, but since, unlike mitral stenosis, the periphery tends to become calcific and stiff early in the process, the restoration of flexibility to the valves by commissural division

is less often as satisfactory as in mitral stenosis. In some cases calcification does not occur or if present, occurs only in small amounts and late in the disease. This presents a more suitable situation for operation. Fusion of the commissures is not always symmetrical (Fig. 19A).

The earlier phases of aortic stenosis may be asymptomatic. In this stage there is adequate compensation for the moderate degrees of obstruction by left ventricular hypertrophy. However with continuing cardiac enlargement, conduction defects may develop with the production of arrhythmias. Ease of fatigue, anginal pain, episodes of fainting, dyspnea on exertion and congestive failure are the usual symptoms. Angina pectoris, syncope or congestive failure is an extremely serious prognostic sign. Patients may die in an episode of syncope, in a Stokes Adams attack or in ventricular fibrillation.

The murmur of aortic stenosis is harsh in character and is usually heard best over the second right intercostal space or the third left intercostal space. It is frequently accompanied by a thrill which is transmitted into the neck. The aortic second sound is decreased or absent. There may be murmurs of associated valvular disease. Cardiac enlargement is present. The blood pressure may be normal or the pulse pressure may be diminished.

X rays of the thorax show the cardiac sil



Figures 19 A, Acquired aortic stenosis with one commissure open. View as seen at aortotomy B, Congenital aortic stenosis. Note the preservation of the remnants of the commissures.

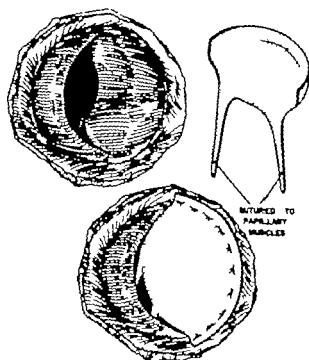


Figure 17 Mitral insufficiency with destruction of a leaflet. Artificial leaflet with chordae which are sutured to ventricle.

tient. When there is generalized dilatation of the annulus insertion of sutures at selected intervals can narrow the annulus with out impinging upon the leaflets of the valve to restore competency of the valve (Fig. 18) In some cases the sutures are preferably placed in the annulus in the region of the commissures and in others these areas should be avoided with the sutures being placed opposite each other at intervals over the circumference of the annulus. In some cases it is necessary to lengthen or shorten chordae tendineae or to place mattress sutures at particular points within the annulus to secure proper approximation. In the presence of extreme calcification or deficiency of leaflet substance the addition of substance may be necessary. Mobilization of the leaflets may be difficult and a leaflet or entire valve may be replaced with silicone rubber or Dacron leaflets (Fig. 17)

This lesion which has been difficult to attack is now becoming one of the standard procedures in which good results are to be anticipated. As in all lesions in which there is major enlargement of the left ventricle, operation is best done before the patient has maximal left ventricular enlargement. Operation should be advised when the patient begins to have symptoms or shows signs of progression of the lesion.

Results of operation are good in patients

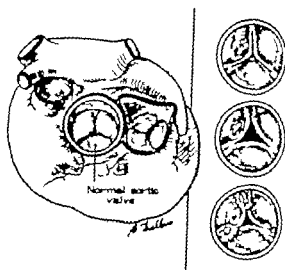


Figure 18 Aortic stenosis. The gradual narrowing of the aortic outflow tract is depicted with the associated thickening and calcification of the cusps at the periphery

who do not have irreversible changes in the left ventricle. Risk is high in patients who have congestive failure which is uncontrollable or who have valves which cannot be satisfactorily mobilized at operation.

AORTIC STENOSIS

Aortic stenosis may be congenital, arteriosclerotic, rheumatic or postendocarditic. Brucellosis has also been suspected as an etiologic agent. The majority of such cases are probably rheumatic in origin and the exact role of arteriosclerosis in aortic stenosis is not entirely clear

Certain anatomic features of the aortic valve are important to a clear understanding of its pathologic lesions. The root of the aorta is continuous with the anterior aortic leaflet of the mitral valve. The three cusps attach at their inferior margins to the myocardium of the left ventricle. The coronary arteries arise from the right and left aortic sinuses respectively. The noncoronary sinus lies posteriorly and to the right. The left coronary artery passes between the aorta and the pulmonary artery into the atrioventricular groove, at which point it divides into the anterior descending and circumflex branches. Since the aortic root lies deeply behind the pulmonary artery and has its origin in the myocardium with the ventricle the annulus of the valve is concealed anteriorly by the outflow tract of the right ventricle.

Congenital valvular aortic stenosis is associated with fusion of the commissures of

stance to one or more leaflets because of associated aortic insufficiency. To avoid the production of aortic insufficiency by operation in pure lesions, it is essential that the division of the commissures be accurately accomplished. When aortic and mitral disease coexist, both lesions are corrected at a single procedure. The results of open operation have been increasingly satisfactory as more experience has been gained.

Some surgeons have continued to use the closed approach to aortic stenosis, either from above through the aortic root, or from below through the ventricle (Fig. 20). Finger fracture combined with dilatation from above has been satisfactory in many instances. Advocates of the transventricular approach believe that satisfactory openings can be obtained by insertion of a dilator through the ventricle and the risk is less than in the other approaches.

It would appear that open operation offers great advantages, particularly in allowing plastic procedures upon the valve itself or total valvular replacement when indicated.

With the advances in the methods for increasing the mobility of the valve and reconstruction of the leaflets by open technique, risk of operation has greatly decreased. Those patients with acquired aortic stenosis who are not in terminal stages have an operative risk of approximately 10 per cent. Patients with severe conduction changes and uncontrollable heart failure have high mortality rates with operation. Operation should be advised when the patient first shows signs of progression of the lesion, for this is already late in the disease.

AORTIC REGURGITATION

Aortic insufficiency is the most treacherous of the acquired valvular lesions. Its causes are (1) congenital, (2) rheumatic, (3) syphilitic, (4) traumatic, (5) subacute bacterial endocarditis, (6) acute bacterial endocarditis, (7) dissecting aneurysm, (8) aneurysm of sinus of Valsalva and (9) cystic medial necrosis without dissecting aneurysm. The onset of serious symptoms may be rapid after production of an acute form of this lesion or may occur only after many years in the chronic forms. In the latter type symptoms may be manifest early after severe myocardial damage has occurred.

Free aortic insufficiency is usually associated with ease of fatigue, dyspnea, paroxysmal nocturnal dyspnea, edema and angina pectoris. In its early stages no symptoms may be present. Sudden death due to ar-

rhythmias is common and in those patients with active rheumatic fever there may be severe myocardial damage with impairment of cardiac contraction.

The heart is usually greatly enlarged by the time symptoms are evident. The enlargement is due both to hypertrophy and dilatation of the left ventricle. The early blowing diastolic murmur is usually best heard along the left sternal border and in the second right intercostal space. It may be associated with a systolic murmur even though the anatomic amount of stenosis may be quite insignificant. A ventricular diastolic gallop rhythm is an early sign of failure. A systolic murmur at the apex due to dilatation of the left ventricle is common. A diastolic rumbling murmur at the apex is also frequently associated and represents the Austin Flint murmur of aortic insufficiency in the absence of mitral stenosis. The blood pressure shows elevation of the systolic pressure and a decrease in the diastolic pressure. The pulse is water hammer in type. The abdominal aorta and the vessels in the neck are frequently violently pulsatile.

X-ray examination of the chest shows the characteristically boot-shaped heart when extreme left ventricular enlargement with dilatation and hypertrophy is present. The aorta shows varying degrees of dilatation and, on fluoroscopy marked pulsation with rapid expansion in systole and collapse in diastole ("aortic rock") is noted.

The electrocardiogram shows left ventricular enlargement and/or strain. There may be abnormalities of ventricular or atrial conduction. Varying degrees of heart block may be present, as well as signs of myocardial ischemia.

Once the patient with free aortic insufficiency with a diastolic pressure of less than 50 has begun to show signs of congestive failure the average prognosis is for two years or less of life. If the aortic insufficiency has been rapidly produced by rupture of a cusp or trauma, prognosis is for an even shorter time.

Since the treatment of aortic insufficiency is still in its early phases, the first patient having been treated less than seven years ago the criteria for operation are changing. Patients should be selected who have evidence of free aortic insufficiency with marked lowering of their diastolic pressure and major cardiac enlargement, and who are beginning to have early signs of progression of their lesion. The majority of patients surgically treated to date have had Austin Flint

bouette with evidence of left ventricular enlargement. This enlargement is usually not as great in pure aortic stenosis as it is in aortic insufficiency or in combined aortic stenosis and insufficiency. In valvular aortic stenosis of either the acquired or congenital type there may be some poststenotic dilatation of the aortic root visible on the right cardiac border. Calcification of the aortic valve can frequently be demonstrated in acquired aortic stenosis by fluoroscopy.

Catheterization of the left heart by the transbronchial left atrial or left ventricular percutaneous route shows a pressure gradient between the left ventricle and the aorta and elevation of the left ventricular pressure. In the presence of extreme failure the left ventricular systolic pressure may be lower than expected and this level may be much lower than it had been prior to the onset of failure. Direct aortic blood pressure tracings show characteristic contours in the presence of serious degrees of stenosis. The critical orifice size for the aortic valve would appear to be 0.7 sq. cm.

Patients most suitable for operation are those who show onset of symptoms such as syncope, angina, progressive fatigue, dyspnea or evidence of myocardial failure which responds to intensive medical treatment. Patients who are in the terminal phases of the disease with advanced failure which cannot be controlled medically have an extremely serious prognosis with or without operation and should be offered operation only with the understanding that it carries an extremely high risk. Surgical risk is least when the patient is treated at a time when he is showing progression of his lesion with good medical therapy but while he still can be adequately controlled.

Congenital aortic valvular stenosis is the most amenable of the aortic valvular lesions to surgical approach. With inflow occlusion and total body perfusion the aortic valve may be approached either through the right thorax or by midline sternotomy.

Under total body perfusion the aorta is clamped just proximal to the innominate artery and aortotomy is performed just above the valve. The rudimentary lines of the commissures can be identified and the valve opened to essentially normal size after the heart has been filled with blood and the coronary circulation resumed (Fig. 19B). If the incisions are properly made, no insufficiency results and the gradient across the valve is abolished. The risk of this procedure has been extremely low and the results ex-

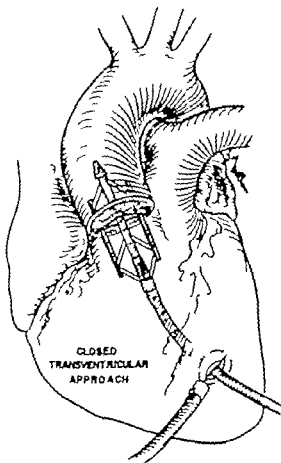


Figure 20 Closed approach to aortic valve by means of a dilator introduced into the left ventricle.

cellent. The procedure can also be accomplished under hypothermia, but the time safely available under these conditions is short and is less satisfactory. Cardiac arrest by drugs, anoxia or cold may be used, but operation is usually accomplished without these adjuncts.

Subaortic stenosis is treated satisfactorily by an approach to the obstruction through the aortic root. With the patient on cardiopulmonary by pass the aorta is opened, the normal valve is retracted and the obstruction in the ventricle is visualized and removed by excision. In some cases the area of obstruction may be a considerable distance below the aortic valve, but it can be satisfactorily visualized.

Acquired aortic stenosis offers difficulties in the mobilization of the valve when extensive calcification of the valve is present (Fig. 19A). The direct approach through aortotomy as in congenital aortic stenosis, has permitted reconstruction of the valve by incision along the commissural lines and removal of the calcific deposits in the base of the aorta to satisfactorily mobilize the valve. In some cases it is necessary to add sub-

incident lesions such as coarctation are corrected at the operation for aortic insufficiency.

The present surgical correction of aortic insufficiency has followed three general methods: insertion of a rigid prosthetic aortic valve into the distal portion of the ascending arch; reconstruction of the valve and/or aortic root; and insertion of cusp valves to replace one or more leaflets. The ball valve prosthesis has been widely used. It is inserted into the descending aortic arch just distal to the left subclavian artery. This prosthesis is composed of a Plexiglas chamber and a silicone rubber ball. The valve is maintained in position by "multiple point fixation" which utilizes Nylon rings to exert pressure at many individual points around the circumference of the aorta (Fig. 21). The procedure is done through a left thoracotomy incision which gives easy access to the correction of other lesions such as mitral stenosis and coarctation. The mortality rate following this procedure has diminished and at present approximates 5 per cent. Complications following this procedure are uncommon, but embolization and aneurysm formation have been reported. A modification of the ball valve for insertion in the subcoronary artery position has been devised.

Direct procedures upon the aortic valve and the aortic root have been developed to meet the particular anatomic causes of aortic valve leakage. No single procedure is applicable to all types of aortic insufficiency. These procedures utilize extracorporeal circulation to obtain a bloodless aortic root. Midline sternotomy is the usual approach. Before the ascending aorta is opened the chest is filled with carbon dioxide to minimize the danger of trapping air in the heart

with subsequent embolization. After the aorta has been opened the valve is inspected to determine the type of deformity which may be present in the valve leaflets. When the aortic annulus is dilated and the cusps are minimally deformed removal of the non-coronary cusp and suture of the commissures of the remaining cusps will totally correct the aortic leak (Fig. 22). Similarly if the noncoronary cusp is markedly deformed and the other cusps are intact, removal of the noncoronary cusp will be curative. In most cases in which the commissures are separated but the leaflets are still mobile approximation of the commissures by suture produces good results.

When serious deformity of a single coronary leaflet is present, this leaflet can be replaced with a self-sealing silicone rubber leaflet (Fig. 23). In still other instances, substance may be lost from one or more cusps and this may be replaced with compressed Ivalon or silicone rubber with an Ivalon base without replacement of the total cusp (Fig. 24). Total valve replacement with three self-sealing cusps incorporated into a single unit has been accomplished experimentally and modifications of this technique will soon be used clinically (Fig. 25).

Perforation of a cusp or rupture of a cusp can be repaired under direct vision either by direct suture or by use of a cloth patch.

Aneurysms of the sinus of Valsalva are treated by excision and reconstruction of the aortic wall at the origin of the aneurysm. Ruptured aneurysm of the sinus is treated similarly with closure of the opening into the atrium or ventricle. When dissecting aneurysm is the cause of the aortic insufficiency closure of the laceration of the inner layers of the wall can be accomplished,

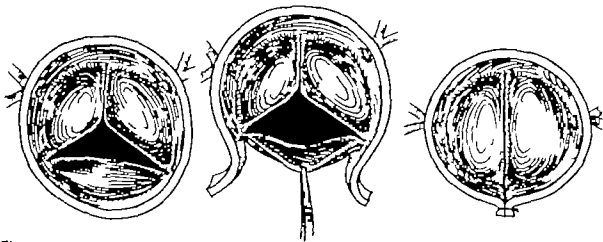


Figure 22. Aortic insufficiency with deformity of noncoronary cusp. Excision of noncoronary cusp has been begun in middle figure and completed at the left.

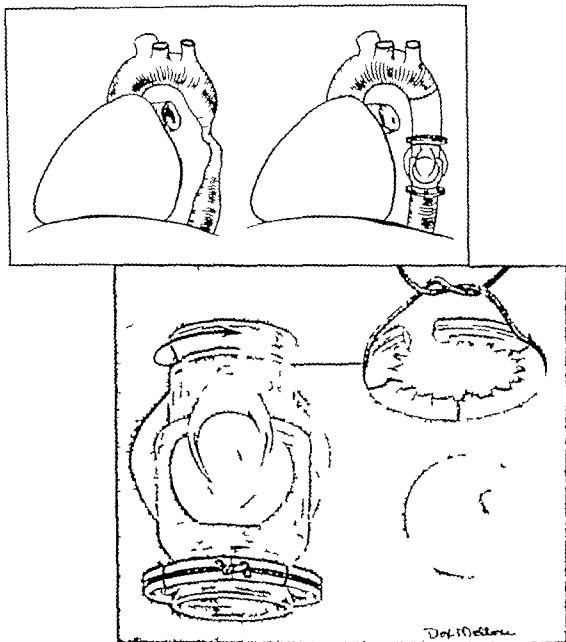


Figure 81 Aortic insufficiency and coarctation of the aorta corrected by resection of the coarctation, insertion of aortic autograft and a plastic valvular prosthesis. Below is shown the plastic ball-valve prosthesis and the "multiple point" fixation rings.

murmurs and signs of congestive failure of varying degree and most have had angina pectoris. Patients who are in the terminal phases of their disease should not be offered operation, except as desperate risks. In this phase of the disease, their myocardium is usually irreparably damaged and cannot recover in spite of improvement in valvular function. Patients who have had acute rupture of the cusp should be treated as soon as practical by operation because of the extremely bad prognosis with medical management. Every effort should be made to screen the patients to determine those who have activity of their rheumatic process. Pa-

tients with aortic valvular lesions tolerate operation very poorly if the rheumatic process is active.

The optimal patient for operation is one who is under fifty five years of age, with no more than moderate cardiac enlargement, but who has begun to have symptoms. Those patients who have had rapid production of severe aortic insufficiency should be offered operation very early because acute aortic insufficiency carries an extremely poor prognosis with conservative management. When multivalvular disease, such as combined mitral disease and aortic disease, is present, combined operation is advised. Similarly,

cardial and mediastinal arteries) can form additional communications with the coronary circulation and occasionally the entire cardiac blood supply may be derived from extracoronary sources.

Surgical efforts to correct the effects of coronary artery disease have taken several forms. These have included attempts to (1) develop collaterals by vascular grafts or by pericardial adhesions to the myocardium, (2) stimulate natural anastomotic channels, (3) decrease the incidence of ventricular fibrillation by nerve section, (4) increase the blood myocardial contact time by obstruction of the venous outflow tract, (5) reverse the venous circulation with arterialized blood, (6) direct removal of obstructive lesions in the coronary arteries.

At the present time several operations are in widespread use. The simple procedures have had wider application since they have lower operative risk. These include ligation of the internal mammary artery distal to its pericardiophrenic branch, and simple pericardial poudrage with talc or asbestos. Neither of these procedures has been shown to be unequivocally helpful. Both procedures, however, appear to have features which recommend them for selected cases.

Many different surgical methods have been tried to produce vascular adhesions between the pericardium and the myocardium in order to increase the flow of blood to the heart muscle. The epicardium has been shown experimentally to be a barrier for the ingrowth of blood vessels between these two structures. Most observers now believe that it is necessary to remove the epicardial barrier by physical or chemical means to secure adequate development of vascular communications. Abrasion or epicardiolytic also develops communications between the right and left coronaries to obtain a more equal distribution of the blood flow.

Poudrage procedures combined with the epicardiolytic utilize talc or asbestos to stimulate the development of vascular communications. The combination of poudrage, epicardiolytic and ligation of the great cardiac vein, or partial ligation of the coronary sinus is a single procedure which is frequently advocated.

Reversal of circulation by anastomosis of a systemic artery to the coronary sinus has been used, combined with partial ligation of the coronary sinus distal to the point of anastomosis. This procedure has been associated with considerable operative risk and is not widely used.

Resection of the anterior plexus has been shown to decrease the incidence of ventricular fibrillation following experimental ligation of the circumflex coronary artery or the left anterior descending coronary artery. *There is considerable evidence to show that ventricular fibrillation does not occur purely as a result of cardiac ischemia and that neurogenic impulses, or currents of injury in the myocardium mediated through the anterior cardiac plexus play a role in the development of fibrillation.*

Implantation of the isolated internal mammary artery into the myocardium, leaving two to four of its terminal branches unligated, has also been advocated as a method of increasing the blood supply to the myocardium. It has been shown that such implants are patent after prolonged periods and that anastomoses develop between the implant and the branches of the coronary arteries. The results of this procedure combined with epicardiolytic of the heart and sometimes with ligation of the great cardiac vein have been promising.

The success which has been obtained in the treatment of obstruction of peripheral arteries by endarterectomy has led to the application of this technique to the coronary vessels. When a localized obstruction exists, this may be removed along with the thickened intima of the vessel. Localization of the point of obstruction is obtained by means of retrograde aortography and filling of the coronary arteries through the aortic root. The injection of small amounts of contrast material following the termination of systole gives excellent coronary arteriograms. This technique utilizes the electrocardiogram to trigger the injection and the radiographic exposures. The use of an image amplifier permits the following of the contrast medium through the coronary circuit. Some workers have employed momentary cardiac arrest induced by the injection of acetylcholine making the injection into the aortic root during the period of arrest. The results of coronary endarterectomy have been encouraging, but it is still too early to evaluate them.

Patients who are most suitable for operation are those who manifest severe angina pectoris which is not controllable by medical means and those patients in the younger age groups in whom the prognosis is serious. These indications will gradually be extended as the benefit of operation is more fully demonstrated. An increasing number of patients previously incapacitated by coronary

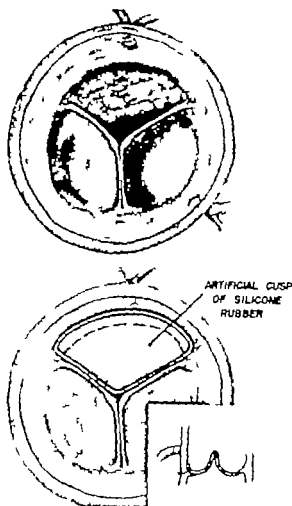


Figure 23. Replacement of a single disease-involved cusp.

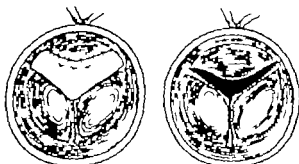


Figure 24. Addition of substance to a scarred leaflet. This addition to the valve permits the leaflets to again form a seal.

the valvular lesion corrected and the aortic root reconstructed or replaced.

When the indirect method for the correction of aortic insufficiency is used and the plastic valve is placed in the distal arch, the flow from the upper extremities remains regurgitant. In spite of this, marked improvement has been manifest in essentially all of the patients who have had successful

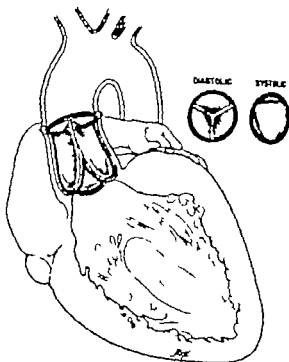


Figure 25. Cusp-type valve for replacement of aortic valve.

operations of this type. When successful repair can be accomplished by a direct operation, blood pressures return to normal in all extremities. A large number of patients now have had direct repair. The risk of direct operation is greater than with the previous type but is steadily decreasing. It is now possible to repair or replace the diseased aortic valve by direct means in almost all cases. The results of this type of operation are excellent with restoration of essentially normal dynamics. Operation is advised early and in these cases direct reconstruction of the valve can be accomplished with great safety.

CORONARY ARTERY DISEASE

Obstructive disease of the coronary arteries, with the resultant decrease in myocardial blood flow, is one of the principal causes of death. Coronary occlusion causes death by one of the following mechanisms: congestive failure, ventricular fibrillation, cardiac aneurysm, perforation of the interventricular septum and rupture of a papillary muscle or chorda tendina.

Anastomoses between the right and left coronary arteries are present to a varying degree in different individuals. With the development of obstruction to a coronary artery the collateral channels between the right and left coronary vessels may increase. Extracoronary collaterals (bronchial, peri-

deceleration. Penetrating wounds may also give rise to aneurysm but more commonly give rise to pulsating hematoma or arteriovenous fistula. Those associated with non-penetrating injuries usually occur just distal to the left subclavian artery and tend to be moderate in size.

Mycotic aneurysms associated with infectious processes are seen throughout the ascending portion of the aorta in the transverse arch. They are occasionally seen in major branches. Following septic embolism, they may be found in any arterial vessel.

Dissecting aneurysm is believed to be associated with medial cystic necrosis of the aortic wall. A dissecting aneurysm should be distinguished from a leaking or rupturing aneurysm of other types. Dissection is associated with the establishment of a secondary lumen in the media which communicates with the true lumen through a break in the intima. As the dissection proceeds the two coats are further separated to a variable degree by the blood under systolic pressure. Re-entry of a dissection through a second opening in the intima is frequently associated with spontaneous cure. The majority of dissections begin in the ascending aortic arch. A considerable number of such dissections however begin at the level of the left subclavian artery. When the dissection begins very low in the ascending aorta at the level of the aortic valve, aortic insufficiency may be associated. Similarly in this location the coronary artery may be occluded or avulsed.

Aneurysms of the aorta and its major branches are serious and progressive lesions, unless surgically treated they are almost universally fatal. The incidence of the various types of origin of aneurysm varies with the local incidence of syphilis. Prognosis in all such lesions is poor but varies with the etiologic basis of the aneurysm. It is uncommon for a patient with aneurysm which is of sufficient size to be detected by physical examination or x ray to survive for more than five years after its discovery. In individual cases of prolonged survival in patients with large aneurysms have been reported, but these are exceptional. Smaller aneurysms within the thorax seldom produce symptoms and are usually discovered by routine x-ray examination. Aneurysms of larger size may produce symptoms of varying degree. These are due to the encroachment of the aneurysm upon surrounding structures or distention of the aortic wall.

Principal symptoms include pain in the chest or back, cough due to bronchial or tracheal compression fever from pulmonary infection secondary to bronchial obstruction, hemoptysis, hematemesis and dyspnea.

There may be no physical signs which are abnormal. There may be evidence of a mass in the thorax or mediastinum atelectasis congestive heart failure, evidence of superior or inferior vena caval obstruction, nerve root compression, peripheral embolization or evidence of a pulsating mass in the mediastinum or thorax. Murmurs or bruits may be present but are most frequently absent.

X ray examination shows abnormal dilatation of the aortic shadow with intrinsic pulsations on fluoroscopy. In syphilitic aneurysm there is a characteristically linear calcification in the ascending arch. In the lower portion of the descending aorta the aneurysm may lie behind the cardiac shadow and oblique or lateral views may be necessary to delineate it. Angiocardiography and retrograde aortography may be helpful in delineating multiple masses and in differentiating saccular from fusiform aneurysms. Differential diagnosis of masses which lie in juxtaposition to the aorta may be difficult without angiocardiography since these masses may appear to pulsate because they transmit the aortic impulse. Technical developments have made possible the removal of essentially all aneurysms, providing there is no systemic contraindication in the form of the patient's general condition or other more serious disease. The treatment of choice is resection and replacement by a plastic cloth prosthesis. In exceptional instances freeze-dried homografts may be preferable. The older methods which have included wiring and wrapping, sclerosing agents and electrocoagulation seldom find a place in the modern treatment of this disease.

Saccular aneurysms offer the simplest problem in treatment. These aneurysms usually have a narrow neck which opens into the aorta with an orifice of 1 to 6 cm. in diameter. After the aneurysm has been dissected from its surrounding structures a clamp can be applied across its base, including in it the normal aortic wall beyond the sac which can then be excised. The lateral opening is closed by sutures. In some cases there is such an extensive involvement of the aortic wall that it is preferable to remove the full diameter of the aorta and replace it

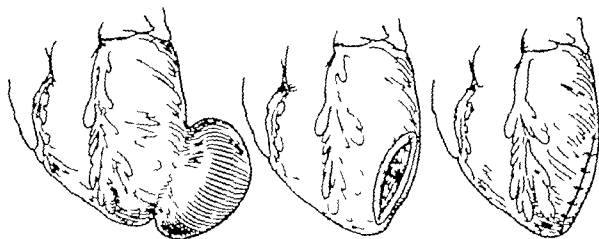


Figure 26 Ventricular aneurysm. The edges of the aneurysm are fibrous and strong. The sutured ventricular wall is shown after removal of the aneurysm.

disease are being returned to active useful lives by surgical treatment. The results of internal mammary artery implantation are particularly striking.

VENTRICULAR ANEURYSM

Ventricular aneurysm is a complication of coronary artery disease. In the immediate postinfarction period, the aneurysm may rupture, but after fibrosis is established rupture is rare. Congestive heart failure however is a common accompaniment of large aneurysms. The loss of effective contraction and the paradoxical filling of the aneurysm appear to be significant contributing features in this failure.

Surgical correction is accomplished by direct excision with total bypass of the heart. The edges of the aneurysm as it joins the ventricular muscle are fibrous and strong. Reconstruction of the ventricle is extremely satisfactory. Clots which may be present in the aneurysmal sac can be removed with safety. Risk of operation is low.

ANEURYSMS OF THE AORTIC ARCH AND THORACIC AORTA

Aneurysms of the aortic arch are of six principal types: syphilitic, congenital, arteriosclerotic, traumatic, dissecting and mycotic. Syphilitic aneurysms are most common in the ascending aortic arch but may occur in the transverse or descending thoracic aorta. Less frequently they occur in the abdominal aorta. While these are frequently localized and saccular they may involve the entire ascending and transverse arch in a fusiform manner. The saccular form may be large in total size and still have an opening into the aorta itself which is quite small. Others may be saccular in type but involve

a considerable portion of the aortic circumference. Fusiform aneurysms of the ascending arch frequently involve the aortic annulus and are associated with aortic insufficiency. The presence of linear calcium in the descending aorta is a frequent finding in syphilitic aortitis and may be helpful in establishing the diagnosis.

Congenital aneurysms are relatively uncommon, except in the sinus of Valsalva. Aneurysms of the sinus of Valsalva tend to elongate downward into the region of the right atrium or the right ventricle. When rupture occurs, it may be either into the right atrium or into the right ventricle or occasionally the rupture may occur into the left ventricle.

In coarctation, the aneurysms which are found in the aorta and intercostal vessels are rarely present at birth but develop later in life although they may be apparent as early as six years. Other developmental abnormalities of the aorta may give rise to aneurysm either in youth or later. Overt or occult Marfan's syndrome and other forms of medial degeneration fall into this category.

Arteriosclerotic aneurysms may involve any portion of the thoracic aorta. These tend to be almost exclusively fusiform in character although they may be multiple and give x-ray appearance of aneurysms. Arteriosclerotic aneurysms tend to involve large portions of the aorta and are associated with generalized dilatation of the aortic diameter in some individuals. Tortuosity and elongation of the aorta are sometimes confused with true aneurysm.

Traumatic aneurysms are usually associated with nonpenetrating injuries of the chest in which there is sudden and rapid

occurs, the heart beat restarts and when a satisfactory beat is obtained by pass is discontinued.

By the use of one or a combination of these methods it is possible to resect essentially all aneurysms of the thoracic aorta and to replace them by prostheses. Aneurysms of the ascending arch, transverse arch and entire arch are still difficult problems. Aneurysms of the descending arch can be resected with a low mortality rate.

Rupture of an aneurysm is seldom immediately fatal but is an acute surgical emergency. The diagnosis of leakage or rupture is the prelude to death. In a high percentage of cases, the diagnosis can be made sufficiently early for surgical treatment to be highly effective. Hesitancy and excessive delay lead only to disaster.

Dissecting aneurysm has a more rapid course and more specific symptomatology than other types. These are frequently seen in a younger age group than are aneurysms of the arteriosclerotic or syphilitic type. If re-entry does not occur survival is extremely rare. There is usually an adequate period between the onset of symptoms of dissection and occurrence of death for surgical therapy to be undertaken. Dissecting aneurysm sometimes presents itself with signs of obstruction of one of the major vessels of the extremities. Obstruction of the subclavian, carotid or iliac vessels may produce signs of ischemia in the area involved. As dissection proceeds it may also give rise to an abdominal mass which is pulsatile and suggestive of an abdominal aneurysm of the arteriosclerotic type. Onset of dissection is usually heralded by sudden severe pain which tends to radiate through the back, up into the neck or downward along the course of the thoracic aorta. When the abdominal aorta is also involved a secondary complaint may be pain in the abdomen or in the legs. Differences in the volume of the pulse or in the blood pressure on the right and left sides may give a clue to the proper diagnosis. X rays of the chest may show enlargement of the aortic shadow to the right or to the left. Immediately after the onset, the aortic shadow may not be markedly abnormal, but over a period of several hours or days this may change markedly. Angiocardiography may show a double lumen or an irregular dilatation suggestive of aneurysm.

Operation is indicated as soon as the diagnosis is made. Diagnosis of dissecting aneurysm is also a surgical emergency which requires immediate preparation of the pa-

tient for operation. The usual procedure is to produce a re-entry point high in the thoracic aorta. Left thoracotomy is performed and the first portion of the descending aorta is freed from its surrounding structures. If the origin of the dissection is in the proximal aorta no attempt is usually made to seek the point of origin. If the origin is at the level of the left subclavian artery the aorta proximal to this point is normal the area which includes the point of origin is freed so that a clamp can be placed proximal to it. When the descending aorta is cross-clamped, the double lumen of the dissection is visible. The blood is evacuated from both chambers and on the proximal side a segment of the inner wall is removed. This permits the entry of blood into the true aortic lumen at this point. In the distal segment, the false lumen is obliterated by suturing the intima to the media with multiple sutures. This converts the distal aorta into a single-lumen tube. The re-

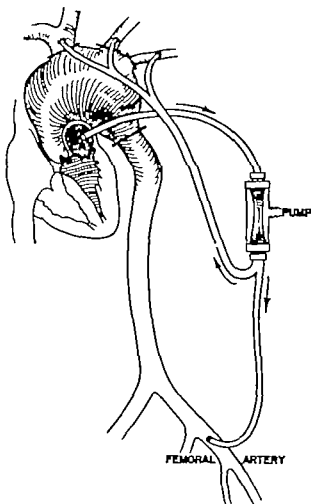


Figure 29. Aneurysm of the aortic arch showing catheters inserted into cranial arteries and femoral artery to supply the brain and viscera during the period of interruption of aortic circulation.

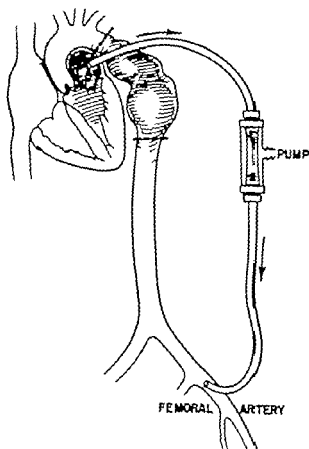


Figure 27 Aortic aneurysm of descending arch. Blood is supplied distal to the aortic clamps by a pump which removes oxygenated blood from the left atrium and replaces it by way of the femoral artery.

with a prosthesis. Teflon or Dacron materials of the seamless elastic type are preferred for this purpose.

Fusiform aneurysms which involve the descending aorta or traumatic aneurysms in this area are also excised and replaced with a prosthesis of the type described above. Since occlusion of the descending aorta cannot be safely maintained for more than thirty minutes without damage to the spinal cord or other peripheral viscera, cooling the body to 30° C. is used to prolong this safe period of occlusion. With the aneurysm isolated and all its branches ligated and divided, the aorta can be occluded above and below the lesion and the involved area excised. A prosthesis is then sutured to bridge the gap between the divided ends of the aorta. These two anastomoses can usually be completed within twenty minutes. When the aneurysm is very large and its resection cannot be safely accomplished without interruption of the circulation, blood may be shunted around the aneurysm to allow prolonged clamping of the aorta. Several meth-

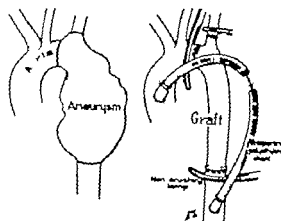


Figure 28 Resection of a thoracic aortic aneurysm involving the left subclavian artery may be accomplished by the shunting technique. Blood is supplied to the lower thoracic aorta through a plastic or heterograft by-pass while the aorta is occluded.

ods are used to achieve this. A catheter may be placed into the left atrium where oxygenated blood can be withdrawn by gravity into a pump which then propels the blood back into the arterial system through a catheter in the femoral artery (Fig. 27). This withdrawal of blood from the left atrium reduces the load on the left ventricle while the aorta is clamped and at the same time supplies blood to the viscera distal to the aneurysm. An alternate procedure for the shunting of blood employs the suture of a prosthesis or graft to the side of the aorta proximal to the aneurysm and the other end to the aorta distal to the aneurysm (Fig. 28). Circulation is then maintained through this temporary shunt during the period of occlusion and restoration of the circulation. At the conclusion of the operative procedure the shunt is removed.

Aneurysms involving the transverse arch may be removed by means of a similar system, but one which includes additional shunts to supply the brain by cannulation of the head vessels during the period in which they are occluded (Fig. 29). An additional method employs total body cooling to a point of cardiac arrest. When the body temperature has reached 18° C. the proximal and distal portions of the aorta are clamped, together with all of the branches. The aneurysm is resected and the prosthesis sutured in place. During this period the extracorporeal circulation is stopped. Following placement of the graft, the circulation is restored by means of the extracorporeal heart lung and the blood is warmed to restore effective cardiac action. As warming

duce murmurs associated with other valvular lesions. Metastatic tumors may be associated with pericardial effusion and tumor cells can usually be isolated from this fluid. Myxomas of the atrium are prone to give rise to emboli. Such emboli when removed from the peripheral vessels show a gelatinous character which is quite characteristic.

Primary tumors of the heart are best approached by open direct vision operation. With utilization of total cardiopulmonary by-pass, the involved chamber usually the left atrium, is incised. After aspiration of the blood in the heart, the tumor is visualized and removed with a portion of the surrounding muscle. This defect is closed by direct suture. After the heart is refilled with blood it can be permitted to resume its normal function. Excision of that portion of the septum or cardiac wall to which the tumor is attached can result in complete removal. Tumors of the ventricle may be approached in a similar manner with excision of a portion of the ventricular wall. Malignant tumors of the ventricle however show invasion of such large muscle masses that total removal is frequently impossible. Angiocardiology and cinefluoroscopy are frequently helpful in localizing such intracardiac tumors. The prognosis after removal of benign tumors is excellent.

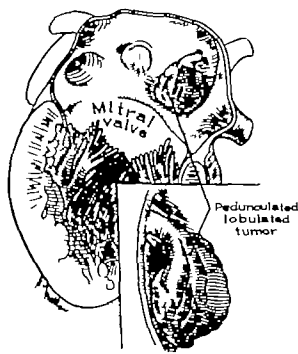


Figure 32. Pedunculated myxomatous tumor of the left atrium, which partly obstructed the mitral valve. This type of tumor is particularly suitable for surgical removal.

CARDIAC ARREST

Cardiac arrest is the general designation for the sudden cessation of an effective heart beat occurring most often during anesthesia or surgical operation. This is an emergency which every physician must expect to meet and for which he must have a positive plan. It is imperative that every surgeon be fully aware of the problems of cardiac arrest and the mechanics of cardiac resuscitation. It demands immediate and definitive action to save the life of the patient. The well prepared surgeon can expect to restore an effective beat with survival of the patient in a high percentage of cases. While acute cardiac arrest most commonly occurs in the anesthetized patient, it may also be seen in the immediate postoperative period associated with the passage of a nasopharyngeal tube removal of the endotracheal tube or other pharyngeal or vagal stimulation.

The major factors precipitating arrest are hypoxia, hypercapnia, blood loss or anemia, overdose of anesthetic agents, cardiac diseases with conduction defects or coronary insufficiency and vagal or glossopharyngeal stimulation.

Adequate ventilation and the prevention of the accumulation of carbon dioxide by the anesthesiologist are the most important preventive measures for this disaster. The use of an electrocardiograph monitor should be routine in all patients who are known to have serious heart disease.

The term "cardiac arrest" includes asystole, ineffective contractions and ventricular fibrillation, since all result in cessation of an effective heart beat. The differential diagnosis between these is made only after the thorax has been opened.

The diagnosis of cardiac arrest is made by the absence of the blood pressure and the absence of a palpable major peripheral pulse. If the anesthesiologist cannot obtain a blood pressure reading, the surgeon should immediately palpate the most accessible major blood vessel. If the surgeon has the abdomen open, the aorta or iliac arteries can be felt. The anesthesiologist should constantly check the patient's carotid pulse during all procedures. If this suddenly ceases to be palpable and the blood pressure can not be obtained, the diagnosis of cardiac arrest must be presupposed. No time should be wasted in attempting to obtain an electrocardiogram, listening for a heart beat or other time-consuming procedures.

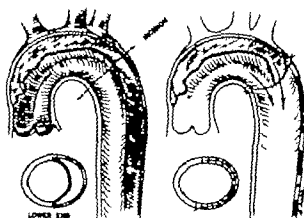


Figure 30 A dissecting aneurysm arising in the ascending aorta shows the characteristic double lumen. Transection of the aorta distal to the left subclavian artery permits the establishment of a re-entry site. The double lumen is obliterated distally by approximation of the two walls.

removal of the intima also leaves a single lumen proximally. The aorta is now reanastomosed, approximating these two layers (Fig. 30). If the aortic wall is extremely thin in its proximal segment, it may be reinforced externally with a layer of Dacron or Teflon cloth after the anastomosis has been completed and the circulation re-established. If the origin of the dissection can be found distal to the left subclavian artery the aorta is sectioned proximal to the dissection. The distal segment is then converted to a single lumen and anastomosis of the two segments is then accomplished either directly or by interposition of a prosthesis (Fig. 31). These two procedures offer successful correction in an otherwise hopeless situation. Ideal therapy in acute or chronic dissection or when chronic dissection is associated with an aortic valvular lesion is direct reconstruction

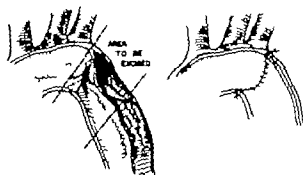


Figure 31 Dissecting aneurysm which has arisen at the level of the left subclavian artery. The intimal tear has been excised and the distal lumen obliterated by sutures. Direct anastomosis or the interposition of a prosthesis gives complete restoration of normal flow.

of the aortic root with cardiopulmonary bypass. In such cases false lumen is obliterated by nature of the two walls or suture of the inner and outer layers together with reapproximation. In some cases it is necessary to replace the ascending aorta with a prosthesis to properly reconstruct the vessel. This procedure has been successfully accomplished only in a few cases but would appear to offer a more logical approach to the problem.

TUMORS OF THE HEART

In 1783 de Senar stated, "the heart is an organ too noble to be attacked by a primary tumor." In spite of this by 1945 over 300 primary cardiac tumors had been reported in the eighty years following the first report of a sarcoma of the atrium in 1863.

Primary cardiac tumors are infrequent but are found more often than ever before. The incidence has been variously estimated as from three to fifteen in 10,000 autopsies. The majority (41) of primary tumors are benign. The diagnosis of tumor of the heart has not been frequently made ante mortem until recently when improved methods and increased awareness have led to more diligent efforts to localize the lesion and in some cases to treat it surgically.

The benign tumors most frequently encountered are fibroma, myxoma, lipoma and rhabdomyoma.

Myxomatous tumors occur frequently in the left atrium where they tend to be pedunculated and attached to the atrial wall or septum (Fig. 32). They may project over the mitral valve and stimulate mitral stenosis or ball valve thrombus. Prolapse of the tumor through the valve may cause acute mitral obstruction and death.

Muscle tumors may appear on the surface of the ventricle or deep within its substance.

Malignant tumors are dominantly sarcomas and include rhabdomyosarcoma, lymphosarcoma, fibrosarcoma, myxosarcoma, leiomyosarcoma and hemangioendothelioma.

Metastatic tumors of the heart are common. Malignant disease within the thorax, particularly bronchogenic carcinoma, is associated with direct or indirect invasion of the heart.

Signs and symptoms of cardiac tumors may be bizarre. These lesions often mimic mitral stenosis or constrictive pericarditis or pericardial effusion. They sometimes pro-

the chest in anticipation of cardiac arrest and with continuous monitoring of the electrocardiogram or an oscilloscope electric shocks of sufficient intensity to elicit an effective cardiac contraction can be immediately instituted if standstill occurs. When ventricular fibrillation supervenes under these controlled conditions, external defibrillation may be attempted, using a shock of 700 or more volts, applied by means of the external electrodes. If these measures fail to give an immediate response in the production of an effective sustained cardiac beat, the chest must be immediately opened and the procedures previously outlined instituted.

Every operating suite should be equipped with an emergency cardiac resuscitation tray which is kept sterile at all times. The necessary drugs—epinephrine, procaine or Nylcaline, calcium chloride, digitalis, quinidine and Pronestyl—should be in a standard location in every operating room.

Following restoration of the effective cardiac beat, catheter drainage of the pleural space should be routine. The use of antibiotics intrapleurally and intramuscularly is to prevent sequelae of contamination of the pleura and pericardium. Infection of the chest or pericardium even when this procedure is done without the usual aseptic precautions is rare.

The failure to recognize cardiac arrest immediately or hesitation in undertaking definitive action to secure cardiac massage within the first five minutes, usually results in failure in the attempt of resuscitation. If the delay has been excessive, it is sometimes possible to resuscitate the heart, but irreversible brain damage has occurred. This results either in brain stem damage with failure to resume respiration or in the loss of the higher cortical functions. Successful restoration of the heart beat should be possible in approximately 70 per cent of the patients with cardiac arrest.

READING REFERENCES

Bailey, T. G. Riker, W. L., DeBoer, A., and Potts, W. J.: Surgical Correction of Transposition of the Aorta and the Pulmonary Artery. *J. Thoracic Surg.* 34:460-484, Oct. 1957.
 Balmann, H. T.: Definitive Treatment of Sacular Aneurysms of the Aorta with Excision of Sac and Aortic Suture. *Surg. Gynec. & Obst.* 96:383-402, April 1953.
 Beck, C. S.: Acute and Chronic Compression of the Heart. *Am. Heart J.* 14:515-525, Nov. 1937.
 Beck, C. S., Brofman, B. L., and Meutz, F. R.: Symposium on Coronary Artery Disease. (Beck, Blood

Supply to Ischemic Myocardium Distal to the Occlusion of A Coronary Artery. Brofman: Surgical Treatment of Coronary Artery Disease, Medical Management and Evaluation of Results. Meutz: Anatomical and Physiological Considerations in the Development of A Collateral Circulation to the Myocardium. *Dis. Chest* 31:243-288, March 1957.
 Blalock, A.: Surgical Procedures Employed and Anatomical Variations Encountered in the Treatment of Congenital Pulmonary Stenosis. *Surg. Gynec. & Obst.* 87:385-400, Oct. 1948.
 Blount, S. G., Davies, D. H., and Swan, H.: Atrial Septal Defect—Results of Surgical Correction in One Hundred Patients. *J.A.M.A.* 169:210-213, Jan. 17, 1950.
 Brock, R., and Fleming, P. R.: Aortic Subvalvular Stenosis: Report of 5 Cases Diagnosed During Life. *Guys Hosp. Rep.* 105:391-408, 1956.
 Brock, R. C.: The Surgical and Pathological Anatomy of the Mitral Valve. *Brit. Heart J.* 14:489-513, Oct. 1952.
 Brunton, L.: Preliminary Notes on the Possibility of Treating Mitral Stenosis by Surgical Methods. *Lancet* 1:352, 1903.
 Cooley, J. C., Clagett, O. T., and Kirklin, J. W.: Surgical Aspects of Chronic Constrictive Pericarditis. (Review of 72 Operative Cases.) *Ann. Surg.* 147:488-493, April, 1958.
 DeBakey, M. E., Cooley, D. A., Crawford, E. S., and Morris, C. G., Jr.: Aneurysms of the Thoracic Aorta. (Analysis of 179 Patients Treated by Resection.) *J. Thoracic Surg.* 36:993-1000, Sept. 1958.
 Fallot, A.: Contribution à l'anatomie pathologique de la maladie bleue (Cyanose cardiaque). *Mar. scille-méd.* 25:77 138, 207, 270 341 403, 1868.
 Fauteux, M.: Treatment of Coronary Disease with Angina, by Pericoronary Neurectomy Combined with Ligation of the Great Cardiac Vein, Case Report. *Am. Heart J.* 31:260-269, March, 1946.
 Fyler, D. C., Rudolph, A. M., Wittenberg, M. H., and Nadas, A. S.: Ventricular Septal Defect in Infants and Children. (A Correlation of Clinical, Physiologic and Autopsy Data.) *Circulation* 18: 833-851, Nov. 1958.
 Gammella, J. H., Anderson, J. G., and Oropesa, R.: The Surgical Treatment of Aortic Insufficiency by Open Plastic Revision of the Tricuspid Aortic Valve to a Bicuspid Valve. *Surg. Gynec. & Obst.* 106:670-686, June 1958.
 Gerbode, F., and others: Left Atrial Myxoma. Successful Removal with the Aid of Extracorporeal Circulation. *Ann. Surg.* 147:320-325, March 1958.
 Glover, R. P.: The Present Status of Patients Subjected to Mitral Commissurotomy Five or More Years Ago. *Surg., Gynec. & Obst.* 102:623-625, May 1956.
 Grom, R. E.: Complete Surgical Division of the Patent Ductus Arteriosus. A Report of 14 Successful Cases. *Surg. Gynec. & Obst.* 78:36-43, Jan. 1944.
 Grom, R. E.: Coarctation of the Aorta. *Circulation* 7:757-768, May 1953.
 Grom, R. E., and Hufnagel, C. A.: Coarctation of the Aorta, Experimental Studies Regarding Its Surgical Correction. *New England J. Med.* 233: 287-293, Sept. 6, 1945.
 Gross, R. E., and Longino, L. A.: The Patent Ductus

The left side of the chest is exposed as rapidly as possible. All anesthetic drugs are discontinued and the patient is ventilated by positive pressure with oxygen. A left anterolateral incision should be made over the precordium beneath the breast. No time should be wasted in preparing the field, putting on gloves or counting ribs. The chest is opened through an intercostal space and immediate compression of the heart is begun through the intact pericardium. The heart is rhythmically compressed with the hand in such a fashion that the blood is forced from the apex to the base of the heart. If a spontaneous beat is not obtained after several such compressions, the pericardium is immediately opened anterior to the phrenic nerve and compression continued. Inspection of the heart will reveal whether the heart is at standstill or in ventricular fibrillation. Bimanual compression may be necessary if the heart is large or if it is atonic. With manual compression of the heart, the peripheral pulses should become palpable. Unless a pulse and blood pressure are obtainable the compression is ineffective. Asystole and ventricular fibrillation differ in the details of management in the later phases but in both instances the initial therapy is the same—the rapid establishment of effective cardiac compression and adequate ventilation of the lungs.

When the heart is found to be in asystole it may resume a good beat with the first few compressions of the heart. If it does not, the rhythmic compression is continued. When the heart begins to recover it becomes firmer and a few spontaneous beats are noted and gradually the contractions become stronger. If no improvement is noted, 5 cc. of a 10 per cent solution of calcium chloride are administered into the left atrium. The assistant compresses the aorta in its descending portion to obtain more effective circulation to the brain. The head of the patient is tilted downward. With further massage being maintained continuously at a rate of fifty to seventy per minute, spontaneous beats may occur. If the heart remains in standstill or is inefficient in its contractions 1 cc. of 1:10,000 solution of epinephrine may be injected into the left atrium. Continuing cardiac massage and the cautious administration of calcium chloride and epinephrine in increasing doses will usually restore an effective beat. Epinephrine should be used cautiously because of the danger of precipitating ventricular fibrillation in an anoxic heart and it should be

given only when the color of the heart can be maintained well by massage.

If on inspection the heart is found to be fibrillating, the initial procedure is the same as for cardiac arrest, that is immediate rhythmic myocardial compressions. Injection of 5 to 10 cc. of a 1 per cent solution of procaine hydrochloride into the left atrial appendage frequently will cause a restoration of a normal beat in patients who have a coarse type of ventricular fibrillation. Aortic compression and continuous massage are used as they are in patients having cardiac arrest. Block of the sinoatrial node by injection of 5 cc. of 1 per cent solution of procaine or Xylocaine may also help restore a normal rhythm.

Electric shock is frequently necessary for the defibrillation of the heart. An alternating current of 250 volts at 2.5 amperes for one twentieth to one tenth of a second is applied to the heart by means of electrodes which are placed on either side of the heart. The electrodes are wrapped in gauze which is soaked in saline solution. It is important that the electrodes be in firm contact with the surface of the heart and that they are not touching each other. The application of shock will usually result in cardiac standstill. The heart may then resume an effective contraction or it may remain in asystole. With compression and the cautious use of calcium chloride and epinephrine restoration of the beat may then be accomplished.

The use of molar sodium lactate may be helpful in the defibrillation of hearts which are refractory. In patients who are hypothermic, injection of tetraethylammonium chloride into the clamped aortic root may also make possible the restoration of normal rhythm, either spontaneously or when combined with electric shock. Injection of small graduated doses of potassium citrate into the aortic root also may make possible the conversion of fibrillation which does not respond to the usual methods.

Following restoration of a normal beat, it may be necessary in some cases to digitalize the patient rapidly. If ventricular fibrillation has been present, the use of quinidine sulfate may also help prevent a recurrence.

Outside the operating room, the external pacemaker and defibrillator may be used to meet other emergency situations, particularly those which can be anticipated, such as in patients with Stokes Adams attacks or ventricular tachycardia. The electrodes of the external pacemaker may be placed on

THE ABDOMINAL WALL AND PERITONEUM

By WALTER C. MACKENZIE, M.D.,
and ROBERT A. MACBETH, M.D.

WALTER CAMPBELL MACKENZIE was born in Nova Scotia, the only son of Canadian-Scottish parents and the grandson of a sea captain. He is the Professor of Surgery and Director of the Department of Surgery at the University of Alberta. His college and medical education were received at Dalhousie University in Halifax. He was a Fellow in Surgery at the Mayo Foundation and Clinic.

He served his country as surgical consultant to the Royal Canadian Navy and has played an important role in medical education and research in Canada.

ROBERT ALEXANDER MACBETH, born in Edmonton, the son of a doctor, was educated at the University of Alberta. Trained in surgery at McGill University, he has been a research Fellow of the National Research Council and a teaching Fellow in Anatomy. With the approach of a perfectionist, he has a great interest in medical education, research and medical history. He is an Associate Professor of Surgery at the University of Alberta.

INTRODUCTION

A painstaking examination of the abdomen is one of the most rewarding diagnostic procedures available to the physician. The student must develop an ability to appreciate even minor variations from normal while conducting this examination. To do this he must avail himself of every opportunity during his training to examine the abdomens of normal individuals, of patients presenting

with abdominal complaints and of postoperative patients.

The physical findings elicited by examination of the abdomen, together with an appreciation of the physiologic mechanisms involved, are the key to the diagnosis of numerous pathologic conditions of the abdominal wall and more particularly of the peritoneal cavity. In an acute disease process, physical examination is most rewarding.

- Arteriosus, Observations from 412 Surgically Treated cases. *Circulation* 3:125-137 Jan. 1951
- Holman, E. The Recognition and Correction of Constrictive Pericarditis. *J Thoracic Surg.* 18:643-651 Oct. 1949
- Hufnagel, C. A. The Use of Rigid and Flexible Plastic Prostheses for Arterial Replacement. *Surgery* 37:165-174 Feb. 1953
- Hufnagel, C. A. Direct Approaches for the Treatment of Aortic Insufficiency. *Am Surgeon* 25:321-327 May 1959
- Lewis, P. J. Winchell, P., and Baubour, F. A. Open Repair of Atrial Septal Defects. *J.A.M.A.* 165:923-927 Oct. 26, 1957
- Lillehei, C. W. Warden, H. E. and Varco, R. L. Complete Anatomical Correction of the Tetralogy of Fallot Defects. *Arch Surg.* 73:526-531 Sept. 1956.
- Potts, W. J. and Gibson, S. Aortic Pulmonary Anastomosis in Congenital Pulmonary Stenosis: Report of 45 Cases. *J.A.M.A.* 137:343-347 May 22, 1948
- Reid, L. C. Del Minner, P. A. and Hinton, J. W. Cardiac Arrest: A Review. *New York J Med.* 58:4035-4049, Dec. 15, 1958.
- Shumacker, H. B., Jr., and Lurie, P. R. Patent Ductus Arteriosus with Pulmonary Hypertension. *Arch Surg.* 76:179-182, Feb. 1958.
- Souttar, P. Surgical Treatment of Mitral Stenosis. *Brit. M J* 2:603, 1925.
- Swan, H., and Zelevin, L. Cessation of Circulation in General Hypothermia. III. Techniques of Intracardiac Surgery under Direct Vision. *Ann. Surg.* 139:385-396 April 1954.
- Thevenet, A., Hodges, P. C., and Lillehei, C. W. The Use of a Myocardial Electrode Inserted Percutaneously for Control of Complete Atrioventricular Block by an Artificial Pacemaker. *Dis. Chest* 34:621-631 Dec. 1958
- Vineberg, A. and McMillan, G. C.: The Fate of the Internal Mammary Artery Implant in the Ischemic Human Heart. *Dis. Chest* 32:84-85 Jan., 1958.
- Williams, G. R., Jr. and Spencer, F. C.: The Clinical Use of Hypothermia Following Cardiac Arrest. *Ann. Surg.* 148:462-468, Sept. 1958.

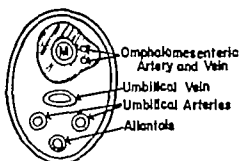


Figure 4 Diagrammatic cross section of umbilical cord of 3.5-mm. human embryo. M omphalomesenteric duct.

embryo stage can probably be best appreciated by reference to Figure 4 which is a cross section of the cord of the embryo in Figure 3 at Y. The patent omphalomesenteric duct (M) with its accompanying artery and vein on its surface, lies in the extra-coelom (shaded) and occupies the superior portion of the cord. Inferior to it, embedded in mesenchyme, are respectively the umbilical vein, the paired umbilical arteries and the allantoi. The umbilical vein divides, just before entering the embryo into right and left branches which pass cephalad to unite with the hepatic circulation.

Figure 5 represents a diagrammatic sagittal view of a 5.2-cm (approximately ten week) embryo. The left umbilical vein is now shown superiorly in the cord as it courses towards the porta hepatis. The right umbilical vein disappears before the em-

bryo is 10 mm long. The left vein remains patent until shortly after birth, at which time it becomes occluded and forms the ligamentum teres of the liver. The paired umbilical arteries are seen inferiorly just above and on either side of the allantoi. On entering the abdomen they pass inferiorly on the deep surface of the anterior abdominal wall on either side of the bladder to join their parent trunk the aorta. After birth the umbilical arteries become occluded as far proximal as that portion which becomes the internal iliac artery. The fibrous cords representing the remains of the impervious portion are referred to as the lateral umbilical ligaments in the adult.

Considerable liberty has been taken in this diagram in reference to the omphalomesenteric duct. Normally this duct disappears when the embryo is between 4 and 12 mm. in length, though its vessels persist long after this. At the age indicated, the bowel has already entered the extraembryonic coelom undergone its rotation and recently re-entered the intraembryonic coelom (peritoneal cavity) and normally no vestige of the duct remains. It is depicted in Figure 5 as a patent tube arising from the terminal small bowel, extending to the umbilicus and passing toward the vestigial yolk sac in the extraembryonic coelom. This has been done so that its congenital malformation may be more easily understood.

The allantoi, as shown in Figure 5 has differentiated in its most caudal portion into the bladder. In the well-differentiated embryo its intra abdominal portion, extending from the apex of the bladder to the umbilicus, is referred to as the urachus. Even in normal embryos the obliteration of the allantoi and the urachus is an irregular phenomenon and sections at a wide variety of ages may still demonstrate patent segments.

THE ABDOMINAL WALL

Little has been added to our knowledge of the embryology of the abdominal wall since the original researches of Bardeen and Lewis, reported in 1901.

The muscles of the abdominal wall are segmental in origin, arising from the lower thoracic and upper lumbar somites. They arise by differentiation of cells of the myotome (muscle plate) which multiply and, in the case of the anterolateral abdominal muscles, migrate into the somatopleuræ. Here the muscle fibers take up a longi-

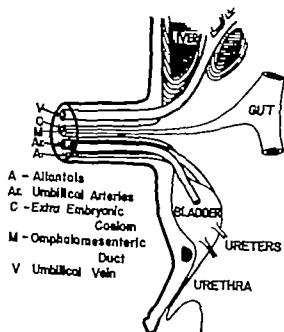


Figure 5 Diagrammatic sagittal section of 5.2-cm. (approximately ten-week) human embryo.

and yields far more information than all laboratory investigations

Examination of the abdominal wall is of the utmost importance in initially establishing a diagnosis and repeated examination of the abdomen enables the physician to follow the course of the pathologic process. It should be emphasized that the pathology of disease is a continuing process and that the clinical findings elicited on abdominal examination vary with the stage of the process present in the abdominal wall or peritoneal cavity.

THE ABDOMINAL WALL

EMBRYOLOGY

THE UMBILICAL REGION

The embryology of the umbilical region has been beautifully illustrated by Brodel and described by Cullen in the latter's classic book on the umbilicus and its diseases. Figures 1 to 5 illustrate diagrammatically the essential features in this process.

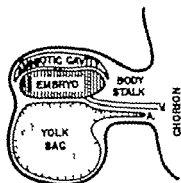


Figure 1 Diagrammatic representation of 0.7-mm. human embryo; A, allantois; V, vascular pedicle.

Figure 1 represents a human embryo 0.7 mm. long. As can be seen, the embryo proper is capped dorsally by its amniotic cavity while it sits ventrally on its relatively large yolk sac. This entire structure is attached to the chorion and thence to the uterine wall by its broad body stalk. The body stalk is seen to contain the fetal vascular pedicle (V) made up of the paired umbilical arteries and umbilical vein and, in addition, the allantois (A) which is a diverticulum of the yolk sac.

By the time the embryo reaches 17 mm. long (Fig. 2) its cephalic and caudal extremities have curled ventrally pinching off the most dorsal portion of the yolk sac which now takes a form indicative of its future development into the gastrointestinal

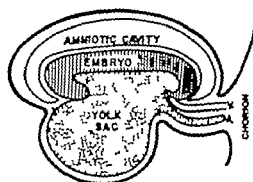


Figure 2. Diagrammatic representation of 1.7-mm. human embryo; A, allantois; V, vascular pedicle.

tract. Note how the allantois (A) arises from that portion of the yolk sac which is taken into the embryo so that one may now refer to this structure as arising from the primitive hindgut. It is of importance, for understanding of future events, to note at this point the depression which is evident at the neck of the yolk sac (N) and which, as a result of further development, is to contribute the coelom.

In the 3.5-mm. embryo (Fig. 3) a stage has been reached at which one may describe an umbilical cord. Enlargement of the amniotic cavity has now compressed the elongated body stalk and the elongated yolk sac stalk into a short compact structure, the anlage of the umbilical cord. The yolk sac stalk may now properly be referred to as the omphalomesenteric (vitelline) duct (M) as it lies free in the extraembryonic coelom, or exocoelom.

The structures in the cord at the 3.5-mm.

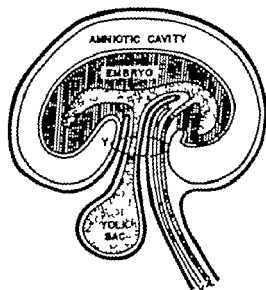


Figure 3. Diagrammatic representation of 3.5-mm. human embryo; A, allantois; M, omphalomesenteric duct; V, vascular pedicle.

curve, commonly referred to as the linea semilunaris of Spiegel. It is to be noted that in the upper abdomen, muscle fibers of the transversus are found medial to the lateral border of the rectus where they contribute to the posterior rectus sheath.

The rectus sheath itself requires little comment. The formation of the sheath superior to the linea semicircularis of Douglas is depicted diagrammatically in the left hand portion of Figure 8. It may be seen that the aponeurosis of internal oblique splits into two lamellae which enclose respectively the anterior and posterior surfaces of the rectus and are joined in the former instance by the aponeurosis of external oblique and, in the latter by the transversus abdominis or its aponeurosis. A feature of some importance surgically is the fact that the aponeurosis of external oblique fuses with the anterior rectus sheath 2 to 3 cm medial to the lateral margin of the rectus. This fusion allows incision of the deeper of the two leaves of the anterior sheath without disruption of the entire sheath when advancement of portions of the aponeurosis is used in herniorrhaphy. Below the linea semicircularis, which is usually situated about midway between the symphysis pubis and the navel the aponeuroses of all three anterolateral muscles pass anterior to the rectus muscle with the result that no posterior sheath exists and the rectus lies on the transversalis fascia. The decussation of the anterior and posterior rectus sheaths in the ventral midline forms the linea alba.

The structures superficial to the myoaponeurotic layer consist of the skin, superficial fascia and deep fascia, from without in. The skin of this area requires little special comment. Cosmetic considerations are seldom of paramount importance in the placement of abdominal incisions but where they are, the long axis of the incision should parallel the tension lines of Langer. These are illustrated in Figure 8.

The superficial fascia of the anterior abdominal wall has a characteristic feature in that the fibrous stroma which supports the fat cells is condensed, in its inferior portion, to form a rather well-defined fibrous layer. This layer replaces the soft fatty tissue in the deepest portion of the superficial fascia and is referred to as Scarpa's fascia. In children it has occasionally been mistaken by the uninitiated for the aponeurosis of the external oblique muscle during herniorrhaphy. Many surgeons repair it as a sep-

arate layer in lower abdominal wound closure.

The deep fascia of the anterior abdominal wall has no peculiar features save that while it is readily demonstrable over the muscular portion of the external oblique it becomes fused with and inseparable from the aponeurotic portion of this muscle.

The structures deep to the myoaponeurotic layer consist of the transversalis fascia, the extraperitoneal fat and the peritoneum from without in. The transversalis fascia is characterized by its firm attachment medially to the posterior rectus sheath, its loose attachment to the transversus abdominis muscle and its uninterrupted continuity posteriorly with the fascia over the quadratus lumborum and psoas muscles. Superiorly it is continuous with the fascia on the inferior surface of the diaphragm and inferiorly with that on the visceral surface of the muscle lining the pelvic cavity.

The distribution of the nerves of the abdominal wall is illustrated in Figure 9. The lower five intercostal nerves and subcostal nerve can be seen to pass downward and medially in the plane between the internal oblique and transversus muscles. They pierce the posterior leaflet of the aponeurosis of the internal oblique muscle at the lateral margin of the rectus sheath, continue their course posterior to the rectus muscle and terminate by supplying branches to this muscle and the overlying skin. The distribution of the iliohypogastric and ilioinguinal nerves from the first lumbar segment is

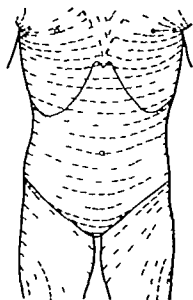


Figure 8 The tension lines of the skin of the ventral surface of the trunk.

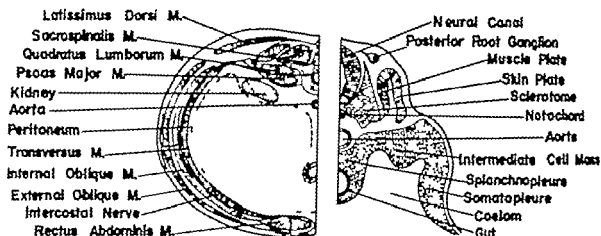


Figure 6 Left half transverse section showing the structures at the level of the first lumbar segment in the adult.

Right half corresponding section showing diagrammatically the structures of an embryo at three weeks.

tudinal direction, extending between the septa which separate the segments. With further development, muscle fibers of adjacent segments fuse, undergo lamination into layers and finally the fibers of the various laminae modify their direction to conform with the final pattern as seen in the adult. The final step in this process is the fibrous replacement of certain areas of the muscles with formation of their respective aponeuroses. Reference to Figure 6 indicates the source of these muscle fibers.

The basic vasculature of the abdominal wall, too, is derived from the segmental vessels. The arteries, for example, arise in pairs from the aorta, contribute a branch to the epaxial muscles, spine and skin of that segment and then course ventrally in the somatopleure (body wall) to anastomose with the corresponding paired ventral longitudinal vessels (superior and inferior epigastric). The venous pattern is similar as is, in essence, the pattern of the peripheral nerves though no ventral longitudinal nerve completes the analogy.

ANATOMY

In this discussion there are certain features of the anatomy of the abdominal wall which warrant brief review.

The anterior abdominal wall may be looked upon as a laminated structure of which the middle or myoaponeurotic layer functions as the essential supporting framework. This layer is composed of the anteriorly placed rectus muscles and the laterally situated flat muscles of the abdominal wall, with their aponeuroses. The fibers of the paired recti run longitudinally from the crest of the pubis to their insertion into the

cartilages of the fifth, sixth and seventh ribs. Lateral to the recti, and forming what are commonly referred to as the muscles of the anterolateral abdominal wall, are the external oblique, internal oblique and transversus muscles in that order from without in. The origin of these muscles and the direction of their fibers needs no comment here but the point at which their muscle fibers become aponeurotic is a feature of some importance, which is seldom referred to in anatomic texts in the English language. Reference to Figure 7 indicates that the line of transition from muscular to aponeurotic fibers in the case of the external oblique may be represented by a line dropped vertically from the tip of the ninth costal cartilage to join the lateral portion of a line drawn from the anterior superior iliac spine to the umbilicus. In the case of the transversus, the line of transition is indicated by a sigmoid

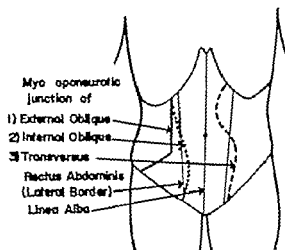


Figure 7 Myoaponeurotic junctions of the anterolateral abdominal muscles.

sions of the urinary tract are very common and considerable discussion centers around which of the two lesions is the primary one. Congenital malformations of the gastrointestinal tract, mainly in the form of malrotation occur in at least 20 per cent of cases. Respiratory and urinary infections have been the most common fatal complications. Treatment consists of external support to the abdominal wall early surgical correction of associated urinary and gastrointestinal abnormalities and general measures directed toward the maintenance of an optimal nutritional state and the prevention of complicating infections.

ABNORMALITIES IN THE REGION OF THE UMBILICUS

Omphalomesenteric Duct Anomalies (Fig 11) While Meckel's diverticulum is by far the most common lesion in this class, occurring in some 2 to 4 per cent of persons, it is dealt with in the chapter on congenital lesions of the gastrointestinal tract.

Instances of completely patent omphalo-

mesenteric duct are extremely rare. In 1916 Cullen was able to collect only forty-eight cases. However Gross reports eleven cases seen in as many years at the Boston Children's Hospital and reports are becoming more frequent in the literature. The diagnosis should be suspected in a newborn infant presenting with a reddish moist pouting collection of intestinal mucosa at the navel. The intestinal origin of the tissue as opposed to the more common umbilical granulation, may be suspected by the clinical demonstration of the mucoid nature of its secretion. Intestinal mucosa may of course also occur at the navel in association with umbilical polyps and umbilical sinuses, but these three can usually be differentiated by judicious probing and by means of x ray examination following Lipiodol injection of the sinus if one exists. The diagnosis is immediately confirmed if on crying or straining, the infant is observed to pass gas or discharge fecal material from the navel. Soutar and his associates warn against complacency in those instances in which normal gastrointestinal tract function appears to exist in the presence of a completely patent omphalomesenteric duct. At least thirty seven cases of ileal prolapse are recorded and this may occur suddenly and rapidly endanger life. The degrees of ileal prolapse are illustrated in Figure 12. The treatment consists of complete excision of the umbilicus and the tract. The ileum is closed transversely following longitudinal excision of the tract from its wall. When the operation is

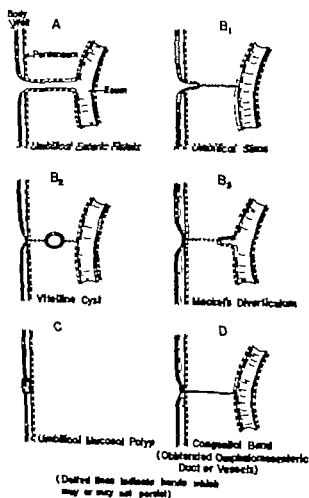


Figure 11 Congenital omphalomesenteric duct anomalies.

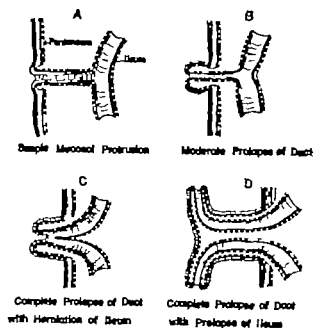


Figure 12. Degrees of prolapse associated with completely patent omphalomesenteric duct.

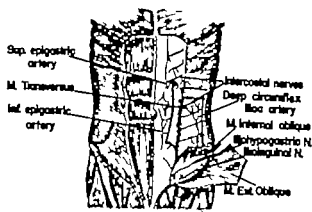


Figure 9 The anterior abdominal wall. In the right half of the figure the rectus muscle has been removed to show the structures lying on the posterior rectus sheath and the external oblique muscle has been reflected downward and a window has been opened in the internal oblique to show the intercostal neurovascular bundles.

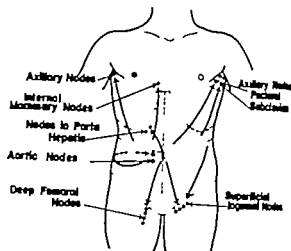


Figure 10 Lymphatic drainage of the abdominal wall. Right half drainage of superficial structures. Left half drainage of deep structures.

slightly different. As illustrated, they pierce the internal oblique muscle just medial to the anterior superior spine and thus lie in the plane between the internal oblique muscle and the aponeurosis of the external oblique, in which situation they are displayed at herniorrhaphy.

The segmental arteries, the posterior intercostal and lumbar branches of the aorta, follow the distribution of the corresponding nerves and terminate in anastomoses deep to the rectus muscle with the superior and inferior epigastric arteries. In their course they anastomose with the ascending branch of the deep circumflex iliac artery deep to the internal oblique muscle. The veins of the abdominal wall course with their corresponding arteries.

The lymphatic drainage of the abdominal wall is of importance with regard to the spread of infections and malignant disease. It is illustrated diagrammatically in Figure 10. On the right hand side of the figure the superficial drainage is shown. A line drawn around the body from just above the umbilicus to the disk between the second and third lumbar vertebrae, separates the superior from the inferior watershed. The medial portion of the superior watershed drains into the pectoral group and the lateral portion into the subscapular group of the axillary nodes. The inferior watershed, including the integumentum of the umbilicus, drains into the superficial inguinal nodes. There are no significant anastomoses between the superficial lymphatics of the left and right sides of the abdominal wall except at the umbilicus.

The lymphatic drainage of the deep structures of the abdominal wall is shown on the left half of the figure. The lymphatics are seen to course to the internal mammary, the axillary, the abdominal aortic and the deep femoral nodes from fairly well-defined areas of the wall. Again the unilaterality of the lymphatic drainage is preserved except for the area immediately around the umbilicus. The umbilical lymphatics communicate freely with channels which pass to all four drainage areas bilaterally and also by means of lymphatics traveling along the round ligament of the liver drainage may be to nodes in the porta hepatis.

CONGENITAL ABNORMALITIES ABNORMALITIES OF THE MUSCLES OF THE ABDOMINAL WALL

Agenesis or absence of the muscles of the abdominal wall is a rare abnormality and, even in the cases reported, extreme deficiency rather than absence would appear to be a more appropriate term. Baxter reports a ratio of about one case of agenesis of the muscles of the abdominal wall to seventeen cases of the relatively more common anomaly affecting the pectoral muscle group. Silverman and Huang were able to collect only forty-eight cases which had appeared in the literature up to 1950. They note that the transversus abdominis, the portion of the rectus abdominis below the navel, the internal oblique, the external oblique and the rectus abdominis above the navel are affected in decreasing order of frequency. An interesting sex incidence is recorded in that only two of forty five acceptable cases occurred in female children. Associated le-

should be restricted to incision, drainage and loose packing of the cavity with gauze. Frequently as a result of the infection, the transitional epithelium of the wall of the cyst is destroyed and the simple drainage procedure results in permanent cure.

Urachal bladder diverticula are considered in connection with the urinary tract.

Vascular Anomalies. Vascular anomalies involving the omphalomesenteric vessels are rare and of little clinical significance. Aberrations in the normal fate of the umbilical vessels are apparently rare if one excludes the possibility that portal vein obstruction may be due to extension into that vessel of the obliterative process that is normally limited to the umbilical vein and ductus venosus. Anomalies of the urachal vessels are apparently of little or no clinical significance.

Somatic Anomalies. Omphalocele, umbilical hernia and extrophy of the urinary bladder are somatic anomalies which occur frequently enough to require serious consideration for surgical treatment.

Endometriosis of the umbilicus is rare. It usually presents as a small mass at the umbilicus which enlarges and becomes tender at the time of the menstrual period. External bleeding may also occur. Treatment is by simple excision. Coexistent pelvic endometriosis will require separate treatment.

INFECTIONS OF THE ABDOMINAL WALL

INFECTIONS OF THE UMBILICUS

Omphalitis of the Newborn. While this condition is much less common now than twenty-five or more years ago, it may be a serious lesion and therefore demands prompt recognition and treatment. It usually arises as a result of contamination of the cord at the time of section or during subsequent dressing of the umbilicus. The common organisms are the *Staphylococcus aureus* and the hemolytic streptococcus. The widespread lymphatic drainage and the persistence of patent vascular channels in this area account for the rapid dissemination of the infection.

Locally the condition is manifest by redness, heat, swelling and tenderness which may spread centrifugally from the navel with alarming rapidity. Usually pus exudes from the folds of the umbilicus. Occasionally widespread sloughing of skin occurs. Blood stream invasion may occur along the incompletely obliterated umbilical vein or umbilical arteries with signs of septicemia. Spread sometimes occurs to the adjacent peritoneum with signs of fulminating peritonitis.

Treatment of the omphalitis must be initiated promptly with antibiotics, local hot moist compresses and surgical incision of any purulent collections. The localized type of omphalitis usually responds rapidly to treatment, but once septicemia or peritonitis has occurred the outlook is grave.

Omphalitis in the Adult. Unlike neonatal omphalitis, umbilical infections in the adult tend to pursue a relatively benign and chronic course. They are commonly seen in office practice but seldom require hospital care. The one common etiologic factor in almost all cases appears to be neglect of personal hygiene in this area. Congenital aberrations in the depth and configuration of the umbilicus may contribute by making cleansing of the area difficult.

The usual complaints are of tenderness in or around the navel and the presence of a seropurulent discharge with an offensive odor. Examination reveals the base of the umbilicus thrown up into a cluster of red, moist, swollen, angry looking folds which excite the offensive discharge. Extension into periumbilical tissues is uncommon but may occur along with inguinal adenitis.

In the acute phase omphalitis rapidly responds to warm moist saline compresses and hydrogen peroxide instillations. Antibiotics are seldom indicated. Once the acute symptoms have subsided, the patient should be instructed to wash the umbilicus carefully while bathing and dry it with equal care afterward. Gentle cleansing with alcohol dipped absorbent cotton on the end of an applicator is a useful means of eliminating moisture and excretion in deep clefts.

INFECTIONS OF THE ABDOMINAL WALL

Infections of the abdominal wall proper are essentially similar to superficial infections occurring elsewhere in the body. Progressive bacterial synergistic gangrene and other relatively rare infections of this area may produce serious postoperative complications.

TRAUMA TO THE ABDOMINAL WALL

Injuries to the abdominal wall often tax the diagnostic acumen of the examiner. The major problem is to differentiate those lesions limited to the wall from those traumatic intra-abdominal lesions which may rapidly endanger life and for which immediate surgical intervention is mandatory.

Wounds of the abdominal wall, as opposed to blunt trauma where no open wound exists, may be considered under two broad

carried out before ileal prolapse with vascular embarrassment has occurred, the mortality is minimal.

Umbilical sinuses and polyps are treated by excision. Vitelline cysts are usually asymptomatic unless obstruction occurs around the congenital band which joins them to the ileum or to the navel. Small cysts situated close to the navel or within the abdominal wall, or larger cysts occurring more proximally along the duct, may be palpable. The only satisfactory treatment is excision.

Congenital bands of either omphalomesenteric duct or vascular origin, are of importance only as an etiologic factor in intestinal obstruction. Bremer makes the interesting observation that a band derived from the obliterated omphalomesenteric artery corresponds in position to that shown for the congenital band in D of Figure 11, while that derived from the corresponding vein runs, not to the antimesenteric border of the bowel, but rather to the base of the mesentery at a more proximal level, or to the porta hepatis.

Urachal Anomalies (Fig 13) Cullen in his book briefly presents some sixty cases of congenital completely patent urachus reported in the literature up to 1916. Gross was able to report on ten cases observed at the Boston Children's Hospital. The condition is characterized clinically by the intermittent discharge of urine from the navel, the quantity and the frequency depending on the caliber of the fistula and the presence or absence of some degree of obstruction of the lower urinary tract. A small granular tumor may mark the site of the ostium at the umbilicus or the navel may appear normal on superficial examination. No symptoms apart from the discharge are associated with the anomaly unless infection occurs. Surgical excision is the only satisfactory treatment, but this should be performed only after the patency of the urethra has been confirmed. The entire tract is excised and the site of attachment to the bladder is ligated and inverted with a purse-string suture. In the absence of pre-existing infection the procedure can usually be accomplished extraperitoneally with minimal risk.

In the so-called acquired type of completely patent urachus, no abnormality is apparent at birth and only at some later date does intermittent discharge of urine occur. Such cases are almost invariably associated with lower urinary obstruction which forces urine through an incompletely obliterated urachal channel. Treatment is

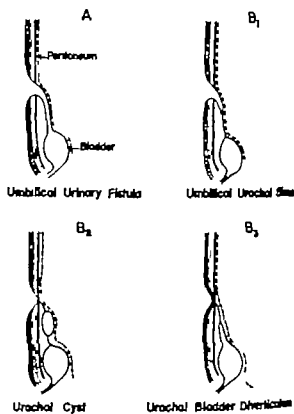


Figure 13. Congenital urachal anomalies.

excision following correction of the obstructing lesion. The acquired type of anomaly is more common than the congenital type.

Umbilical urachal sinuses present no additional features except that the discharge is minimal and watery and is not urine. Treatment consists of surgical excision.

Urachal cysts are the most common urachal abnormality. If uninfected they are usually asymptomatic and, when small, are recognized only at surgery or autopsy as an incidental finding. Larger cysts may be apparent to the patient as a subumbilical fullness or swelling or may be discovered incidentally by the surgeon in the course of abdominal examination. Infection is common and this is the usual method of clinical presentation. The picture then, will be one of an acute inflammation deep in the subumbilical abdominal wall. In cases in which the cyst is not infected, extraperitoneal excision is the treatment of choice and usually presents no difficulties unless the cyst is of large size. It is wise in such cases to demonstrate the cord, which almost invariably runs from the cyst to the bladder and to deal with it as though it were patent by the method previously referred to. This precaution is also desirable in cases of umbilical urachal sinus. In the case of infected urachal cysts, excision without peritoneal contamination is virtually impossible and treatment

this finding after abdominal trauma is of extreme importance. It may arise, on the one hand, from a relatively benign injury to the abdominal wall or on the other from a grave intraperitoneal insult which will be rapidly fatal if surgery is withheld. It should be possible in every case to differentiate these two on a basis of complete and careful abdominal examination.

Abdominal wall rigidity as an early manifestation of purely thoracic wounds is now well recognized. Frequently the rigidity which is usually ipsilateral but may be bilateral, is associated with subcostal tenderness of such intensity in the early hours after injury that one is tempted to carry out a laparotomy with a diagnosis of ruptured intra-abdominal viscus. The knowledge that this syndrome may exist, along with the absence of signs of peritoneal contamination, should enable one to avoid needless laparotomy. The abdominal findings rarely persist over forty-eight to seventy-two hours.

The converse situation is frequently observed, namely unilateral restriction of movement of the thoracic cage and diminished air entry into the base of the lung on that side, following injury restricted to the abdominal wall. Percussion of the chest along with radiologic examination will quickly clarify the situation.

HEMATOMA OF THE RECTUS SHEATH

In recent years this uncommon condition has aroused much interest because of the fact that it may mimic acute intra-abdominal disease. It is seldom diagnosed preoperatively because it is not considered. In approximately 90 per cent of cases the hematoma is situated below the navel and it lies to the right of the midline about twice as often as it does to the left. The frequent occurrence of the hematoma below the semicircular line of Douglas, causing peritoneal irritation, is considered to account for the gastrointestinal manifestations which are often seen. It is seldom possible to demonstrate the exact site of hemorrhage, but the inferior epigastric vein is more commonly the source than the artery.

Insofar as etiology is concerned, there is general agreement that the condition is more frequent during pregnancy. Even in this group it is usually possible to elicit a history of indirect trauma or upper respiratory infection with cough as the immediate cause of the hemorrhage. Apart from pregnancy the cases may be broadly classified into those associated with

1. Trauma—either direct or indirect, of which the latter is far more common.
2. Infections and Debilitating Disease—in which case a severe paroxysm of coughing is often the immediate cause of the hemorrhage.
3. Degenerative Vascular Disease Blood Dyscrasias and Abnormalities of Coagulation—which includes an increasing number of cases ascribed to anticoagulant therapy.
4. The Postoperative Patient—in whom the injudicious positioning of rectus muscle retractors or retention sutures is usually the cause.
5. Idiopathic—this latter group implies that the lesion can occur spontaneously without associated disease or injury. This appears most unlikely and it is probable that one of the above factors is present, though overlooked, in cases so classified.

The symptomatology is classical. Following trauma, which may be mild or severe, there is usually a rather sudden onset of excruciating abdominal pain. In cases in which the bleeding occurs slowly the pain will be more gradual in onset and of less severity. It is usually localized to the site of rupture but may be generalized. Nausea and vomiting are relatively common and constipation may occur. Characteristically after a period of increased pain, there is a gradual improvement which leaves in its wake a persistent dull ache.

Apart from the occasional patient who appears acutely ill and shocked, the significant findings are limited to the abdomen. Here one finds a hard exquisitely tender mass in one or the other rectus, which may be misinterpreted as localized muscle spasm. If one demonstrates that the mass is restricted to the confines of the rectus sheath and that tenderness and rigidity in the adjacent lateral abdominal muscles are lacking, the diagnosis is supported. Fothergill has described a sign which assists in differentiating abdominal wall masses from those arising within the peritoneal cavity. When the recti are contracted, as in attempting to sit up a mass situated in the wall may still be felt and is fixed, while an intra-abdominal mass is no longer easily palpable. An additional late sign, which has been observed in a number of cases, is the appearance of ecchymosis about the umbilicus which may extend down into the scrotum or perineum.

The hematoma is usually accompanied by a mild elevation of temperature and pulse

headings. By convention, involvement of the peritoneum is considered the dividing line between these two types of injury.

Nonpenetrating wounds involve only the abdominal wall proper. Like wounds elsewhere they show all gradations from clean incised wounds to gaping defects with much loss of tissue. In depth, they vary from disruption of skin continuity alone to involvement of all layers including muscle. Apart from those wounds associated with considerable loss of tissue, where some type of plastic procedure may be necessary, the treatment of nonpenetrating abdominal wounds is similar to that of wounds elsewhere in the body and follows the principles laid down for the surgical care of soft tissues.

Penetrating wounds constitute an entirely different problem. Such injuries occur mainly as a result of gunshot and stab wounds, although almost every conceivable foreign body has at some time been recorded as the traumatizing agent. Not all gunshot and stab wounds are, of course, penetrating. Loria, reviewing a series of 4341 such cases encountered at the New Orleans Charity Hospital, reports approximately two-thirds to be penetrating and one-third to be nonpenetrating. There is rather universal agreement, however, that unless such a puncture type of wound is very obviously nonpenetrating it should receive prompt surgical attention. In penetrating trauma the injury to the abdominal wall is usually of minor importance and the demonstration and correction of the intra-abdominal component are the prime considerations.

Firstly it is usually unwise to attempt to probe the wound tract. If the depth of the wound cannot be determined it is far better as pointed out by Gaston and Mullolland, to examine the parietal peritoneum through a planned, initially small incision adjacent to the wound. This incision may be closed, if the peritoneum is intact, or rapidly extended to give wide access to the abdominal cavity if peritoneal penetration has occurred. Secondly where wounds of entrance and exit exist, the incision is selected which will give the best access to the anticipated intraperitoneal damage and no attempt should be made to incorporate such wounds into the incision. Thirdly on completion of the intraperitoneal procedure and after consideration of the condition of the patient, the puncture wounds may be either simply cleansed and dressed or excised and closed,

depending on their character and degree of contamination.

Blunt trauma to the abdomen is linked in the minds of surgeons with the multitude of puzzling intra-abdominal lesions which so frequently accompany this form of injury and little attention is usually given to isolated abdominal wall lesions which may occur. Contusion and hematomas of the subcutaneous tissue and muscle layers are common but seldom are of serious significance or require more than symptomatic treatment. Their main interest to us stems from the pain and muscle spasm which they produce and which must be differentiated from similar symptoms and signs produced reflexly by intra-abdominal injury. Occasionally as a result of blunt trauma, subcutaneous rupture of the muscles of the abdominal wall occurs without disruption of the skin, as recorded by Jamieson.

SPASM OF THE MUSCLES OF THE ABDOMINAL WALL

Spasm of muscles of the abdominal wall particularly the rectus abdominis is a common finding on examination. A relatively simple classification of the causes of abdominal wall spasm would include

- I. Central or Cerebral Origin.
 - a. Organic, as seen in spastic paraplegia.
 - b. Psychogenic, as seen in the nervous patient, the patient anticipating some painful experience or in psychopathic drug addiction.
- II Spinal Cord Origin, as seen in those diseases characterized by irritation of spinal cord neurons, as for example tabes dorsalis.
- III Thoracic Nerve Trunk Origin, as seen in pleurisy infections of the chest wall, chest and spinal column injury, epidemic pleurodynia and severe alcoholic neuritis.
- IV Reflex Origin, as seen when the peritoneum is irritated by infection, blood or foreign material or when juxtapertoneal organs are the origin of extremely painful stimuli, as in renal colic.
- V Local Origin as seen in local trauma to or infection of the abdominal wall.

While peritonitis, in its broadest sense, is by far the most common cause of true abdominal wall rigidity the interpretation of

domen. Less commonly a vague feeling of pressure on the bladder or bizarre abdominal pains may cause the patient to seek advice. On examination, a mass is usually palpated deep to the skin and unattached to it. Characteristically it is firm, smooth and discrete. Bouchacourt's sign which is similar to that of Fothergill but applies to intramural masses situated anywhere in the anterior abdominal wall, is helpful in localizing the mass to the muscular layer of the abdominal wall. The rectus abdominis is the muscle most commonly affected, but the infiltrative nature of the tumor frequently results in multiple muscle involvement.

The gross pathologic features of the tumor are its denseness and hardness, its glistening white or pinkish color and its apparent infiltrative character. Microscopically it varies from an acellular fibroma to a low-grade cellular fibrosarcoma and the inclusion at its periphery of engulfed and sequestered degenerating muscle fibers is characteristic.

Desmoid tumors are clinically benign and metastases do not occur. Local recurrence is the rule, unless excision has been complete. Treatment is by wide surgical resection which, because of the large size of some of these tumors, often results in defects which require plastic procedures for closure. The place of radiation therapy in the management of these tumors is controversial and no opinion as to its value is possible at this time.

MALIGNANT TUMORS

The malignant tumors constituted 38 per cent of all neoplasms and, of this group it is interesting to note that secondary malignant tumors were as common as primary malignant growths. This is in marked contrast to other soft tissue areas where secondary deposits are extremely rare. The remainder of this group consisted of malignant lymphomas. These latter cases were instances in which the initial presentation of the disease took the form of an abdominal wall mass.

Primary Malignant Tumors. Of this group approximately one half were sarcomas. The more common sarcomas, in order of frequency were neurogenic sarcoma, spindle cell sarcoma, synovioma and rhabdomyosarcoma. The carcinomas encountered were mainly epidermoid in type with occasional basal cell lesions and very rarely an adenocarcinoma. There were eight melanomas in the entire series.

Most of the sarcomas arose from the

deeper layers of the wall and not infrequently gave rise to blood borne metastases though lymph node involvement did not occur. As a group these sarcomas tend to be radioresistant and the treatment if treatment is possible consists of wide excision of the full thickness of the abdominal wall including the peritoneum. Synoviomas and rhabdomyosarcomas appear to have a particularly unfavorable prognosis.

Carcinoma of the skin of the abdominal wall is relatively uncommon, as compared with its occurrence elsewhere in the body. It does, however, present some interesting and rather unusual features when it occurs in this situation. In twelve of the seventeen cases in the series referred to epidermoid carcinoma arose in an area of pre-existing tissue abnormality such as an abdominal scar, an abdominal sinus or an area of radiation dermatitis. Epidermoid carcinoma, arising in normal skin, has a relatively good prognosis following total surgical removal. Superficial lymphatics are scarce in the abdominal wall and lymph node metastases are unusual. Epidermoid carcinoma arising in laparotomy scars and postoperative draining sinuses is a clinical entity with an extremely grave prognosis for while lymph node metastases are rare early extensive involvement of the peritoneum is common.

Melanomas appear to be relatively uncommon in the abdominal wall. Stewart, Hay and Varco report an incidence of 3.26 per cent in a group of ninety-two cases, while Pack and Ehrlich could report only eight cases. These lesions appear to have a predilection for the area about the umbilicus which, as we have seen in connection with the anatomy of this region, is a most undesirable situation from the standpoint of facility of lymphatic and venous spread. Melanoma carries with it a grave prognosis and since these lesions are rarely radiosensitive, radical surgical excision is the only available treatment which offers any hope of eradication of the disease. Wide local excision of the lesion is mandatory and this, at the umbilicus, includes ample excision of the full thickness of the abdominal wall and the round ligament of the liver. Block dissection of lymphatic glands in areas of apparent spread should be carried out. If the lesion is situated in a quadrant of the abdomen where its lymphatic drainage can be predicted with assurance then block dissection of the regional lymphatics should be carried out even in the absence of clinically demonstrable metastases. The principle of

and moderate polymorphonuclear leukocytosis is common, further simulating acute intra abdominal disease.

The natural history of the disease is usually one of spontaneous recovery with cessation of pain after a few days and gradual disappearance of the mass in the course of three to four weeks. Occasionally the hematoma becomes infected and forms an abscess or persists and calcifies to give rise to continuing pain. Schafer in his collected series of 101 cases makes it clear that the condition is not to be considered innocuous for he reports nine deaths seven directly attributable to the hemorrhage.

Some surgeons feel that the safest and surest treatment is to confirm the diagnosis by means of a short paramedian incision evacuate the clot and ligate the epigastric artery and vein above and below the assumed point of hemorrhage. It is our belief that most of these cases can be successfully managed conservatively. It should be pointed out that some patients will be operated upon in error with a preoperative diagnosis of appendicitis or some other intra abdominal lesion. In this group surgical control of the hemorrhage is readily carried out.

TUMORS OF THE ABDOMINAL WALL

Pack and Ehrlich, in a report dealing with 391 cases which they observed at the Memorial Hospital in New York, draw attention to the fact that tumors in this region frequently present unique problems which warrant their study as an isolated group.

BENIGN TUMORS

Benign tumors constituted 62 per cent of all neoplasms and 80 per cent of the primary neoplasms. In order of frequency the more common types of benign tumors encountered were lipomas, neuroangiomas, epithelial papillomas, fibromas, neurofibromas, keratosis and desmoid tumors. Lipomas constituting 20 per cent of the benign neoplasms were the most common tumor encountered in the entire series. The treatment of these lesions is similar to that recommended in other situations and presents no special problem. Lipomas, papillomas, fibromas and keratosis are treated by simple excision. In the case of hemangiomas, we also favor surgical excision when the lesion occurs in the abdominal wall since cure is rapidly achieved and the resulting scar is of little consequence. Others favor carbon dioxide snow and radiation therapy for superficial lesions. It is interesting to note

that Scott, in a review of hemangiomas of skeletal muscle found 4.3 per cent in the muscles of the abdominal wall.

Neurofibromas of the solitary type are easily excised surgically but in over half of the cases in which this lesion presented in the abdominal wall it took the form of the plexiform von Recklinghausen type. This lesion, with its known predisposition to malignant transformation was often extensive and invasive and required wide excision, sometimes in stages.

Nevi of the abdominal wall must be treated with considerable respect. Eighteen per cent of the pigmented nevi in this series turned out to be malignant melanomas and seven out of eight melanomas encountered gave a history of chronic irritation of a pre-existing mole. It is obvious that any such lesion should be widely excised surgically especially if it presents in an area in which irritation from a belt, girdle or other clothing occurs.

DESMOID TUMORS

Desmoid tumors are essentially hard fibromas arising from the musculoaponeurotic structures of the anterior abdominal wall. Some include tumors of similar character occurring in other skeletal muscle groups under the same heading. Unlike the usual fibroma, which tends to be situated subcutaneously these tumors arising from the more deeply placed fascial and aponeurotic layers are not encapsulated. They are characteristically infiltrative and tend to attain large size. The etiology of these lesions has never been satisfactorily explained. The preponderance in the female (80 to 90 per cent) and the frequent appearance of the tumor within a year following parturition have led to the suggestion that stretching of and trauma to the abdominal wall during pregnancy or some endocrine factor associated with gestation, may be the important etiologic factor. However the occurrence of this type of tumor in a significant number of male and nulliparous female patients, as observed in the series of Pearman and Mayo, would tend to minimize the etiologic significance of pregnancy. A definite history of antecedent abdominal trauma is also rare, though in some series the occurrence of desmoid tumors in previous laparotomy scars is too frequent to be dismissed as incidental.

Clinically the patient usually presents complaining of the presence of an abdominal mass or a sensation of weight in the ab-

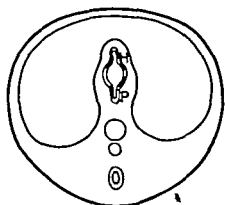


Figure 16. Cross section through that level of the gut destined to become the second part of the duodenum. There is no ventral mesentery at this level and the peritoneal cavity is a continuous space. The hepatic diverticulum (H) arises by evagination ventrally and will give rise to the ventral contribution to the pancreas and to the biliary tree and liver. The pancreatic diverticulum (P) arises by dorsal evagination and forms the major portion of the pancreas.

to two evaginations (Fig. 16) Into the ventral mesentery grows the hepatic diverticulum, from which branches the accessory pancreas. Into the dorsal mesentery grows the diverticulum which is to become the main pancreas. In the dorsal mesogastrium the spleen develops in relation to blood vessels (and not lymphatics) and then bulges into the left leaf of the mesentery (Fig. 17)

The mesenteries of the foregut shift their attachments as the stomach rotates 90 degrees on a long axis and as overgrowth takes

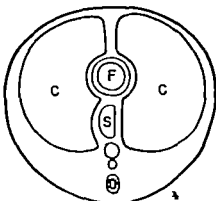


Figure 17. Cross section somewhat cranial to that shown in Figure 16. The peritoneal cavity lies on either side of that portion of the foregut (F) destined to become the stomach. The spleen arises in the dorsal mesogastrium. The hepatic diverticulum, after giving off the ventral pancreas, will grow cranially to the ventral mesogastrium and septum transversum to give rise to the liver. The pancreatic diverticulum will extend cranially in the dorsal mesogastrium and come to be dorsal to the spleen. Subsequent rotation of the stomach with its mesenteries will cause these structures to lie to the right (liver) and left (spleen and pancreas) of the stomach.

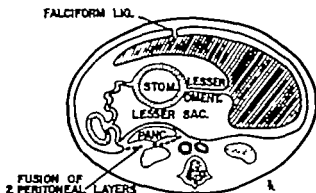


Figure 18. Cross section through a four month fetus at approximately the level shown in Figure 17. The liver and pancreas have grown cranially from their points of origin shown in Figure 16. Rotation has converted the right half of the peritoneal cavity shown in Figure 17 into the lesser sac. It can be seen that fundamentally the spleen and pancreas lie in the dorsal mesentery of the stomach.

place on the greater curvature side (Fig. 18) The ventral mesentery bearing the hepatic diverticulum (common bile duct) shifts to the right and cranially to become the lesser omentum.

The rotation of the foregut is accompanied by a shift in the attachments of its dorsal mesentery bearing the spleen and a major portion of the pancreas. The dorsal mesogastrium balloons to the left and caudally. The cranial portion adheres to the posterior wall of the peritoneal cavity and the adjacent layers of mesogastrium and parietal peritoneum disappear. Thus, the pancreas takes its permanent retroperitoneal position and the cranial portion of the dorsal mesogastrium takes its attachment from the left posterior body wall in the form of the gastrosplenic and lienorenal ligaments, which are really a single mesentery with the spleen protruding from the left anterior leaf.

The caudal portion of the dorsal mesogastrium overlaps the transverse mesocolon and fuses with it, so that the mesogastrium appears to take origin from the transverse colon itself. Considerable further overgrowth of the dorsal mesogastrium between the greater curvature of the stomach and transverse colon produces an apron of redundant tissue which descends into the lower abdomen and then doubles back on itself to form the greater omentum (Fig. 19)

The "physiologic herniation of the midgut" and the process of intestinal rotation should be reviewed frequently because an appreciation of these processes is necessary in order to understand the retroperitoneal position of the duodenum, the obliquity of

excision of the primary lesion with dissection of the regional lymphatic nodes in continuity can frequently be effectively employed in the case of the abdominal wall.

Secondary Malignant Tumors. Reference has already been made to the relatively frequent occurrence of secondary malignant spread to the abdominal wall in contrast to the infrequent occurrence of such spread to other soft tissue areas. The source of the metastatic deposit is most commonly a carcinoma (85 per cent) and in the series of Pack and Ehrlich the common sources in order of frequency were the ovary, stomach, uterus, bronchus, kidney, breast and sigmoid colon. Metastatic carcinoma of the abdominal wall most commonly occurs in the region of the umbilicus. Cullen has collected a series of such cases which would implicate the stomach rather than the ovary as the most common source. An interesting story is often told in this connection. Sister Joseph, who was in charge of St. Mary's Hospital in Rochester and who functioned as the able assistant to Dr. William Mayo in the early days of the Mayo Clinic, is said to have initially drawn to Dr. Mayo's attention her observation that, in the preparation of gastric cases for surgery, she occasionally noted a very firm mass at the patient's umbilicus. She further observed that patients so afflicted did particularly poorly following operation. This astute observation is often acknowledged by applying the term "Sister Joseph's sign" to the demonstration of umbilical metastatic deposits in cases of intra-abdominal malignancy.

The presence of secondary malignant deposits in the abdominal wall, with one notable exception, is a sign of advanced disease and inoperability. The one exception is the implantation carcinoma which is occasionally seen in the scars of operations carried out for malignant disease. These lesions surprisingly do not tend to invade the peritoneal cavity and while lymph node metastases may occur, wide surgical excision of the implanted lesion, along with block dissection of involved nodes, may be rewarded by long term survival.

THE PERITONEUM EMBRYOLOGY*

The primitive coelom is partially divided by the septum transversum, or future dia-

phragm. Strict regard for chronology has been avoided in this description in order that the development of each structure might be treated as a unit.

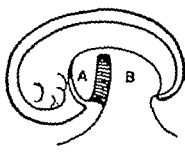


Figure 14. Parasagittal section of an embryo at four weeks. The thick septum transversum divides the coelom into the pericardial cavity (A) and the peritoneal cavity (B). The two cavities communicate over the dorsal aspect of the septum transversum by means of the pleural canals.

phragm, into a pericardial cavity and a peritoneal cavity in the fourth week of intrauterine life. For a time the two cavities communicate over the dorsal edge of the thick septum transversum by the paired pleural canals (Fig. 14). By the seventh week the peritoneal cavity is completely separated from the pleural canals by the pleural peritoneal membranes.

Initially the peritoneal cavity is separated into right and left halves by the primitive gut and its mesentery. The mesentery is designated as ventral or dorsal, according to its relationship to the gut (Fig. 15). The ventral mesentery then disappears except for the lesser omentum and falciform ligament, its contributions to the diaphragm and the suspensory ligament of the bladder. With resorption of the greater part of the ventral mesentery the coelom becomes a single cavity.

That portion of the gut which will become the second part of the duodenum gives rise

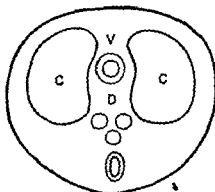
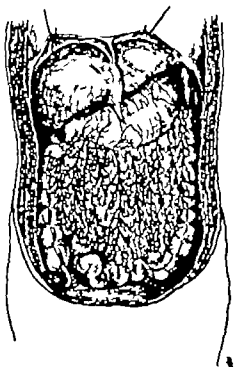


Figure 15. Cross section through the level of the foregut. The peritoneal portion of the coelom (C) is initially divided into two cavities by the gut, the transient ventral mesentery (V) and the dorsal mesentery (D).



Figures 20 through 23: The peritoneal cavity and retroperitoneal space, viewed from the ventral aspect, as successive structures are removed:

Figure 20 View of the abdominal viscera following removal of the anterior abdominal wall. The two halves of the diaphragm are elevated to display the right anterior-superior and the left subhepatic spaces, separated by the falciform ligament. The greater omentum is seen as if "hanging down" from the greater curvature of the stomach.

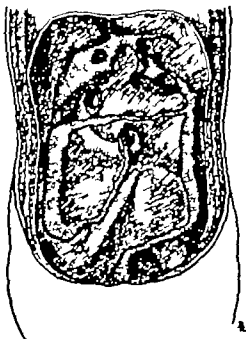


Figure 22 View of the posterior abdominal wall following removal of the viscera, except for the duodenum. The obliquity of the root of the mesentery is shown. The mesentery measures only 15 cm. in length at its attachment to posterior parietal peritoneum and 700 cm. in length at its attachment to small bowel. The paracolic gutters, communicating with the pelvis and subphrenic regions, are shown. The right gutter is particularly important in the spread of peritoneal infection.

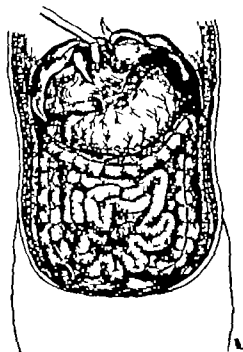


Figure 21 View of the abdominal viscera as seen in Figure 20, except that the greater omentum has been removed. The liver is elevated, showing the right subhepatic and the left anterior inferior subphrenic spaces.

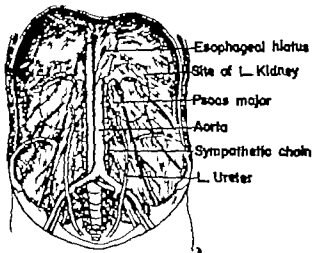


Figure 23 View of the principal retroperitoneal structures after removal of the posterior parietal peritoneum and the vessels of the large bowel. The vessels of the colon become retroperitoneal only because the dorsal mesentery of the colon fuses with posterior parietal peritoneum. The ureters are the next most anterior structure of the retroperitoneum.

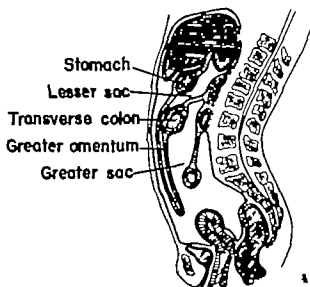


Figure 19 Diagrammatic sagittal section through a fetus, showing the relations of the greater and lesser peritoneal sacs. The two layers of the greater omentum have not yet fused. Basically the greater omentum is the dorsal mesentery of the stomach which has fused with the transverse mesocolon.

the root of the mesentery, and the manner in which the mesenteries of the descending colon and ascending colon become fused with the posterior parietal peritoneum.

ANATOMY

Gross Anatomy The peritoneal cavity was mentioned in the Papyrus Ebers some 3500 years ago but it was not thoroughly described until 1730 when James Douglas of Edinburgh published a lucid account which has not been appreciably improved upon to this day.

The peritoneum is a serous membrane which lines the peritoneal cavity and invests a number of abdominal structures. Except for the openings of the fallopian tubes, the peritoneum is a completely closed sac. In a strict sense, the peritoneal cavity does not contain any organs since the entire gastrointestinal tract and its derivatives are really retroperitoneal in position. It is customary to speak of those structures which are almost completely enfolded by peritoneum, such as stomach, jejunum ileum, transverse colon, sigmoid colon, appendix, cecum liver gall bladder and the spleen, as being "intrapertoneal."

That portion of the peritoneum which invests the intraperitoneal organs, and which makes up the coverings of the mesenteries is the *cisceral peritoneum*. That portion which lines the anterior lateral and posterior abdominal walls, the undersurface of

the diaphragm and the floor of the pelvis is the *parietal peritoneum*. Although these are parts of the same membrane, the distinction is of some importance in regard to differences in sensory innervation.

The peritoneum of the anterior and lateral abdominal wall is reinforced by the transversalis fascia. This fascia is intimately associated with peritoneum in some areas where it strengthens this membrane. Elsewhere it may be separated from the peritoneum by a layer of fatty and areolar tissue.

The peritoneal cavity is divided into the general peritoneal cavity or greater sac, and the lesser sac (Fig. 19). The latter has, as its only natural opening, the foramen of Winslow. It is bounded anteriorly by gastrohepatic ligament, stomach and gastrocolic ligament and by parietal peritoneum posteriorly.

Hanging from the transverse colon and covering much of the ventral aspect of the lower abdominal viscera is the greater omentum (Fig. 20). This structure has a rich vascular supply and carries a variable amount of fat.

The surface area of the peritoneum is about 2 square meters and approximates that of the skin. Unlike skin, however, the peritoneum is a highly permeable membrane and this fact has a number of important physiologic implications.

Normally the peritoneal cavity contains 75 to 100 cc. of clear straw-colored fluid, which facilitates the normal lubricating function of the membrane.

Microscopic Anatomy The peritoneum consists of a surface layer of mesothelium and a deeper loose connective tissue layer containing collagen and elastic fibers, fat cells, reticulum cells and macrophages. The mesothelium is a simple squamous cell layer. As a result of irritation, these cells may become cuboidal and enclose small cystic spaces—a reaction which the pathologist must distinguish from neoplasia.

Normal peritoneal fluid contains 2000 to 2500 cells per cu. mm. The majority of these are macrophages with some desquamated mesothelial cells and lymphocytes. There are few polymorphonuclear neutrophils or eosinophils but the number of granulocytes is greatly increased in the presence of inflammation. Peritoneal aspiration may be of value, not only to obtain fluid for culture and chemical analysis, but also because immediate study of the cells

may be *primary* or *secondary*. The most frequently encountered type is an *acute bacterial inflammation of the peritoneum* which is almost always *secondary to contamination*. This particular variety of peritonitis is usually called, simply "peritonitis" without any qualification.

Peritonitis due to contamination is common because the peritoneum is, to coin a phrase, a *serous sac* completely surrounded by pathogens. Within it is the gastrointestinal tract teeming with microorganisms. Externally the environment harbors streptococci and staphylococci. Above and below are the lungs and the pelvic organs, occasionally a source of bacteria which invade the peritoneal cavity.

HISTORICAL

Peritonitis has been well known since antiquity and death from peritonitis has been the terminal event in many disease processes.

Inability to prevent or to effectively treat peritonitis was the chief obstacle to the early development of abdominal surgery. In the pre-Listerian era, laparotomy was almost invariably followed by the development of peritonitis and this infection, once established, progressed in most cases to a fatal outcome. A considerable part of modern surgical "ritual" in abdominal surgery is directed at the prevention of infection, particularly infection of the peritoneum. This includes scrubbing the hands, use of gowns, gloves, masks and numerous other details of technique.

ACUTE BACTERIAL PERITONITIS SECONDARY TO CONTAMINATION

Incidence. In 5 to 7 per cent of all autopsies, peritonitis is the primary or a contributory cause of death. Generalized peritonitis, while no longer the overwhelming problem it once was, is still the most common cause of death following abdominal surgery.

Etiology. Maingot states that the common lesions responsible for acute secondary bacterial peritonitis are (1) appendicitis (40 per cent) (2) perforated peptic ulcer (20

per cent) (3) operative contamination (20 per cent) (4) pelvic inflammation (7 per cent) (5) intestinal obstruction and strangulation (7 per cent) (6) rupture of the intestine (2 per cent).

The species and virulence of the infecting organisms are important in determining the severity of the peritonitis.

A wide variety of *organisms* may be responsible for peritonitis and not infrequently more than one species of bacterium may be involved. It may be difficult to determine the relative pathogenicity of each organism as organisms often act synergistically to produce severe systemic effects.

The organisms frequently found in peritonitis are (1) *Escherichia coli*, (2) streptococci (both aerobic and anaerobic) (3) staphylococci, (4) pneumococci (5) Friedländer's organisms, (6) *Pseudomonas aeruginosa* (7) gonococci, (8) *Proteus vulgaris* (9) *Clostridium perfringens* and other anaerobes.

The two most common and important pathogens are *E. coli* and *Streptococcus pyogenes*. This may be of some importance, as all streptococci are sensitive to penicillin, whereas the coliforms, which usually exist concomitantly not only resist penicillin but destroy it by producing penicillinase. In a pure infection, the hemolytic streptococcus produces a highly virulent and rapidly lethal infection, whereas the coliforms tend to produce a relatively low-grade infection.

The degree of contamination is an additional factor in determining whether the infection will be rapidly walled off and controlled or whether the infection will spread and become generalized in the peritoneal cavity.

The role of bacterial toxins in producing the lethal effects of peritonitis is unsettled, but there is evidence that toxins produced by clostridia and endotoxins from lysis of coliforms play a part in the peripheral circulatory failure that marks the end stage of the disease.

Pathogenesis. It is important to appreciate the manner in which infection is disseminated in the peritoneal cavity and the way in which the peritoneum reacts to combat infection. Insight into this physiologic process enables one to assist the peritoneum and avoid interfering with its beneficial activity by misguided therapeutic efforts.

The outcome of peritonitis depends upon the number and virulence of the infecting organisms and the resistance of the host. In

It is remarkable that between 1809 and 1829 Ephraim McDowell removed eleven ovarian cysts through an abdominal approach with only one death. The feat seems incredible, but McDowell, in addition to excellent training and considerable capability had the advantage of not having a hospital to serve as a reservoir of infection. The operations were carried out in his own home in Danville Kentucky.

in the fluid may facilitate the diagnosis of tumors inflammatory conditions and intra peritoneal trauma.

PHYSIOLOGY

Relation to Extracellular Fluid Compartment. Since the peritoneum is a highly permeable membrane of considerable surface area,* peritoneal fluid is a physiologically active collection constituting part of the extracellular fluid. Deuterium oxide (heavy water) placed in the peritoneal cavity very rapidly becomes equilibrated with the plasma and interstitial fluid of the patient. The intraperitoneal route of fluid administration is used in experimental animals. Not only are water electrolytes and urea nitrogen rapidly transported across the peritoneal membrane but endogenous and exogenous toxic substances are freely absorbed. Rapid absorption of bacterial toxins is one of several reasons for the very high mortality in untreated peritonitis. In bowel obstruction with distention and impairment of circulation, transperitoneal absorption of bacterial toxins, even without peritonitis, is probable. Furthermore, antibiotics instilled into the peritoneal cavity are very rapidly absorbed and may exert toxic effects upon the whole organism.

In addition to the intimate relation of peritoneum to the circulating blood, there are communications between the peritoneal and pleural cavities which appear to be independent of the blood stream. In cases of *Meigs syndrome*, colloidal radioactive gold (Au^{198}) instilled into one serous sac rapidly appears in the other probably as a result of transdiaphragmatic lymphatic transport.

Sensory Innervation. The parietal peritoneum is well supplied with somatic afferent nerves and is sensitive to all forms of stimuli. This is particularly true of the anterior parietal peritoneum which, along with the rest of the anterior abdominal wall, is supplied by the lower six thoracic nerves. The parietal peritoneum posteriorly and in the pelvis is somewhat less sensitive. The ability of the parietal peritoneum to initiate the sensation of sharp pain in response to an inflammatory process in its vicinity is of the utmost importance in the diagnosis of an acute abdominal condition.

Moreover the nervous pathways from re-

ceptors in the parietal peritoneum, and particularly in that of the anterior abdominal wall permit fairly accurate localization of the source of the painful stimuli. When the parietal peritoneum is the source of a painful sensation, the area of pain indicated by the patient is of considerable diagnostic significance. The parietal peritoneum of the undersurface of the diaphragm is no exception to this rule and painful stimuli in the peripheral part are perceived in the vicinity of the adjacent body wall. Painful stimulation to the parietal peritoneum beneath the central diaphragm, although it is fairly accurately localized, is felt in the shoulder region as a result of *central misinterpretation*. The pain is sharp and the patient is able to indicate quite definitely where his pain is located in the neck or shoulder a situation quite different from the perception of dull and vaguely localized pain from visceral stimulation.

In addition to causing sharp and well-localized pain, stimulation of the parietal peritoneum may cause involuntary rigidity of the abdominal musculature, tenderness, referred tenderness and rebound tenderness, if the stimulus is sufficiently intense. These characteristics identify the parietal peritoneum as the source of the pain, identify the pain stimulus as being inflammatory (which it almost always is) and, further localize the site of the stimuli. Without the pain fibers of the parietal peritoneum, the clinical diagnosis of an "acute abdomen" would be impossible.

The visceral peritoneum is relatively insensitive, but it does register stimuli if they are sufficiently strong or prolonged, particularly in the presence of pre-existing inflammation. The root of the mesentery is quite sensitive to traction.

Most visceral afferent nerve fibers for pain run in the splanchnic nerves to the same six segments of the cord that receive somatic afferent fibers. The stimulus of visceral sensation is usually tension, whether the result of increased intraluminal pressure or increased tissue pressure from inflammation. Visceral pain is usually dull and vaguely localized to the central portion of the abdomen, although there are some exceptions to this rule notably visceral pain initiated by the biliary tree.

INFLAMMATION OF THE PERITONEUM

Peritonitis is an inflammation of the peritoneum. The process may be *acute* or *chronic*; it may be *septic* or *aseptic* and it

*The surface has been estimated to be 22,000 sq. cm. This should be compared with 15,000 sq. cm. as the approximate area of 2,000 glomeruli in two kidneys and with 24,000 sq. cm. as the dialyzing surface of a Skaggs-Leonards artificial kidney.

The most important of these is the *translocation of fluid electrolytes and protein into a "third space"* where they are for the time lost to the body economy. There are really three subdivisions of this "third space":

1. The *peritoneal cavity* where fluid is lost primarily by "weeping" of the peritoneum. Fluid lost by exudation has an electrolyte and nitrogen content similar to that of extracellular fluid. In addition, fluid may be lost into the peritoneal cavity by outpouring from the alimentary canal, the biliary tree, the pancreas or the urinary tract.
2. The *loose connective tissue* beneath the mesothelium of the bowel, mesenteries and plicae where protein rich fluid accumulates as edema of the peritoneum.
3. The *atonic, dilated gastrointestinal tract* where fluid of varying chemical composition is deposited.

Because of this loss of fluid into the third space, many surgeons have appropriately compared the physiologic derangement in peritonitis to that of an extensive burn. The comparison is valid in that there is temporary sequestration of a large amount of fluid having approximately the same composition as plasma. The analogy falls down somewhat in that in a burn there is a variable degree of hemolysis of red cells with early anemia and excess potassium in the extracellular compartment. The latter change is not seen in early stages of peritonitis unless there is associated renal failure.

In addition to loss of fluid into a third space, there are other physiologic derangements. Oral intake is not possible vomiting and, rarely diarrhea may occur. Renal function is the principal mechanism for the protection of the organism against dehydration and biochemical imbalance. The intravenous therapy which the patient receives aids renal function and contributes to fluid and electrolyte equilibrium. A "stress reaction" will alter renal function and the composition of body fluids and this reaction may be magnified by surgical measures employed in the treatment of the peritonitis.

The effect of peritonitis on the fluid and electrolyte balance of the body provided that it is not modified by therapy is the production of a metabolic, hypokalemic alkalosis with marked dehydration.

As in the case of a burn, fluid sequestered in the third space eventually is reabsorbed and becomes available to the economy with the production of diuresis if the patient has

been well maintained. In the burned patient diuresis can be expected to commence between forty-eight and seventy-two hours but no such simple rule can be given for the patient with peritonitis. Usually a longer period must elapse before the third space begins to release its fluid to the circulation.

It has been pointed out by Davis that the shock seen in peritonitis is due to several causes, including (1) reduction of the plasma volume, (2) myocardial damage due to bacterial toxins, (3) widespread vasodilatation due to bacteria or their toxins (4) adrenocortical insufficiency (5) fat embolism (6) hepatorenal insufficiency.

Clinical Manifestations. The clinical course of acute bacterial peritonitis secondary to contamination is highly variable. In an overwhelming peritonitis the patient may succumb within a few hours from toxemia and shock. More often the fatal case terminates after a difficult downhill course of three to seven days. As a result of improved therapy the natural history of the disease today is localization of the infection with gradual control of the process. Some cases of peritonitis masquerade as "prolonged postoperative ileus" and may be undiagnosed.

Symptoms and History. If the patient is seen early in the course of his disease, the symptoms may do no more than suggest a diagnosis, which must be confirmed by examination or in some cases a period of observation. Even if the diagnosis of peritonitis is obvious, a detailed history is of considerable importance in establishing the nature of the initial lesion, as management may be contingent upon this consideration.

Pain may vary in intensity but is the most important and constant symptom. Visceral pain, which may be either steady or crampy, is vague and poorly localized. Pain produced by irritation of the parietal peritoneum is usually sharp well localized, and virtually pathognomonic of an inflammatory process. The fact that it can be localized is of diagnostic importance. Changes in the location and intensity of pain originating in the parietal peritoneum may yield valuable information during a period of observation. Increasing pain and pain not relieved by drugs suggest spread of peritonitis. Pain is likely to be most intense over the spreading edge of the inflammatory process. Pain which subsides in intensity and decreases in area suggests a localizing process. The pain experienced by elderly and debilitated patients is minimal and not always in keeping

addition, the peritoneum exhibits certain peculiar weaknesses and special defensive powers.

The first weakness of the peritoneum is its proximity to the greater part of the gastrointestinal tract with its large bacterial population. Furthermore the whole of the peritoneal cavity is a continuous space through which a contaminant may be rapidly spread and freely disseminated, especially with the aid of respiratory, intestinal and abdominal movements. Dissemination may be aided by the outpouring of fluid into the cavity. Moreover mucus and partially digested intestinal contents from a bowel perforation promote multiplication and dissemination of bacteria and interfere with phagocytosis. Early localization by fibrinous adhesions may be overcome by an outpouring of intestinal contents or pus by the pressure of increasing inflammatory exudate and by gas from bacterial fermentation. The extensive absorptive surface of the peritoneum permits rapid absorption of the bacterial toxins.

The peritoneum like any other tissue, reacts to an irritant by inflammation and this is the basic defense mechanism. The essence of inflammation is phagocytic activity aided by humoral agents and the vascular response. The peritoneal membrane is exceptionally well adapted to produce an effective inflammatory reaction and to combat infection, provided the contamination is not massive or prolonged. A second and rather specific mechanism by which the peritoneum defends itself is by localization of an irritant. Localization is accomplished by bowel, mesentery and particularly omentum adhering to inflamed areas. At the site of inflammation these structures are arrested by the abundant, sticky fibrinous exudate which progressively increases the adherence of the localizing structures. Approximately thirty-six hours is required for the formation of reasonably secure fibrinous adhesions which will protect against diffuse peritonitis. Localization of infection is further aided by sympathetic inhibition of peristalsis to produce a "paralytic" ileus. It is true that prolonged ileus may kill the patient because of the effects of distention and inanition, but it provides temporary protection against continued spread of the peritonitis.

These factors cited above determine whether or not the patient will survive. If the organisms are virulent and contamination is sudden and massive, if the infection is rapidly disseminated over the whole of

the peritoneal cavity or if attempts at localization by the peritoneum are thwarted by injudicious surgery or by stimulation of the bowel, the infection may become overwhelming. If the organisms are less virulent, if there is no source of continuing contamination, if drainage is provided for those cases in which increasing pressure in a poorly walled-off area would otherwise seal the remainder of the peritoneum, if the efforts of the peritoneum to localize the infection are respected, if the patient is adequately supported by intravenous therapy and by gastrointestinal decompression and if the number of pathogens can be kept below the critical level by antibodies and by antibiotics then the cellular defenses of the peritoneum can be expected to overcome the infection.

Pathology. Acute bacterial peritonitis secondary to contamination may be either generalized or localized.

In *generalized peritonitis* the peritoneal cavity contains several hundred cubic centimeters of fluid, which may be thin, watery and odorless in the case of a hemolytic streptococcal infection or which may be purulent, contain flakes of fibrin and have a strong fecal odor in the case of coliform infection. The entire bowel is dilated, its serosa is congested, edematous and covered with a purulent or fibrinous exudate which may glue together the loops of small bowel. Between the loops of intestine may be loculated collections of pus. The omentum is usually attracted to the site of the infection, but it may occupy a normal position or be matted into a firm mass. The subperitoneal vessels are dilated and there is edema of the areolar tissue beneath the mesothelium. The process may go on to abscess formation, repair by fibrosis or complete resolution.

In *localized peritonitis* the changes will be similar but confined to one portion of the peritoneal cavity by adherence of mesentery, bowel and omentum to the source of contamination. After a week or ten days, when the adhesions have become fibrous, the walled-off area may gradually become converted into a shaggy walled abscess.

The microscopic findings include congestion, edema and leukocytic infiltration, with patchy areas of tissue necrosis.

Pathophysiology. An understanding of some of the clinical manifestations of peritonitis as well as a rational approach to therapy is contingent upon an appreciation of the physiologic aberrations which occur



Figure 24. Supine x ray of abdomen, generalized peritonitis (postoperative). Marked dilatation of large and small bowel is seen and there is thickening between adjacent intestinal loops.

spread (Fig. 24). Jejunum, ileum and colon contain air and can be identified by their typical patterns. The dilatation is usually less than in mechanical obstruction and the distribution of small bowel, while central, forms a "mosaic" pattern rather than horizontal loops with sharp hairpin turns as seen in mechanical obstruction. There is diffuse haziness, due to accumulation of exudate, with obliteration of the peritoneal fat line and psoas shadows. There is thickness of bowel wall between adjacent gas shadows due to edema of the bowel wall and the exudate. Free gas may be seen if peritonitis is due to perforation of bowel. Contrast studies are generally unnecessary and the administration of barium by mouth is contraindicated.

Periodic blood chemistry determinations may be of considerable value as guides to the supportive therapy of the patient.

The white blood count is almost always markedly elevated with a preponderance of polymorphonuclear granulocytes.

Diagnosis. There is no substitute for experience in developing diagnostic acumen in the management of patients with peritonitis. The salient clinical features, stated briefly are (1) abdominal pain, sharp steady and localized, (2) the signs of irritation of the parietal peritoneum, (3) a silent abdomen. As Maingot points out "It is

easier to diagnose the presence than the cause of peritonitis."

Differential Diagnosis. The differential diagnosis is that of an "acute abdomen."

Complications. Paralytic ileus, inanition, dehydration, electrolyte abnormalities, toxemia and shock are usually considered characteristic features of the disease, rather than complications. Septicemia and spread of infection by lymphatics to the retroperitoneum are less common and may be considered complications. The spread of localized peritonitis to become a diffuse infection is at the same time, a complication of one and the pathogenesis of the other.

When a patient lives beyond a seven- or eight-day period and the infection persists, this may lead to so-called *chronic septic peritonitis* in which there are numerous pockets of pus walled off by adhesions and coils of bowel. These patients frequently die of toxemia or paralytic ileus or from mechanical bowel obstruction.

A much more common outcome is a localized version of the same process, an intraperitoneal abscess. Most abscesses are confined to their site of origin (e.g., peri-appendiceal) or are pelvic. Therapy is frequently directed toward obtaining localization of the infection in the pelvis. Less common is the subphrenic abscess, difficult of diagnosis and exacting in management.

Another complication which can occur after peritonitis is mechanical small bowel obstruction due to fibrinous adhesions.

Prognosis. Thirty years ago one-third of patients with peritonitis could be expected to succumb. At the present time, with earlier and more accurate diagnosis, a better understanding of the deranged physiology, improved fluid and electrolyte therapy, the use of whole blood where indicated, sulfonamides and antibiotics, gastrointestinal decompression and safer anesthesia, the mortality is between 5 and 10 per cent.

Treatment. Prophylactic treatment. A number of special procedures such as pre-operative bowel preparation, use of drains when indicated and preservation of blood supply are particularly concerned with the prevention of postoperative peritonitis.

One question that repeatedly arises in connection with laparotomy for the purpose of preventing peritonitis is whether drainage should be carried out. An old axiom says "When in doubt, drain." W. J. Mayo turned this about to read "When in doubt, don't drain." The arguments against prophylactic drainage are (1) the drain tract

with the underlying pathologic process. It is well to bear in mind that the posterior and pelvic portions of the parietal peritoneum are not very sensitive and an inflamed organ in these locations may exhibit a more advanced pathologic state than one might suspect from the severity of the pain.

Vomiting is also a common symptom but its nature and mechanism vary with the stage of the disease. Early in peritonitis vomiting is reflex in origin and the vomitus consists of gastric contents. Later it is said to be toxic and the vomitus is brownish or bile stained. In the terminal stages of the disease vomiting accompanies paralytic ileus and the feculent bowel fluid wells up from the mouth without effort on the part of the patient.

Physical Findings. The diagnosis of peritonitis can usually be confirmed on the basis of the physical findings but often examination is not as revealing as interrogation in suggesting the precipitating lesion. When the physical findings are equivocal there is no substitute for repeated examination at frequent intervals. The abdomen should be thoroughly but gently re-examined.

In the early stages of peritonitis the vital signs (temperature pulse respiratory rate blood pressure) which are indices of the systemic effects of the disease may be normal or there may be only tachycardia and a low-grade fever.

The physical examination should be complete and not just confined to the abdomen. A red throat, a dilated fixed pupil, a few rales in a lung base icterus or a diminished femoral pulse may provide a clue to the cause of abdominal pain. Rectal examination is an important part of the physical examination of the patient with an acute abdomen. Inflammation in the pelvis may not cause significant abdominal findings but the exquisite tenderness elicited by rectal examination may be diagnostic.

The patient with peritonitis usually lies very still because he has found that all movements aggravate his pain. His thighs and knees may be flexed in order to relax his abdominal muscles. His breathing is shallow so as to avoid movements of the abdominal wall. After inspection is completed, the abdominal wall should be very gently palpated for minor degrees of involuntary guarding, all efforts being made to reassure the patient and eliminate voluntary contraction of abdominal muscles. Only after this maneuver is carried out should one complete the abdominal examination.

Guarding is of greater significance than tenderness, but once the patient has been hurt, it is difficult to detect minor degrees of this important physical finding. Point tenderness, referred tenderness and rebound tenderness on palpation along with involuntary guarding, point to irritation of the parietal peritoneum. Their presence is important and their location corresponds to that of the underlying inflammatory area.

Auscultation of the abdomen must always be part of the abdominal examination. The stethoscope is the key to distinguishing pain due to simple obstruction from that due to peritonitis. The silent abdomen indicates peritonitis.

Percussion has its place in the examination of the abdomen but does not often yield valuable information. Generalized hyperresonance due to the presence of gas-filled bowel may be elicited in ileus. Occasionally obliteration of the area of liver dullness will be found, suggesting free gas in the peritoneal cavity.

Examination for costovertebral angle and flank tenderness and the use of the psoas and internal obturator tests are also useful.

The Terminal Phase. In the end stages of the disease the previously high fever falls to normal or subnormal and the pulse becomes weak and very rapid, an ominous combination. The pain is severe and steady, the abdomen distended and silent and the vomiting is effortless. Hippocrates who must have seen it often, described the dusky pallor, anxious expression, sunken staring eyes, dry fissured tongue and wet skin that mark the end of the struggle as follows:

"If the eyelid be contracted, livid or pale or also the lip or nose, along with some of the other symptoms one may know for certain that death is close at hand. It is a mortal symptom, also when the lips are relaxed, pendent, cold, and blanched." Death is due to peripheral circulatory and renal failure.

Laboratory Aids. The diagnosis of peritonitis is made on clinical grounds and laboratory aids may confirm the diagnosis. The most important of these is a series of plain x-ray films of the abdomen. Plain films of the abdomen should be taken in three positions: anteroposterior supine, anteroposterior upright and left lateral decubitus. If the peritonitis is localized there may be a segmental ileus of some part of the bowel in only one region of the abdomen. With general peritonitis, the dilatation of the gastrointestinal tract within the abdomen is wide-

appendicitis provided that: (1) the patient is an adult, (2) he is first seen between two and five days after the onset, (3) he is very ill and (4) the infection seems to be localizing satisfactorily. This Ochsner-Sherren treatment must be carried out "at the door of the operating room," that is, with continuous observation and readiness to intervene surgically if the peritonitis begins to spread. This plan has no place in the treatment of children and is only occasionally employed for adults.

When surgical intervention is indicated, a few hours of supportive measures may be required to prepare the seriously ill patient for operation.

What is to be done at laparotomy? First, the source of contamination is removed or closed off (appendix or perforated ulcer, respectively) or in the case of a gallbladder with impending perforation, it may be simply drained. Secondly intraperitoneal fluid or exudate is aspirated to decrease the massiveness of the contamination. Thirdly if it appears advisable a drain may be put down to the source of contamination or to the site where fluid is likely to collect. Finally one may utilize the opportunity to guide a long tube, which is introduced through the usual nasal route, past the pylorus and into the small bowel. These maneuvers should be carried out as quickly as possible and without disseminating infection.

Assisting the peritoneum to localize and overcome infection. The great advances in modern drug therapy of infection should not be permitted to overshadow the importance of some basic and simple therapeutic measures. Rest for the patient and for his gastrointestinal tract is essential in order to permit localization of the peritonitis.

The site of localization is important, for should complete resolution of the infection fail and abscess formation result, the abscess will have to be located and drained. The pelvis is a very desirable location, the subphrenic region is a dangerous area for localization of an abscess. Coffey showed that when a patient lies flat in bed, the paracolic gutter is 1 inch lower than the pelvis and it is well known that the right paracolic gutter communicates with the subphrenic spaces on the right side (Fig. 22). To encourage drainage of fluid from the flank to the pelvis the head of the bed must be elevated 60 to 70 degrees or as

Fowler advocated, 8 to 14 inches higher than the foot of the bed. The patient's trunk, then, should be oblique with the patient either flat on a sloping bed or else in a semisitting position. The knees should not be bent and if the patient tends to slip down in bed it is good for his venous circulation if he must push his feet against a footboard from time to time to regain his position.

Application of heat to the abdomen in the form of hot packs or diathermy will aid bodily defenses and after a few days the use of warm rectal irrigations may promote localization in the pelvis. The basic defense against infection is still phagocytosis, aided by other natural defense mechanisms. Drugs such as sulfonamides and antibiotics must play a secondary role in that they only assist these natural processes. Pulaski has shown that any antibiotic which suppresses the total number of microorganisms to below a critical value of about 1×10^5 per ml. of exudate permits the body's defenses to cope with the remaining bacteria. Thus, it is not necessary to give a specific drug to eliminate completely each of the several bacterial species which may be present, the objective is only to lower the bacterial population below the "critical level."

At the present time it appears that the drugs of choice for the treatment of peritonitis from intestinal contamination are oxytetracycline, chlortetracycline, chloramphenicol, the combination of streptomycin and penicillin, and the sulfonamides. Penicillin is very effective against all streptococci, but it is inactivated by the penicillinase produced by various coliforms. This can be overcome by the use of a penicillin-streptomycin mixture, the only antibiotic combination which appears to have any advantage over the administration of a single drug.

The dose of sulfonamide or antibiotic administered should be large enough so that the partially resistant strains are not just replaced by totally resistant organisms. At the same time, it is well to remember that these drugs are toxic substances and that it is not unusual to encounter complications which necessitate reduction of dose or change of drug. If renal function is inadequate, the blood level of the antibiotics may parallel that of the nonprotein nitrogen. Vitamin K should be given by vein when broad spectrum antibiotics are given for more than a few days. The patient should

is a source of infection from the outside (2) it is impossible to drain the peritoneal cavity (3) a Penrose drain is ineffective after six or eight hours because it becomes sealed. All of these statements are true. It is possible and often desirable however to drain a localized portion of the peritoneal cavity. The last-named obstacle can be overcome by periodic loosening of the Penrose drain or by use of a sump (suction with air vent) drain. The decision as to whether to drain is often a matter of judgment and sometimes of personal preference. In general, prophylactic drainage is advised in cases of

1 A localized area of peritonitis around which secure adhesions have not yet formed

2 Surgery on the extrahepatic biliary tract

3 Injury to or surgery on the liver where there is a possibility of bile leakage

4 A gastrointestinal suture line which is insecure and not protected by other measures (e.g., duodenal stump)

5. Possible continued leakage of fluid (blood, urine, pancreatic juice) even though it is sterile at the time of surgery

6. The involvement of a retroperitoneal area (e.g., retrocecal retroperitoneal appendicitis with contamination)

A drain can be put down to the vicinity of the source of a fluid or contaminant or it can be placed to drain a low point to which fluid can be expected to gravitate (pelvis paracolic gutter Morrison's pouch) or both.

A second group of prophylactic measures, of no less interest to the surgeon, has to do with early diagnosis and treatment of lesions likely to culminate in peritonitis if not adequately treated.

Definitive treatment. The definitive treatment of bacterial peritonitis is based upon an understanding of the etiology and pathogenesis, the pathology and the physiologic derangements which have been discussed previously. Treatment can be resolved into five facets:

1 A decision must be made as to whether there is continuing contamination of the peritoneal cavity and, if there is, operative correction should be undertaken, provided other circumstances do not contraindicate surgical intervention.

2. All possible measures must be carried out to assist the peritoneum in localizing the infection, preferably in a location favorable for future drainage, and in overcoming that infection.

3 The gastrointestinal tract must be decompressed with a long tube, while the defensive nature of the paralytic ileus is respected.

4. Supportive treatment should be given to maintain the patient through his illness; this includes relief of pain, treatment of shock, maintenance of fluid and electrolyte balance and correction of hypoproteinemias.

5 Repeated clinical observations must be made for confirmation of diagnosis, to ascertain effectiveness of therapy and to note the progress of the disease.

The question of surgical intervention. The decision of operative interference depends upon the primary lesion more than on any other single factor but the time interval since the onset, the progress of the disease and the general condition of the patient will modify this decision. We have already pointed out that the peritoneum is well adapted to dealing with infection, but it will be overwhelmed by continuing contamination.

The influence of the primary lesion can be illustrated by some examples. If the peritonitis is due to acute diverticulitis with perforation, any infection will probably be well localized and the perforation will be small and readily sealed off by natural defenses. Operation in such a case if the diagnosis is certain, unless peritonitis is spreading, is useless and even harmful; treatment consists of intensive application of the "antiperitonitis regimen" (points 2 to 5 above) without surgical intervention. In the case of a perforated peptic ulcer one usually operates in the first twenty-four hours, but not later. During the first twenty-four hours there is continuing contamination and a progressively poorer prognosis after that the perforation has usually sealed off. In any case the patient is moribund from his generalized peritonitis, so that operation carries a higher risk than do active conservative measures alone. In acute appendicitis with generalized peritonitis continuing contamination can probably be expected for several days and the appendix should be removed, especially in children. If the patient's condition is such that he will tolerate surgery. The mortality rate in this situation, generalized peritonitis from acute appendicitis, is about twice as high (13 per cent) with conservative treatment as it is with surgical removal of the appendix and intensive supportive measures.

Conservative measures are justified in the treatment of localized peritonitis from acute

case are as much the responsibility of the physician as are the initial diagnosis and original plan of management.

LESS COMMON TYPES OF PERITONITIS

Primary ("Idiopathic") Peritonitis. Primary peritonitis is an infection which is not secondary to an operation or a wound, to an inflammatory perforative or other disease process in or near the peritoneal cavity or to known hematogenous spread of an infection elsewhere. Obviously the origin of the disease is obscure and the term "idiopathic" seems appropriate.

Primary peritonitis was fairly common prior to the advent of sulfonamides and antibiotics but it is rare today. The majority (75 per cent) of cases occur in girls, usually between the ages of two and ten, but it has been described at all ages.

About two-thirds of the cases of primary peritonitis are due to hemolytic streptococci and about one-third are due to pneumococci. Only a single organism is involved in any case; hence it differs from the "polymicrobial" character of most cases of secondary peritonitis. Occasionally primary peritonitis is said to be due to coliforms but such a diagnosis must always be suspect. About half of the pneumococcal cases occur in children with nephrosis.

One concept regarding the pathogenesis of primary peritonitis is that the organisms gain entry through the vagina and fallopian tubes. This is supported by the observation that the disease is one of the underprivileged classes and of unhygienic conditions. The alkaline pH of the vagina in children may account for the age incidence mentioned. Another suggestion has been that most of the infections are really hematogenous and the proponents of this theory point to a high incidence of an associated focus of infection. Neither view completely explains the sex distribution.

Streptococcal infections cause a fulminating infection with a watery odorless exudate and the pathologic features of streptococcal peritonitis. Pneumococcal infections are less fulminating and cause a characteristic gelatinous, sticky pale green, odorless exudate with abundant fibrin formation.

The child is most often (75 per cent) under five years of age. A pre-existing upper respiratory infection, otitis media or pneumonia may mask for a while the onset of the peritonitis. The main clinical features are rapid onset of diffuse abdominal pain, fever, often chills, restlessness, ir-

ritability, vomiting, diarrhea in half the cases, sometimes hematemesis or melena and manifestations of extreme toxicity.

The child usually has a temperature of 104° F., looks very ill and may be markedly dehydrated with dry mucous membranes and sunken eyes. The abdomen may be protuberant and diffusely tender. There may be abundant fluid in the abdomen and sometimes shifting dullness can be demonstrated. The consistency of the abdominal wall varies from doughy in infants to boardlike in older children. Peristaltic activity is increased at first and is then followed shortly by paralytic ileus. In pneumococcal cases there may be cyanosis and herpetic lesions on the lips.

The white blood count usually ranges from 20,000 to 50,000 with polymorphonuclear neutrophils predominating. In cases superimposed on nephrosis there is a heavy albuminuria. Blood culture is usually positive for streptococci or pneumococci.

Pneumonia should be ruled out. This diagnosis may be difficult in cases where there is a paucity of physical and x-ray findings in the chest. A rapid respiratory rate and lack of abdominal tenderness will aid in the differentiation. The two conditions may co-exist.

A more difficult problem is the differentiation between primary peritonitis and secondary peritonitis, usually from a perforated appendix. The rapidity of onset and stormy course, particularly if accompanied by a chill, the finding of generalized rather than right-sided tenderness, the degree of fever and leukocytosis during the entire illness, the finding of a primary focus and a history of nephrosis or a finding of heavy albuminuria may favor a diagnosis of primary peritonitis.

The physician rarely accepts a diagnosis of primary peritonitis while acute appendicitis, which is far more common, remains a possibility. In moribund children and in instances where the diagnosis of primary peritonitis seems almost certain because of known nephrosis or a known coexisting focus of infection and septicemia, the matter may be settled by peritoneal aspiration under local anesthesia. A smear of the exudate showing pure streptococci or pneumococci will allow one to proceed conservatively with assurance.

In the majority of cases the diagnosis can be made at exploration. A right flank incision can be very limited if one suspects a primary

be watched for any evidence of agranulocytosis, diarrhea or mycotic overgrowth in the mouth, pharynx and lungs.

The drugs should be given intravenously or intramuscularly in peritonitis. Rarely can the oral route be used. The question of intraperitoneal administration has not been settled as yet, this route appears to have advantages but is not without danger. Deaths from respiratory arrest have occurred as a result of intraperitoneal administration of neomycin. Chlorotetracycline, tetracycline and streptomycin have been shown to be toxic when given intraperitoneally to experimental animals.

Gastrointestinal decompression. The gastrointestinal tract must be decompressed to prevent the secondary effects of distention upon the circulation in the bowel wall and also upon the patient's respiratory and circulatory systems. This procedure helps one to estimate the amount of fluid lost into the bowel from the body's economy. Gastric suction is effective to some degree but the use of a long intestinal tube is preferable. High concentrations of oxygen by nasal catheter may lessen distention. A rectal tube and application of heat to the abdomen are of value after bowel function begins to return. The use of Prostigmine, Pitressin and all such drugs to stimulate the bowel is definitely contraindicated.

Supportive therapy. The fluid and electrolyte requirements of the patient with peritonitis must be supplied by the intravenous route. The plan of treatment depends on calculation of the "static deficit," the "dynamic losses," and the "base line requirements," as outlined by Randall. Estimation of the first of these and to some extent the second, is nothing more than an "educated guess" and must be revised by noting the patient's response to therapy. It is helpful to realize that the fluid lost into the third space amounts to several liters and that its composition resembles that of plasma. The use of an indwelling catheter to make certain that a urine flow of 25 to 50 cc. per hour is being maintained, along with other clinical assessment of the state of hydration, will aid in determining the quantity of fluid to be administered.

One can judge the mineral requirements of the patient from the considerations just mentioned and serum electrolyte determinations. Some of the common pitfalls of fluid and electrolyte therapy must be kept in mind in the difficult task of caring for these patients.

1. A low serum sodium level of 125 to 130 mEq per liter is to be expected for forty-eight to seventy-two hours after severe stress.

2. Serum potassium is a poor index of the intracellular level. Potassium should be withheld for forty-eight hours after surgery or in the presence of renal insufficiency or elevated nonprotein nitrogen. Otherwise it should be given at the rate of about 80 mEq per day in order to avoid intracellular depletion and prolonged ileus.

3. Overhydration is a danger in the elderly and after resorption from the third space begins.

Depleted serum proteins must also be replaced by the administration of whole blood, serum albumin or plasma stored at room temperature. Protein hydrolysates can be used to reduce the degree of the negative nitrogen balance, provided that one can simultaneously supply enough calories in the form of glucose to meet the demands of the elevated metabolic rate. For the patient with prolonged ileus, intravenous fat preparations may be used to combat inanition.

As a rule, the use of cortisone and hydrocortisone in the presence of an infection, and particularly if it is due to an ulcerative lesion of the gastrointestinal tract, is contraindicated. Their use is occasionally justified, however, in patients who show signs of adrenocortical insufficiency i.e., persistent hypotension and vasoconstriction after adequate blood replacement and a high circulating eosinophil count. This state may result from previous adrenal disease, adrenal atrophy due to previous cortisone administration, or from adrenal exhaustion due to the peritonitis.

Relief of pain is part of supportive therapy. Morphine or meperidine (Demerol) should be administered often enough and in adequate dosage to guarantee the patient pain-free periods when he can obtain necessary rest.

Continued observation. Finally the patient with peritonitis must be frequently reassessed. The diagnosis of the causative lesion, if it has not been confirmed by exploration is not always clear cut. Response of the patient to the therapy administered should be evaluated several times daily during the acute phase of the illness. Awareness of the complications of the disease and their treatment is essential. Repeated examination of the abdomen for evident localization or spread of the peritonitis, careful observation of the

case are as much the responsibility of the physician as are the initial diagnosis and original plan of management

LESS COMMON TYPES OF PERITONITIS

Primary ("Idiopathic") Peritonitis. Primary peritonitis is an infection which is not secondary to an operation or a wound to an inflammatory perforative or other disease process in or near the peritoneal cavity or to known hematogenous spread of an infection elsewhere. Obviously the origin of the disease is obscure and the term "idiopathic" seems appropriate.

Primary peritonitis was fairly common prior to the advent of sulfonamides and antibiotics, but it is rare today. The majority (75 per cent) of cases occur in girls usually between the ages of two and ten, but it has been described at all ages.

About two-thirds of the cases of primary peritonitis are due to hemolytic streptococci and about one-third are due to pneumococci. Only a single organism is involved in any case; hence it differs from the polymicrobial character of most cases of secondary peritonitis. Occasionally primary peritonitis is said to be due to coliforms but such a diagnosis must always be suspect. About half of the pneumococcal cases occur in children with nephrosis.

One concept regarding the pathogenesis of primary peritonitis is that the organisms gain entry through the vagina and fallopian tubes. This is supported by the observation that the disease is one of the underprivileged classes and of unhygienic conditions. The alkaline pH of the vagina in children may account for the age incidence mentioned. Another suggestion has been that most of the infections are really hematogenous and the proponents of this theory point to a high incidence of an associated focus of infection. Neither view completely explains the sex distribution.

Streptococcal infections cause a fulminating infection with a watery odorless exudate and the pathologic features of streptococcal peritonitis. Pneumococcal infections are less fulminating and cause a characteristic gelatinous, sticky pale green, odorless exudate with abundant fibrin formation.

The child is most often (75 per cent) under five years of age. A pre-existing upper respiratory infection, otitis media or pneumonia may mask for a while the onset of the peritonitis. The main clinical features are rapid onset of diffuse abdominal pain, high fever, often chills, restlessness, ir-

ritability, vomiting, diarrhea in half the cases, sometimes hematemesis or melena, and manifestations of extreme toxicity.

The child usually has a temperature of 104 F, looks very ill and may be markedly dehydrated with dry mucous membranes and sunken eyes. The abdomen may be protuberant and diffusely tender. There may be abundant fluid in the abdomen and sometimes shifting dullness can be demonstrated. The consistency of the abdominal wall varies from doughy in infants to boardlike in older children. Peristaltic activity is increased at first and is then followed shortly by paralytic ileus. In pneumococcal cases there may be cyanosis and herpetic lesions on the lips.

The white blood count usually ranges from 20,000 to 50,000 with polymorphonuclear neutrophils predominating. In cases superimposed on nephrosis there is a heavy albuminuria. Blood culture is usually positive for streptococci or pneumococci.

Pneumonia should be ruled out. This diagnosis may be difficult in cases where there is a paucity of physical and x-ray findings in the chest. A rapid respiratory rate and lack of abdominal tenderness will aid in the differentiation. The two conditions may co-exist.

A more difficult problem is the differentiation between primary peritonitis and secondary peritonitis, usually from a perforated appendix. The rapidity of onset and stormy course particularly if accompanied by a chill, the finding of generalized rather than right-sided tenderness, the degree of fever and leukocytosis during the entire illness, the finding of a primary focus and a history of nephrosis or a finding of heavy albuminuria may favor a diagnosis of primary peritonitis.

The physician rarely accepts a diagnosis of primary peritonitis while acute appendicitis, which is far more common, remains a possibility. In moribund children and in instances where the diagnosis of primary peritonitis seems almost certain because of known nephrosis or a known coexisting focus of infection and septicemia, the matter may be settled by peritoneal aspiration under local anesthesia. A smear of the exudate showing pure streptococci or pure pneumococci will allow one to proceed with conservative therapy with assurance.

In the majority of cases the diagnosis will be made at exploration. A right lower quadrant incision can be very limited in extent if one suspects a primary peritonitis. Gross

recommends the following procedure (1) obtain some exudate for a smear to prove the diagnosis—this is done by an assistant immediately not postoperatively—and culture (2) confirm that the appendix is not the source of the infection, (3) put a small drain down into the right iliac fossa, (4) do not remove the appendix if it is normal. Postoperatively, an antiperitonitis regimen should be followed. Sulfonamides and penicillin are the specific drugs of choice.

Bacterial Peritonitis Due to Hematogenous Spread. This entity is blood borne from a known infection elsewhere in the body so that it is really a secondary peritonitis. It differs from contamination peritonitis in that it does not arise from a lesion in the vicinity of the peritoneum.

The source of the infection may or may not be apparent. Many surgeons believe that the majority of cases of so-called primary peritonitis actually arise from a hidden focus, are really hematogenous and so belong in this group. The fact that the majority of the organisms are streptococci and pneumococci favors this point of view.

The clinical features are identical to those which have been described for primary peritonitis.

This disease, like primary peritonitis, should be treated conservatively when the diagnosis can be made.

Tuberculous Peritonitis. Tuberculous peritonitis was at one time fairly common, but fortunately it is now rarely seen. It is almost always associated with tuberculosis elsewhere.

Tuberculous peritonitis is somewhat more common in females. Both children and adults may be affected, the usual age range being between ten and forty years.

The infecting organism is *Mycobacterium tuberculosis* of either the human or the bovine type. The primary lesion is most often tuberculous ulceration of the bowel, but infection may also arise from tuberculous mesenteric lymph nodes, tuberculosis of the fallopian tubes, lymphatic spread from the lung or pleura or hematogenous spread from distant sites.

Basically there are two types of tuberculous peritonitis—the "moist" variety and the "dry" form. Tubercles are found in both varieties, but they may not be immediately apparent on gross examination because of fibrinous or purulent exudate caseation or fibrous adhesions. In the dry form there is usually a dense inflammatory exudate and numerous adhesions matting the coils of

bowel together. Fecal fistula formation may occur in either type particularly in the dry form, and in children the fistula may communicate with the umbilicus. In the moist form the patient is likely to be a child and the ascites may be massive. The fluid is usually thin and lemon yellow and it may coagulate spontaneously. Its specific gravity is over 1.018 and its protein content over 4 per cent, thus distinguishing the fluid from a transudate. The encysted variety is a combination of both of the above forms, with a localized area of fluid which may be mistaken for some other cystic lesion.

The condition is of fairly acute onset in two-thirds of the cases. The moist form is likely to run a more insidious course than the dry. There are systemic manifestations, consisting of wasting, anorexia, and fever accompanied by abdominal pain, vomiting, diarrhea and enlargement of the abdomen. In the moist form the abdomen may become very distended and tense with dilatation of the veins of the abdominal wall and, if the tunica vaginalis is patent, a simulated hydrocele. Redness of the umbilicus may precede formation of a fecal fistula.

The diagnosis is usually not difficult, as there is evidence of tuberculous infection elsewhere. In the ascitic variety peritoneal fluid may be aspirated for culture and guinea pig inoculation. The differential diagnosis includes pyogenic peritonitis, carcinomatosis, other forms of ascites, abdominal tumor and celiac disease.

The chief complications are cachexia and intestinal obstruction. In the preantibiotic era the mortality was 40 per cent, death often being due to toxemia, tuberculosis elsewhere or rarely to amyloidosis.

The treatment consists chiefly of general supportive measures for tuberculosis, which should be carried out in a sanatorium. The use of the antituberculous drugs—streptomycin, para aminosalicylic acid and isonicotinic acid hydrazide—have improved the prognosis markedly. In the ascitic variety laparotomy and evacuation of the fluid seem to have a beneficial effect. In the dry form, operation should be avoided, except when made mandatory by intestinal obstruction. When laparotomy has been carried out in the presence of tuberculous peritonitis, drainage and exteriorization of bowel should be avoided.

Aseptic Peritonitis. Aseptic peritonitis is important for two reasons. A variety of sterile materials (e.g., foreign bodies, bile, blood, meconium) which may gain entrance

to the peritoneal cavity are irritants which set up aseptic inflammatory reactions giving rise to sequelae and clinical symptoms. Secondly the chemical peritonitis produced by any of these substances may be followed by a superimposed bacterial peritonitis.

Foreign bodies may reach the peritoneal cavity from operative procedures, e.g., sponges, suture material, instruments. They may result from penetrating wounds, missiles and bits of clothing. The gastrointestinal and genitourinary tracts may be the source of fish bones, wood splinters, needles, pins and glass. Such foreign bodies usually cause one of three types of reaction: (1) bacterial inflammation in which the foreign body is incidental, (2) aseptic inflammation, which usually results in a localized sterile abscess as the peritoneum walls off the foreign body; (3) a similar process with formation of a sinus or fistula. The relative amounts of exudation and fibrosis vary greatly with the nature of the foreign body.

The clinical manifestations of a sterile intraperitoneal foreign body are highly variable. A surgical sponge may give rise to an abscess with a mass, fever, chills, toxic manifestations and legal implications. This type of foreign body must be removed. In intraperitoneal suture, if sterile, may be simply walled off by fibrous tissue and give rise to few if any manifestations in the emotionally stable patient. The surgeon must guard against being persuaded by the patient to undertake a search for this type of foreign body on the basis of vague subjective complaints.

Bile may ooze through the wall of a gall bladder which is distended but intact. More commonly bile reaches the peritoneal cavity as a result of biliary tract rupture or as a sequel to operations on the biliary system. Sterile bile is a mild irritant in its own right, but should it become secondarily infected, it may give rise to a virulent type of peritonitis. To prevent this unfortunate sequel, the adequate routine drainage of Morrison's hepatorenal space is recommended after biliary surgery or liver trauma.

Blood is only a mild irritant and it is slowly absorbed. However blood in the peritoneal cavity may serve as a nidus for bacterial infection. For this reason it should always be evacuated at the end of an operation. An oozing surface capable of producing hematoma, should be drained.

A most interesting entity is meconium or fetal peritonitis which was first described by Sir James Young Simpson in 1835. The

disease results from perforation of the bowel and inflammation of the peritoneal membrane due to sterile meconium. The perforation of the intestine may occur any time from the third fetal month to the neonatal period. It may result from meconium ileus, congenital diverticula, atresia, stenosis, congenital bands, hernia, volvulus or intussusception. If the opening is still present at birth or if perforation occurs after birth, secondary bacterial infection is a rule and the outlook in such cases is very poor.

Gastric juice is very irritating and the chemical peritonitis which is set up by a perforated ulcer is followed, after several hours, by bacterial infection. Sterile urine from traumatic intraperitoneal rupture of the bladder is also an irritating fluid and this chemical insult is frequently followed by secondary infection. These lesions must be diagnosed early and treated by appropriate operation.

Intraperitoneal pancreatic juice usually becomes infected and in hemorrhagic pancreatitis superadded bacterial infection should always be anticipated. Intensive antibiotic therapy deals adequately with this infection in most instances, but occasionally an abscess or collection will have to be drained ten to fourteen days after the onset.

GRANULOMATOUS LESIONS OF THE PERITONEUM

Separation of foreign body granulomas of the peritoneum from aseptic peritonitis due to foreign bodies is somewhat arbitrary. The basic pathologic process is the same—the response of the peritoneum to a noninfected irritant. The varying degrees of exudation and fibrosis distinguish the two reactions.

Some of the substances which are more commonly reported as causes of granulomatous reaction by the peritoneal membrane are talc, starch, lycopodium spores, mineral oil and silica. These etiologic agents are often introduced into the peritoneal cavity by the surgeon.

In the past, granulomatous lesions due to talc were often seen, because this substance was widely used as a glove powder. Talc is composed mainly (53 per cent) of hydrated magnesium silicate and incites a very intense fibroblastic reaction which may give rise to dense adhesions within a few weeks, or the process may take years. In an attempt to overcome this problem, powdered starch was introduced as a glove powder by Lee and Leliman. The starch is made from either corn or rice and should be relatively in

recommends the following procedure (1) obtain some exudate for a smear to prove the diagnosis—this is done by an assistant immediately not postoperatively—and culture; (2) confirm that the appendix is not the source of the infection (3) put a small drain down into the right iliac fossa, (4) do not remove the appendix if it is normal. Postoperatively an antiperitonitis regimen should be followed. Sulfonamides and penicillin are the specific drugs of choice.

Bacterial Peritonitis Due to Hematogenous Spread. This entity is blood borne from a known infection elsewhere in the body so that it is really a secondary peritonitis. It differs from contamination peritonitis in that it does not arise from a lesion in the vicinity of the peritoneum.

The source of the infection may or may not be apparent. Many surgeons believe that the majority of cases of so-called primary peritonitis actually arise from a hidden focus, are really hematogenous and so belong in this group. The fact that the majority of the organisms are streptococci and pneumococci favors this point of view.

The clinical features are identical to those which have been described for primary peritonitis.

This disease, like primary peritonitis, should be treated conservatively when the diagnosis can be made.

Tuberculous Peritonitis. Tuberculous peritonitis was at one time fairly common but fortunately it is now rarely seen. It is almost always associated with tuberculosis elsewhere.

Tuberculous peritonitis is somewhat more common in females. Both children and adults may be affected, the usual age range being between ten and forty years.

The infecting organism is *Mycobacterium tuberculosis* of either the human or the bovine type. The primary lesion is most often tuberculous ulceration of the bowel, but infection may also arise from tuberculous mesenteric lymph nodes, tuberculous of the fallopian tubes, lymphatic spread from the lung or pleura or hematogenous spread from distant sites.

Basically there are two types of tuberculous peritonitis—the "moist" variety and the "dry" form. Tubercles are found in both varieties, but they may not be immediately apparent on gross examination because of fibrinous or purulent exudate, caseation or fibrous adhesions. In the dry form there is usually a dense inflammatory exudate and numerous adhesions matting the coils of

bowel together. Fecal fistula formation may occur in either type, particularly in the dry form and in children the fistula may communicate with the umbilicus. In the moist form the patient is likely to be a child and the ascites may be massive. The fluid is usually thin and lemon yellow and it may coagulate spontaneously. Its specific gravity is over 1.018 and its protein content over 4 per cent, thus distinguishing the fluid from a transudate. The encysted variety is a combination of both of the above forms, with a localized area of fluid which may be mistaken for some other cystic lesion.

The condition is of fairly acute onset in two-thirds of the cases. The moist form is likely to run a more insidious course than the dry. There are systemic manifestations, consisting of wasting, anorexia, and fever accompanied by abdominal pain, vomiting, diarrhea and enlargement of the abdomen. In the moist form the abdomen may become very distended and tense with dilatation of the veins of the abdominal wall and, if the tunica vaginalis is patent, a simulated hydrocele. Redness of the umbilicus may precede formation of a fecal fistula.

The diagnosis is usually not difficult, as there is evidence of tuberculous infection elsewhere. In the ascitic variety peritoneal fluid may be aspirated for culture and guinea pig inoculation. The differential diagnosis includes pyogenic peritonitis, carcinomatosis, other forms of ascites, abdominal tumor and celiac disease.

The chief complications are cachexia and intestinal obstruction. In the preantibiotic era the mortality was 40 per cent, death often being due to toxemia, tuberculous elsewhere or rarely to amyloidosis.

The treatment consists chiefly of general supportive measures for tuberculosis, which should be carried out in a sanatorium. The use of the antituberculous drugs—streptomycin, para-aminosalicylic acid and isonicotinic acid hydrazide—have improved the prognosis markedly. In the ascitic variety laparotomy and evacuation of the fluid seem to have a beneficial effect. In the dry form, operation should be avoided, except when made mandatory by intestinal obstruction. When laparotomy has been carried out in the presence of tuberculous peritonitis, drainage and exteriorization of bowel should be avoided.

Aseptic Peritonitis. Aseptic peritonitis is important for two reasons. A variety of sterile materials (e.g. foreign bodies, bile, blood, meconium) which may gain entrance

smooth postoperative course with nothing unusual except for a low grade fever and slight increase in pulse rate about the time that discharge from hospital is being considered. Later the temperature spikes to 101° or 103° F and chills are not unusual. There may be associated anorexia, nausea and vomiting. Constipation and some abdominal distention may result from a localized area of paralytic ileus. In long standing cases there may be marked weight loss.

The patient with an abscess usually looks ill. He may be dehydrated and either flushed from fever or pale from associated anemia. Usually a mass is palpated in the right iliac fossa on abdominal examination and there is associated tenderness and guarding. The mass may be palpable by rectal examination and may be fluctuant. When an abscess is present leukocytosis is usually marked with a predominance of polymorphonuclear neutrophils.

Periappendiceal abscess may give rise to other complications, including generalized peritonitis, spontaneous drainage into the bowel, fecal fistula, pyelophlebitis, paralytic ileus, mechanical bowel obstruction, septicemia and death.

Many cases of periappendiceal abscess resolve with conservative treatment alone. In such cases the patient probably has only an indurated mass with a low grade fever and minimal symptomatology. A patient with a high spiking fever will certainly require surgical intervention, as well as supportive measures, the latter consisting of antibiotics, bed rest, heat to the abdomen, relief of pain, intravenous fluids, electrolytes, glucose and sometimes blood as well as intubation. The very ill patient with a hectic fever requires adequate drainage of the abscess as soon as dehydration and ketosis are corrected.

The operation for periappendiceal abscess has three objectives: (1) to drain the abscess, (2) to avoid contamination of the general peritoneal cavity (3) to remove the appendix. The first two are essential the third is not. Although the majority of appendices in such circumstances can be removed, it is better to leave the appendix than to disseminate infection. An extraperitoneal approach should be used, a small incision being made either over the most prominent part of the mass or else lateral to the mass. If it becomes apparent that the uninvolved peritoneal cavity will be traversed, the wound should be closed and a more suitable site for incision chosen. Needle

aspiration is a blind procedure and is neither safe nor adequate. The pus should be completely evacuated and a Penrose drain placed in the depth of the cavity.

Pelvic Abscess. A pelvic abscess may result from disease which is primary in that location or by gravitation of infected exudate from peritonitis elsewhere into the pelvis. The latter development is encouraged by positioning of the patient because if abscess formation cannot be avoided, the pelvis is a relatively favorable location. A pelvic abscess is preferable to a subphrenic abscess because (1) it does not interfere with respiratory function (2) it is relatively easy to diagnose (3) it is easy to drain or if drainage is spontaneous it is usually into the large bowel.

The symptomatology of a pelvic abscess resembles that of a periappendiceal abscess in almost every respect. One significant difference, however is that pelvic abscess is often characterized by diarrhea. Pain tenderness and guarding are usually less marked and, of course the mass is not felt as well abdominally as it is by rectal or bimanual examination.

Supportive therapy of a pelvic abscess is similar to that for a periappendiceal abscess except that if the mass is not fluctuant, it may be encouraged to point toward the rectum by means of warm enemas. The daily rectal examinations which are carried out to evaluate the abscess, may be enough to encourage "spontaneous" rupture into the rectum.

If "spontaneous" rupture does not occur severe symptoms and finding of fluctuation may serve as criteria for operative intervention. The abscess may be opened through the posterior fornix of the vagina if the abscess lies in the cul-de-sac in the female or through the anterior wall of the rectum. These approaches should be used rather cautiously because a loop of adherent bowel may be mistaken for loculated pus. If the rectal route is chosen, dilatation of the anal sphincter will promote continuous drainage. If the abscess is not pointing toward rectum or vagina, and particularly if the mass can be felt suprapublically it is better to open it by an abdominal incision and by an extraperitoneal route.

Subphrenic Abscess. A subphrenic abscess is an abscess located in the space between the diaphragm and the transverse colon. These abscesses are not common, compared to other intraperitoneal abscesses. They are important because they are difficult to diag-

nocuous because it is gradually absorbed by the peritoneum. Unfortunately it has been found that starch may also give rise to a foreign-body reaction although less frequently and to a lesser degree than talc.

It appears that soluble starch is the glove powder of choice at the present time. Gloves should be washed or wiped off before hands are put into the peritoneal cavity and care should be taken to avoid spillage of glove powder should a glove be torn during an operation.

The gross appearance of these granulomatous lesions ranges from a studding of the serous membranes with nodules resembling tubercles or larger masses, to the formation of dense fibrous adhesions.

Microscopically the reaction is seen to be the formation of a chronic granuloma. There may be monocytes epithelioid cells, multi nucleated giant cells, lymphocytes plasma cells, fibroblasts and sometimes areas of necrosis. Particles of the particular foreign material responsible can often be identified in the monocytes or giant cells by its characteristic appearance. Starch granules can be differentiated from talc by their blue staining with Gram's iodine and by formation of "Maltese crosses" when viewed with polarized light.

INTRAPERITONEAL ABSCESES

An intraperitoneal abscess may be regarded as a complication or sequel of either localized or generalized peritonitis. If the peritoneum succeeds in completely walling off the infectious process but is not able to overcome the infection, an abscess develops. The situation may be looked upon as a stalemate between host and pathogen. However the patient may be very ill from the infection and the presence of the abscess constitutes a constant threat to the life of the individual.

An intraperitoneal abscess may arise in one of two ways. First, it may result from a slowly advancing inflammatory process within the abdomen, in which case the abscess will develop in the immediate vicinity of the diseased organ, because the defenses of the peritoneum have had sufficient time to localize the infection. An appendiceal abscess is the most common example of this variety but much the same process may occur from an acutely inflamed gallbladder with perforation, from acute diverticulitis, from hemorrhagic pancreatitis from acute salpingitis and numerous other conditions which give rise, as a rule to localized rather

than generalized peritonitis. In the second group there is usually sudden massive contamination of the entire peritoneal cavity as in a perforated peptic ulcer or a penetrating wound with injury to bowel in which, if the patient survives, the infection becomes secondarily localized. Such localization generally occurs in dependent parts of the abdominal cavity to which the infected exudate tends to gravitate. These abscesses usually occur in the pelvis and this is a favorable location, or in the subphrenic region, which is a very undesirable site.

Periappendiceal Abscess. Most periappendiceal abscesses lie in the right iliac fossa, but, largely because of the variable position of the appendix, the abscess may be pelvic, may lie in the paracolic gutter may be in front of or behind the distal ileum or may be retroperitoneal in position. The postappendectomy abscess may also involve the operative wound.

An abscess need not arise from perforation of an organ. Infection may pass through the inflamed but intact wall as is usually the case with peridiverticular abscess. In the case of appendicitis, however perforation usually has occurred by the time an abscess is diagnosed. As a result of inflammation and exudation of fibrin, omentum, mesentery and bowel become adherent about the appendix and localize the infection. There is no sharp demarcation between localized peritonitis and abscess formation—the exudate becomes progressively more purulent and the fibrinous adhesions become organized and within five to ten days an abscess has developed.

The pus of a periappendiceal abscess is never sterile. The organisms are those found in peritonitis, with *Escherichia coli* streptococci, and staphylococci predominating. Mixed infections are not uncommon.

The symptoms of a periappendiceal abscess immediately follow and cannot be sharply distinguished from those of the preceding appendicitis and localized peritonitis. When a suppurative or gangrenous appendix has been removed, perhaps with some contamination at the time of surgery there may be an interval of several days before the clinical features of an abscess are noted. If the infection has been suppressed by antibiotics the abscess may not be apparent for several weeks or even several months.

The patient with a periappendiceal abscess usually presents with spiking fever abdominal pain and malaise. Following appendectomy there may be an apparently

cesses in the lesser sac are comparatively rare and are sealed off by adhesions about the foramen of Winslow.

Incidence Subphrenic abscess is relatively uncommon. It is more often found in males than females (3:1) correlating with the higher incidence of primary etiologic lesions in males. In the first quarter of the present century the disease was more common in the second through the fourth decades, whereas it is now more common in the fifth and sixth decades of life. This may reflect earlier and better treatment of acute appendicitis and other abdominal emergencies and an increasing number of elective operations on the gastrointestinal tract of older patients.

Etiology Subphrenic abscess is usually a sequel to abdominal surgery and to suppurative and perforative intra-abdominal lesions. At one time surgery was not a common cause, but elective operations now precede almost half the cases. Over half of the remainder follow appendicitis, perforated peptic ulcer and suppurative disease of the biliary tract. Occasionally it follows pancreatitis. Only 10 per cent of cases arise outside of the abdomen.

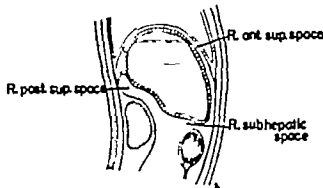
The infection either originates in the subphrenic region or it begins elsewhere in the abdomen and reaches the subphrenic region by spread of contamination over the peritoneal surface. Subphrenic abscess occasionally arises by hematogenous or lymphatic dissemination, by rupture of a liver abscess or by spread from a retroperitoneal phlegmon.

The organisms are the pyogenic bacteria which cause peritonitis—chiefly coliforms, streptococci and staphylococci. Tubercle bacilli are rarely the etiologic agent today. Occasionally in the southern United States subphrenic abscess is due to *Entamoeba histolytica*.

Pathology Subphrenic contamination is often overcome by the peritoneum with or without the aid of antibiotics. It has been estimated that only 30 per cent of subphrenic infections go on to suppuration.

Subphrenic abscess is found on the right side far more often (75 per cent) than on the left. Gas is present in 25 per cent of subphrenic abscesses. It results from the pneumoperitoneum following perforation of bowel, as a sequel to surgery or from gas-forming organisms.

Most complications of subphrenic abscess are thoracic and the most common is pleural effusion. Other complications include pneu-



Figures 27 and 28. Diagrammatic representations of parasagittal sections, showing the subphrenic spaces on the right and left respectively.

Figure 27. Subphrenic spaces on the right side. Note that the right posterior superior space is really a recess arising from the right subhepatic space.

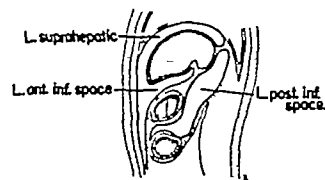


Figure 28. Subphrenic spaces on the left side. The left posterior-inferior space is the lesser peritoneal sac.

monitis, empyema, bronchopleural fistula, lung abscess, perforation of the diaphragm, pericarditis, perforation into a hollow abdominal viscus, spontaneous drainage to the outside, generalized peritonitis and hepatic suppuration. Occasionally death may supervene without complications. Complications are the result of delayed diagnosis and treatment and markedly influence the prognosis. The mortality is three times as high when thoracic complications have been allowed to develop as when they are absent.

Clinical picture The length of time that may elapse between operation or the onset of the primary disease on the one hand, and the time the diagnosis is made, on the other varies from a few days to over a year and the average is five months. A long latent period has always characterized a number of subphrenic abscesses but has been noted in an increasing proportion of cases since the introduction of antibiotics.

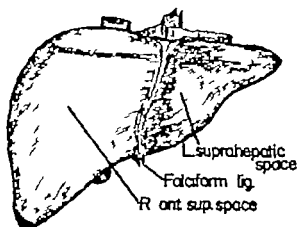
The onset of clinical symptoms may be abrupt, insidious or may blend imperceptibly with the initiating intra-abdominal infection.

nose and treat and because the life of the patient may depend upon adequate management. Untreated, they carry a 90 per cent mortality. Improperly treated, that is drained through the pleural or peritoneal cavity the mortality rate is 35 per cent to 40 per cent. Early diagnosis and proper treatment reduce the mortality rate to approximately 10 per cent.

Anatomy The location of a subphrenic abscess influences the clinical picture and the surgical approach indicated to provide drainage. The subphrenic region is subdivided into various spaces. A subphrenic abscess, because it is walled off by inflammatory adhesions, may not exactly coincide with the space in which it is situated. As a

rule, an abscess is found to occupy only one subphrenic space. When the right posterior superior space is involved, however the right subhepatic space is frequently involved by an extension of the process.

There are six intraperitoneal subphrenic spaces or potential spaces. The liver conveniently divides the subphrenic compartment into suprahepatic and subhepatic areas and the falciform ligament and ligamentum venosum demarcate the right and left sides (Figs. 20, 21, 25 and 26). There are three spaces above the liver and three below there are three on the right and three on the left

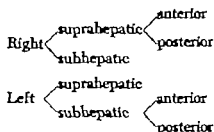


Figures 25 and 26 Views of the superior and inferior surfaces of the liver, showing the ligaments attached to it and the potential subphrenic spaces demarcated by the liver and its ligaments

Figure 25. Superior surface of liver showing the right anterior-superior and the left suprahepatic spaces of the subphrenic compartment. Compare with Figure 20.



Figure 26. Inferior surface of the same liver, which has been rotated upward 180 degrees. Compare with Figure 21.



On the right side there are two suprahepatic spaces and one subhepatic space (Fig. 27). The right anterior-superior space is a large space between the right lobe of the liver and diaphragm which is limited posteriorly by the anterior leaf of the coronary ligament and which communicates with the general peritoneal cavity anteriorly over the free edge of the liver. The right posterior-superior space is a small recess bounded by diaphragm, liver and posterior leaf of coronary ligament. This right posterior superior space is really an upward extension of the right subhepatic space. The reason for designating the right posterior superior space as a separate subphrenic space is that it is the site of a subphrenic abscess more often than any other. Exudate from the right paracolic gutter drains to this area in a recumbent patient. The right subhepatic space corresponds to the hepatorenal pouch of Morison.

On the left side, there is only one suprahepatic space but two subhepatic spaces (Fig. 28). The left suprahepatic space lies between the left lobe of the liver and the diaphragm and is bounded posteriorly by the left triangular ligament. Anteriorly this space communicates over the liver edge with the left anterior-inferior space. The left anterior inferior space lies below the left lobe of the liver in front of the gastrophrenic ligament, stomach and gastrocolic ligament, and it is sometimes designated the "perigastrotic space." The left posterior inferior space corresponds to the lesser sac.

have been carried out, there is still doubt about the presence of a subphrenic abscess. Surgical exploration may be necessary as an ultimate diagnostic procedure. Aspiration is not a permissible diagnostic or therapeutic procedure.

Prophylaxis. Measures for prevention of subphrenic abscesses are identical with those carried out in the prevention and treatment of peritonitis. Of particular importance

is positioning of the patient so that infective exudate drains toward the pelvis and away from the subphrenic area.

Treatment The treatment consists of both supportive measures and surgical drainage. It is absolutely essential that drainage be carried out by a route that avoids contamination of the unprotected pleural or peritoneal cavities. For abscess of the posterior superior and subhepatic spaces on the right side the "extrascrota route" of Ochsner and Nather through the bed of the twelfth rib is employed. Although it is true that one actually goes through peritoneum to drain the subhepatic abscess the suppurative process is walled off anteriorly and there is no general dissemination of infection. For the right anterior superior and left suprahepatic spaces, the anterior incision of Clairmont and Meyer along the costal margin, is used. Left anterior inferior and left posterior inferior space infections are sufficiently well walled off by omentum and transverse colon that they may be drained by cautious approach through the anterior abdominal wall.

After free drainage has been established, several soft rubber drains are put into the depth of the cavity and are slowly withdrawn over a period of several weeks. If fever does not subside rapidly after drainage serious consideration of the possibility of a second subphrenic abscess is indicated and appropriate investigation initiated.

ADHESIONS AND BANDS

Adhesions are abnormal attachments between peritoneal surfaces they may be either fibrinous or fibrous. A band is a long, narrow fibrous adhesion.

Etiology Most adhesions are inflammatory in origin and follow either a surgical operation or peritonitis. Some adhesions are thought to be congenital. Others are due to mechanical violence and foreign bodies, but for the most part they may be considered to be inflammatory.

The amount of fibrin produced from the inflamed mesothelial surface of the peritoneum varies according to the chemical irritant or the microorganisms involved. If the action of the irritant is of only a few days' duration, the fibrin is usually absorbed. Intact mesothelium is necessary for resorption of fibrin. If the fibrinous adhesions are not absorbed for any reason, they are invaded by fibroblasts and capillaries and organization begins between five and ten days after the initial insult.

Factors favoring the development of



Figures 29 and 30: Posteroanterior and lateral x-rays of chest. The patient has a subphrenic abscess involving the right anterior superior space. The films are unusual in that one seldom sees the typical features quite so well demonstrated.

Figure 29 Posteroanterior view. Note the elevation and thickening of the right hemidiaphragm and the presence of an air-fluid level beneath it.



Figure 30. Lateral view. The same three diagnostic features are seen.

Any abscess produces two groups of manifestations (1) the systemic indications of suppuration, (2) the symptoms and signs which are peculiar to the location of the abscess. The inaccessible and deep-seated location of subphrenic abscess results in the localizing manifestations becoming apparent late in the course of the disease. Typically subphrenic abscess is characterized early by systemic evidences of an abscess and little to indicate its location. Maignot quotes Barnard in the well known axiom "Signs of pus somewhere signs of pus nowhere." At the present time the problem of diagnosis is often made still more difficult by the use of antibiotics.* These agents may suppress for a time the systemic manifestations of the disease while the attenuated pathologic process slowly progresses.

The systemic manifestations of an untreated subphrenic abscess are those of any suppurative process: spiking fever, chills, rapid pulse, dehydration, toxemia, and leukocytosis. If the infection has been masked by antibiotics, there may be little or no fever and only such nonspecific symptoms as weakness, malaise, anorexia and, as Blades has expressed it, "a lack of a sense of well-being." It is apparent that if antibiotics are being administered, minimal systemic symptomatology calls for a thorough inquiry and examination for local symptoms and signs.

The most important of the local symptoms is pain, which is usually present but which varies greatly in intensity. The pain is felt in the upper abdomen, along the costal margin, in the flank and renal region, or in the neck or shoulder. The site of pain is of some localizing value. Pain is aggravated by deep inspiration or movements of the trunk. In addition, there may be hiccup, cough or dyspnea.

The most consistent and valuable of the local physical findings is tenderness, the location of which may suggest the site of the abscess. There may be tenderness over the twelfth rib posteriorly with a posterior su-

perior space abscess, tenderness along the costal margin in an anterior superior space infection and tenderness with muscle guarding over the upper rectus in a subhepatic space abscess. Tenderness between the two heads of the sternocleidomastoid muscle due to irritation of the phrenic nerve has been described. There may be limitation of respiratory excursion, findings of pleural effusion, elevation and limited movement of a hemidiaphragm or crepitations in a lung base. Occasionally four distinct levels can be percussed: resonance (normal lung), dullness (pleural effusion), resonance (gas in abscess) and dullness (fluid of abscess and liver). In suprahepatic infections the liver will be displaced downward.

Minimal clinical suspicion demands preliminary x ray studies. If fluoroscopy or chest films in full inspiration and full expiration show elevation, limited movement or thickening of a hemidiaphragm or pleural effusion, a full-scale radiologic investigation is necessary (Figs. 29 and 30). This includes planigrams and views in several positions with a horizontal x ray beam in an attempt to demonstrate an air fluid level and to try to distinguish elevated diaphragm from pleural fluid. If these studies are unrewarding, they may have to be repeated, because it is absolutely essential to establish the diagnosis and highly desirable to determine the location, should a subphrenic abscess exist.

Diagnosis The first requisite for early diagnosis is awareness of the disease. The very obvious systemic manifestations without other apparent cause demand a searching investigation for physical signs and radiologic evidences of subphrenic abscess. If antibiotics are being administered, a still higher index of suspicion is necessary. If a patient has recently undergone surgery or has had an intra-abdominal infection and has only minimal symptoms under antibiotic cover physical and radiologic examination is warranted. If no evidence of subphrenic abscess is found at this point, antibiotic therapy should be discontinued and the disease allowed to declare itself. A swinging fever and other full blown systemic manifestations, as well as localizing symptoms and signs, will soon become apparent if the vague symptoms have been due to a suppressed subphrenic infection. It is better to allow the disease to become manifest and then proceed with proper therapy than to allow the masked process to progress.

If after all other investigative measures

*The administration of antibiotics to treat a fever of undetermined origin is wrong in principle. When a patient develops a fever five to ten days after operation or intra-abdominal infection, however, and conscientious investigation reveals no cause, giving antibiotics for a few days is reasonable and justified. No doubt this practice aborts many subphrenic infections, although some progress to suppuration despite the drugs. The physician must appreciate that he is increasing his responsibility to detect lesions which may be masked by the drugs when he elects such a course.

formed the thirty first operation for adhesions. This series of successive operations may last, as Hertzler stated "as long as the patient does, or the hopeful persistence of the surgeon endures." The most satisfactory solution to this problem is to recognize that a patient of this kind will inevitably re-form adhesions and to replace haphazard adhesions with an organized pattern of non-obstructing adhesions such as one obtains with the Noble plication procedure.

INTRABDOMINAL TRAUMA

Abdominal injuries like peritonitis have afflicted mankind from the remote past to the present day. It is a sad reflection upon our species that an undue proportion of these have been sustained in warfare* and today a very large number of abdominal injuries result from automobile accidents. Abdominal trauma makes up approximately 10 per cent of incapacitating injuries.

Injuries to the abdomen may be divided into two groups, *penetrating* and *nonpenetrating*, the latter being more common. These may be subdivided into injuries of solid or hollow viscera.

All types of abdominal trauma, penetrating or nonpenetrating, and regardless of the organ injured, may produce two effects—shock and peritonitis—singly or together. At the present time one-half to two-thirds of deaths from abdominal injuries are due to shock. Modern methods of treating peritonitis have greatly reduced the mortality from this complication.

PENETRATING INJURIES

A penetrating wound is one in which a foreign body has entered or traversed the abdominal cavity.

Penetrating injuries of the abdomen are usually produced by weapons and are important in military surgery. They are by no means rare among civilians. Loria states that the Charity Hospital in New Orleans, in a fifteen-year period, admitted over 800 such patients about 60 per cent of the injuries were due to firearms and most of the remainder to various types of sharp instruments.

Wounds of the abdominal wall should be considered penetrating until proved otherwise. An external wound of the chest, back, buttock or perineum in fact any wound from the shoulders to the knees may penetrate the abdominal cavity.

Loria states that the Ionian Greek word for physician, *lotros*, means "an extractor of arrows."

If there are wounds of entry and exit the path of the blade or missile unless it has been deflected by bone may be plotted as a straight line this is of some value in determining the probability of injury to the fixed organs. Any penetrating wound of the abdomen must be assumed to have perforated bowel wall unless there is very good evidence to the contrary.

The organ most frequently injured is small intestine. Less frequently there is injury to liver, spleen, large bowel, stomach, mesenteries and major vessels. Several abdominal organs may be injured and there may be associated injuries often of the thorax or urinary system. Trauma to multiple organs markedly increases the mortality.

The history should include information on the type of weapon and the position of the patient when wounded. The patient's symptoms (e.g. shoulder tip pain) will be of some value in diagnosing and localizing injuries but one should not attach too much importance to the patient's denial of symptoms especially if he is in shock or has multiple injuries. Often by the time a patient complains of symptoms, much valuable time has been lost.

Physical examination yields more information than does the history. The circulatory status, the blood pressure and the pulse and their response following treatment, are of the utmost importance in determining the presence of continued bleeding. Shock is to be expected, but if ordinary resuscitative measures restore pulse and blood pressure to normal and these remain stable, one may assume that there is no continuing hemorrhage.

The appearance of the wound may be deceptive. A small wound may be the only visible evidence of an underlying potentially lethal injury.

The most reliable evidence of injury to a hollow viscus, according to Rob, is the absence of bowel sounds. If they remain absent for over an hour abdominal exploration is mandatory. Abdominal tenderness and guarding may be present with either intra-peritoneal blood or injury to a hollow viscus. Rectal examination should be carried out and it should be ascertained that the patient can pass urine normally. Stool and urine should be checked for blood.

If there is any likelihood of a kidney injury an intravenous pyelogram should be obtained early. The kidneys will usually excrete the dye even in the presence of moderate shock. This is essential not only to

fibrous adhesions following chemical or bacterial peritonitis include (1) continuing action of the irritant, (2) necrosis of the mesothelium due to intense action of the irritant, (3) a predisposition in certain individuals which is not well understood.

A surgical operation promotes fibrinous exudation by mechanical factors i.e., handling viscera, sponges, instruments, foreign bodies, blood and, in some cases, low grade and unsuspected infection. Fibrinous adhesions become fibrous in postoperative patients for the same reasons that they do in postperitonitic patients, but in the former there may be an additional factor of denudation of mesothelium—areas which are not adequately reperitonealized at the conclusion of operation. Fortunately even fibrous adhesions may become attenuated and disappear in time.

Formation of adhesions is a protective process without which irritants could not be localized and abdominal surgery would be impossible. These beneficial effects should be considered before attempts are made to abolish adhesion formation by means of drugs and chemicals.

Pathology The chief complication of adhesions and bands is intestinal obstruction. At the present time this lesion has displaced external hernia as the most common single cause of mechanical intestinal obstruction. The adhesions and bands may involve intestine, mesentery, parietes, omentum or any other abdominal structure. They are usually fibrous by the time they give rise to obstruction. Small bowel is most often obstructed, usually the distal ileum. This is probably because more inflammations and operations occur in the right lower quadrant than in any other location.

Adhesions and bands obstruct the bowel by several mechanisms. Adhesions by contraction of the fibrous tissue may kink compress or otherwise distort the bowel to cause a nonstrangulating obstruction. A pocket may form into which bowel prolapses or a narrow aperture may be produced through which a loop of bowel may herniate. A band may also serve as a pivot point for a volvulus.

Mechanical bowel obstruction due to fibrinous adhesions is relatively uncommon and usually occurs during the first week or ten days after operation or peritonitis.

Clinical Picture. Adhesions may produce symptoms other than those of intestinal obstruction but this happens rarely and such a diagnosis should be made with reserva-

tions. The great majority of patients present with a mechanical small bowel obstruction and "a scar on the belly." The obstruction may be strangulating or nonstrangulating, a differentiation which frequently cannot be made with assurance on clinical grounds.

Prevention. There is no reliable way by which the formation of adhesions can be prevented, even if it were desirable to do so. The most a surgeon can hope to accomplish is the reduction of adhesion formation to a minimum. Of all the methods that have been tried to minimize adhesions, nothing has been shown to be as worthwhile as careful attention to the details of surgical technique.

The points of surgical technique that are usually effective in reducing the number of adhesions are (1) all denuded areas should be paid to hemostasis and blood should be evacuated from the peritoneal cavity at the end of the operation, (2) tissues should be handled gently, (3) warm moist sponges and padded retractors should be used, (4) preoperative bowel preparation by mechanical cleansing and enteric antibiotics and sulfonamides before intestinal surgery is helpful, (5) the area about an open viscus should be packed off and spillage should be avoided, (6) drains should be used where indicated but should be removed as soon as it is safe to do so, (7) local use of sulfonamides and antibiotics has no advantage over systemic administration and these chemicals may act as foreign bodies and irritants.

Treatment. When a mechanical small bowel obstruction develops within a week or ten days after surgery or after peritonitis without surgical operation, and one believes that the obstruction is due to fibrinous adhesions, the patient may be treated with supportive measures and a long intestinal tube. The patient must be watched carefully and, if there is no response to this management, operative intervention may be necessary. As a rule the tube will pass through the small bowel and the obstruction will be relieved.

In cases of mechanical small bowel obstruction due to fibrous adhesions, the patient will come to surgery. Supportive measures and the use of the long tube are part of the preoperative preparation and not a substitute for operation.

In patients with a predisposition to form adhesions, ordinary methods of prophylaxis and treatment will prove futile. Wangenstein writes of one such patient on whom he per-

formed the thirty first operation for adhesions. This series of successive operations may last, as Hertzler stated "as long as the patient does, or the hopeful persistence of the surgeon endures." The most satisfactory solution to this problem is to recognize that a patient of this kind will inevitably re-form adhesions and to replace haphazard adhesions with an organized pattern of non-obstructing adhesions such as one obtains with the Noble plication procedure.

INTRABDOMINAL TRAUMA

Abdominal injuries, like peritonitis, have afflicted mankind from the remote past to the present day. It is a sad reflection upon our species that an undue proportion of these have been sustained in warfare* and today a very large number of abdominal injuries result from automobile accidents. Abdominal trauma makes up approximately 10 per cent of incapacitating injuries.

Injuries to the abdomen may be divided into two groups—penetrating and nonpenetrating, the latter being more common. These may be subdivided into injuries of solid or hollow viscera.

All types of abdominal trauma, penetrating or nonpenetrating, and regardless of the organ injured, may produce two effects—shock and peritonitis—singly or together. At the present time one-half to two-thirds of deaths from abdominal injuries are due to shock. Modern methods of treating peritonitis have greatly reduced the mortality from this complication.

PENETRATING INJURIES

A penetrating wound is one in which a foreign body has entered or traversed the abdominal cavity.

Penetrating injuries of the abdomen are usually produced by weapons and are important in military surgery. They are by no means rare among civilians. Loria states that the Charity Hospital in New Orleans in a fifteen year period, admitted over 800 such patients about 60 per cent of the injuries were due to firearms and most of the remainder to various types of sharp instruments.

Wounds of the abdominal wall should be considered penetrating until proved otherwise. An external wound of the chest, back, buttock or perineum, in fact any wound from the shoulders to the knees may penetrate the abdominal cavity.

Loria states that the Ionian Greek word for physician, *letras*, means an extractor of arrows.*

If there are wounds of entry and exit, the path of the blade or missile unless it has been deflected by bone may be plotted as a straight line. This is of some value in determining the probability of injury to the fixed organs. Any penetrating wound of the abdomen must be assumed to have perforated bowel wall unless there is very good evidence to the contrary.

The organ most frequently injured is small intestine. Less frequently there is injury to liver, spleen, large bowel, stomach, mesenteries and major vessels. Several abdominal organs may be injured and there may be associated injuries often of the thorax or urinary system. Trauma to multiple organs markedly increases the mortality.

The history should include information on the type of weapon and the position of the patient when wounded. The patient's symptoms (e.g., shoulder tip pain) will be of some value in diagnosing and localizing injuries, but one should not attach too much importance to the patient's denial of symptoms, especially if he is in shock or has multiple injuries. Often by the time a patient complains of symptoms, much valuable time has been lost.

Physical examination yields more information than does the history. The circulatory status, the blood pressure and the pulse, and their response following treatment, are of the utmost importance in determining the presence of continued bleeding. Shock is to be expected, but if ordinary resuscitative measures restore pulse and blood pressure to normal and these remain stable, one may assume that there is no continuing hemorrhage.

The appearance of the wound may be deceptive. A small wound may be the only visual evidence of an underlying potentially lethal injury.

The most reliable evidence of injury to a hollow viscus, according to Rob, is the absence of bowel sounds. If they remain absent for over an hour abdominal exploration is mandatory. Abdominal tenderness and guarding may be present with either intra-peritoneal blood or injury to a hollow viscus. Rectal examination should be carried out and it should be ascertained that the patient can pass urine normally. Stool and urine should be checked for blood.

If there is any likelihood of a kidney injury an intravenous pyelogram should be obtained early. The kidneys will usually excrete the dye even in the presence of moderate shock. This is essential not only to es-

establish whether a kidney has been injured, but also whether the opposite kidney is functioning normally. Similarly if there is any possibility of a bladder injury there is only one reliable diagnostic procedure: cystography following instillation of 250 cc. of radiopaque dye, 5 per cent sodium iodide.

A chest film and plain supine and upright (or left lateral decubitus) films of the abdomen should be taken to search for free intraperitoneal and retroperitoneal gas, missile fragments, ileus and evidence of fluid.

The supportive treatment will often be initiated even before preliminary assessment of the patient is completed. Blood is drawn for crossmatching, even if there is no immediate intention of giving a transfusion. Intravenous fluids are started through a large needle or cut-down. A Levin tube of generous diameter is placed in the stomach for decompression. In the shocked patient urinary output as measured by a Foley catheter in the bladder is often a useful guide to the quantity of intravenous fluid required. A penetrating wound of the abdomen is one of the few instances in which prophylactic administration of antibiotics is justified. Narcotics are not administered to the patient with abdominal trauma until the surgeon responsible for the case has had an opportunity to examine the patient and has made a decision regarding operative intervention.

The patient with a penetrating injury to the abdomen will almost always require exploration. In this regard, Sir Gordon Gordon Taylor states "A penetrating wound in the abdomen probably means a penetrating wound of the bowel or other abdominal viscus and demands the earliest surgical intervention, unless a wisdom of prescience born of great experience justifies restraint. The only two instances when laparotomy may be deferred are (1) when a patient is moribund, (2) in the occasional instance in which thorough and repeated examination of the patient reveals no abnormality whatsoever."

Usually a patient should not be taken to the operating room until he has been resuscitated and his pulse and blood pressure are stabilized (e.g. to the table tilt test). There are times, when a patient is actively bleeding as fast as one can pump blood into him in which immediate intervention is indicated. If one is able to locate a controllable hemorrhage the patient's condition will improve almost "miraculously" on the operating table.

When resuscitation may be carried out at a more leisurely pace the outlook for the patient is improved, but operation should not be delayed beyond a few hours. Three factors influence the mortality: (1) the severity of the injury, (2) the duration of shock and (3) the duration of peritoneal contamination. The objectives of operation are to control bleeding and to prevent continuing contamination of the peritoneal cavity.

NONPENETRATING INJURIES

These are also known as closed or subcutaneous injuries, or injuries due to blunt trauma. They are more important than penetrating injuries because they are not only more common but they are more difficult to manage, since clear-cut indications for operative intervention may be absent.

Approximately half of the serious nonpenetrating injuries to the abdomen which occur in North America are caused by automobile accidents. The steering wheel is a particularly dangerous instrument. Falls, athletic injuries, mining accidents and being struck by falling objects account for a certain number.

The forces that produce the injury to the abdominal contents are of four types: (1) an anterior posterior or lateral squeezing force which crushes the viscus, usually against unyielding bone, (2) a tangential force which moves a viscus beyond the limits of its mobility with a tearing of its attachments or capsule, (3) a sudden compressing force which, in effect, bursts a hollow organ as if it were a paper bag, (4) a blasting force with widespread shattering of tissues with petechial hemorrhages.

Fixed organs are more likely to be injured than those which are more mobile. The organs injured, in approximate order of frequency are liver, spleen, small bowel, large bowel, kidneys, stomach, bladder, diaphragm (left side) and, rarely a pregnant uterus. The proximal jejunum and distal ileum are the parts of the small intestine most frequently injured (near points of fixation) and bowel is more often injured on its antimesenteric border. The same lesion may spill fecal material and bleed profusely. The vulnerable portion of the duodenum is the third part where it crosses the body of the third lumbar vertebra. A hollow viscus is always more susceptible to injury when distended than when empty.

The injuries frequently involve more than one organ, in which case the mortality rate

s increased from less than 10 per cent to more than 30 per cent. The lethal factors—shock and peritoneal contamination—frequently coexist in the same patient.

Delayed hemorrhage may occur. The most frequent type presents in delayed rupture of the spleen. This variety of injury occurs once in seven instances of splenic rupture. Delayed intra-abdominal hemorrhage may also result from injury to liver to duodenum and pancreas and to kidneys.

The difficult clinical problem in cases of blunt trauma to the abdomen is to establish the diagnosis of a potentially lethal injury sufficiently early to avoid the high mortality rate which results from prolonged contamination of the peritoneal cavity. A ruptured hollow viscus may produce few symptoms during the most favorable time for closure. The physical findings may be no more than could be explained on the basis of a small amount of blood in the peritoneal cavity or on the basis of a retroperitoneal hemorrhage. Routine operation is not a solution to this problem because a negative exploration also carries an increased mortality. The only answer is thorough initial investigation and frequent re-evaluation by the same surgeon.

The history may be very misleading. The initial trauma may be remarkably trivial. A history of a severe blow to the abdomen followed by rapid recovery and an interval of several hours without symptoms in no way rules out intra-abdominal injury. The nature and location of the blunt trauma will enable the physician to visualize the possible underlying injuries. The presence and location of pain are of some diagnostic value. A history of persistent vomiting is suggestive of bowel rupture, particularly if the vomitus is bloody. Difficulty in voiding or hematuria will direct attention to the urinary tract.

The history will only suggest the presence of a serious lesion, but this diagnosis must be made from the physical findings supported by laboratory aids, chiefly the x ray films.

The patient with significant nonpenetrating trauma to the abdomen will usually be in shock on admission. Resuscitation with whole blood should be carried out concomitantly with the clinical assessment of the patient. Military experience in Korea has indicated that severely injured persons may require quantities of blood far in excess of the amount of blood lost from hemorrhage or the amount of fluid displaced into traumatized tissues. These resuscitative measures

are in a sense diagnostic as well as therapeutic. Failure of the patient to respond to liberal administration of whole blood or a temporary response to blood indicates either very severe trauma or continued hemorrhage.

One should search for an area of contusion on the abdominal wall. Tenderness guarding and rebound tenderness are usually due to rupture of a hollow viscus but can be caused by blood in the peritoneal cavity. The absence of bowel sounds always suggests severe injury but it is not by itself an absolute criterion for exploration, as paralytic ileus may be caused by a retroperitoneal hematoma. Obliteration of liver dullness is almost pathognomonic of rupture of some part of the gastrointestinal tract. Shifting dullness in the flanks and fluid in the cul-de-sac on rectal examination are occasionally noted.

Scout films of the abdomen and chest should be taken. Fractured lower ribs should alert one to the possibility of rupture of spleen or liver but rib fractures are found in only 50 per cent of fatal cases. Finding of free gas on the upright or left lateral decubitus films will make exploration mandatory. A fracture of the pelvis in the vicinity of the pubic rami or which opens up the pelvic ring makes a cystogram absolutely indispensable. If there is any suspicion of kidney trauma, intravenous pyelography should be carried out early.

Peritoneal aspiration under local anesthesia is a useful diagnostic adjunct. The finding of blood, bile, fecal material or fluid with a high amylase content may influence the management.

Whether or not operation is indicated, a program of supportive therapy should be instituted.

The objectives of operation are the same for both penetrating and nonpenetrating trauma to the abdomen: control of hemorrhage and prevention of continued contamination of the peritoneal cavity. The treatment of bleeding from a ruptured spleen has high priority and the hemorrhage often becomes profuse when the abdomen is opened. Splenectomy is the only permissible procedure. The essential considerations in the treatment of liver injuries are (1) to stop bleeding, (2) to excise devitalized fragments and (3) to drain. Bleeding may be controlled with mattress sutures and hemostatic sponges (Gelfoam and Oxycel) packs should not be used. Drainage is essential because bile leakage will occur and may

lead to fatal peritonitis. Trauma to the pancreas usually requires only drainage. An opening in the stomach may be treated by excision of wound edges and closure with sutures. The small bowel should be systematically inspected from the ligament of Treitz to the ileocecal valve; wounds are treated either by resection or by a method of suture that does not narrow the lumen. The anterior surface of the duodenum should be inspected for an intraperitoneal rupture which can usually be closed with sutures. At the same time, one should be alert for bile stained blood and gas behind the peritoneum, indicating a retroperitoneal rupture of the duodenum; this requires mobilization of the duodenum, suture and drainage. Openings in the large bowel beyond the hepatic flexure must either be exteriorized or sutured and protected with a proximal colostomy. It is not so important to protect a suture line in the cecum or ascending colon, but a catheter cecostomy is often useful. A wound of the gallbladder is treated by cholecystectomy. Injury to the common bile duct is treated by suture and T tube drainage. Bladder wounds are closed, the usual care being exercised not to put chromic catgut or nonabsorbable sutures through the mucosa. A urethral catheter or suprapubic tube is inserted and the extraperitoneal tissues about the bladder are drained. Nephrectomy is seldom necessary unless the kidney is severely fragmented.

Postoperatively the patient is maintained on supportive therapy until recovery is complete.

HEMOPERITONEUM

Hemoperitoneum may be either *traumatic* or *spontaneous*. The trauma may be trivial, especially, if bleeding comes from a previously diseased organ. Delayed hemorrhage from nonpenetrating trauma to the abdomen may also occur.

The lesions which may cause spontaneous hemoperitoneum include ectopic pregnancy, blood dyscrasias, rupture of an artery which is the seat of a degenerative process, torsion of omentum, spleen, fibroid tumor or ovarian cyst, mesenteric vascular occlusion and pancreatitis.

The most probable source of a large amount of blood in the male is rupture of the spleen. In the female of childbearing age it is ectopic pregnancy.

Hemorrhage should be controlled and the blood evacuated. Blood in the peritoneal cavity is a much greater hazard to the pa-

tient than any possible benefit that might accrue from having this blood as a source of iron.

PNEUMOPERITONEUM

Pneumoperitoneum is the presence of gas within the peritoneal cavity. The detection of free intraperitoneal gas is of great value in the diagnosis of a perforated abdominal viscus. There are however other important causes of pneumoperitoneum which are included in the following etiologic classification.

CLASSIFICATION OF PNEUMOPERITONEUM

A. From Within the Lumen of the Gastrointestinal Tract

1. Perforated peptic ulcer
2. Other perforative disease
3. Blunt and penetrating trauma to the abdomen
4. Endoscopic instrumentation
5. Infections
6. Postanesthetic

B. From Outside the Lumen of the Gastrointestinal Tract

1. Postoperative
2. Diagnostic
 - (a) Rubin Test
 - (b) Needle
3. Therapeutic
4. Penetrating trauma

C. Idiopathic

Perforated Viscus. Perforated peptic ulcer is the commonest disease process producing pneumoperitoneum. The demonstration of free intraperitoneal air in a patient with an acute abdomen usually indicates a perforation of the gastrointestinal tract.

Demonstrable pneumoperitoneum occurs with more than half of perforated peptic ulcers. The detection of air depends upon many factors, including (1) the time elapsed since perforation, (2) the location and size of the perforation, (3) the effectiveness of reaction in walling off the perforation, (4) the amount of air in the stomach at the time of perforation and (5) the amount swallowed subsequently.

Benign and malignant ulcerating lesions and obstructing lesions in which perforation of the gastrointestinal tract occasionally occurs may produce pneumoperitoneum.

Free gas in the peritoneal cavity seeks the highest level and, in the upright position, tends to accumulate under the diaphragm. Although absence of liver dullness in the midaxillary line is clinically indicative of

pneumoperitoneum, accurate detection requires careful radiologic examination (Figs. 31 and 32). A few cubic centimeters of air may be seen on a scout film of the abdomen if sufficient time is allowed for the gas to collect in whatever part of the cavity is uppermost. Erect and left lateral decubitus views are commonly used.

Traumatic. Pneumoperitoneum following blunt trauma to the abdomen indicates a perforated viscus. Pneumoperitoneum following a penetrating or perforating abdominal wound reveals only peritoneal entrance, since air may enter with the missile. Visceral involvement, however, is common if the peritoneum is entered and the detection of free intraperitoneal air is a positive indication for laparotomy.

Accidental. Instrumental visualization of abdominal organs, often accompanied by air insufflation, may perforate a normal or diseased viscus and produce pneumoperitoneum. Perforation during sigmoidoscopy under direct vision is rare. Pneumoperitoneum occasionally follows gastroscopy, but usually gross perforation is not found at laparotomy. In the absence of clinical peritonitis these patients have been successfully treated conservatively.

Infections. Certain gas-forming organisms may cause pneumoperitoneum in an intra-abdominal abscess. *Clostridium welchii* and related bacteria are the commonest infecting organisms that produce gas, but *Escherichia coli*, anaerobic streptococci and *Proteus* have also occasionally been cultured. The formation of gas in an abdominal abscess may be a valuable aid in diagnosis and localization.

Postanesthetic. Pneumoperitoneum following surgery is occasionally related to anesthesia. Excessive airway pressure during assisted respiration, or rapid oxygen flow through a nasal catheter may produce gastric dilatation and, rarely, gastric perforation. Intraperitoneal gas following anesthesia is not always associated with perforation or peritonitis; the gas may diffuse beneath the mucosa along the vessels of the stomach into the peritoneal cavity.

Postoperative. The most frequent cause of pneumoperitoneum is abdominal surgery. The peritoneal cavity is normally a potential space in which there is a negative pressure. When the peritoneum is opened at operation an inrush of air may be noted. The duration of postoperative pneumoperitoneum is variable; small quantities of air are usually absorbed within a few days. In the majority of patients the air has disappeared

by fourteen days but rarely it may persist for as long as six weeks.

There is no apparent correlation between pneumoperitoneum and postoperative pulmonary complications. The presence of intraperitoneal air following surgery may



Figures 31 and 32: Posteroanterior and lateral x-rays of chest. The patient has a perforated peptic ulcer, the most common cause of pneumoperitoneum other than laparotomy.

Figure 31: Posteroanterior view showing a thin rim of free air beneath both leaves of the diaphragm.



Figure 32: Lateral view same patient. These films were taken in the upright position. If patient is too ill to be upright for a few minutes before films are taken, the left lateral decubitus position should be used, with patient lying on his left side.

confuse the assessment of postoperative abdominal complications.

Diagnostic. Direct injection of gas into the peritoneal cavity to outline the abdominal and pelvic organs has been used in selected cases as an aid in the radiologic diagnosis.

The potential communication between the peritoneal cavity and the exterior existing in the normal female via the fallopian tubes and the uterine cavity is utilized in the Rubin test.

Therapeutic. Artificial pneumoperitoneum has been used in the treatment of pulmonary and abdominal tuberculosis for many years. Five hundred to 1000 cc. of air or oxygen is injected directly into the peritoneal cavity with periodic refills. Elevation of the diaphragm with reduction of pulmonary volume tends to promote collapse of tuberculous cavities, particularly if they are located in the base of the lungs. This treatment often combined with phrenicectomy and chemotherapy is considered of value in some instances. Another use therapeutically is the preparation of the patient with a very large ventral hernia for operation. The air is introduced into the peritoneal cavity while the hernia is under control with strapping. This has been a very useful procedure in selected cases.

Idiopathic or Undetermined Cause. Spontaneous idiopathic pneumoperitoneum occurs rarely with no demonstrable perforation of a viscus known exogenous cause or underlying disease process. It produces few clinical manifestations and usually subsides without ill effect.

TUMORS OF THE PERITONEUM

The great majority of peritoneal tumors are secondary. Primary tumors of the peritoneum or of any serous membrane are very rare.

PRIMARY TUMORS

Neoplasms may arise from any of the fibrous, fatty, vascular or reticular tissues which lie beneath the peritoneal mesothelium, but such are best regarded as tumors of the viscera, parietes or retroperitoneum. The tumor of mesothelium, the mesothelioma, is the only primary tumor of the peritoneum. Although it is very rare, most pathologists agree that the primary peritoneal mesothelioma is a recognizable entity. Both benign and malignant varieties of mesothelioma exist. The lesions may be localized or diffuse and they occur in plaque-like and nodular forms. The tumor may

spread over and encompass the viscera, producing fibrous thickening of the peritoneum and shortening of the mesentery. Intestinal obstruction, viscid ascites and obliteration of the peritoneal cavity have been reported. Even malignant forms do not invade widely and seldom metastasize.

The neoplasm may have the microscopic appearance of a papillary fibrous, adenar or even squamous cell tumor. It is very difficult for the pathologist to establish the diagnosis on the basis of examination of ascitic fluid or a small amount of biopsy material.

The benign form, if localized, can be effectively treated by surgical excision.

SECONDARY TUMORS

The primary lesions are usually found in stomach, colon, ovary, uterus, breast, biliary system, pancreas, lung and lymph nodes. Most secondary tumors of the peritoneum are metastatic carcinomas. The malignant tumor reaches the peritoneal surface by permeation through the wall of a viscus, by hematogenous and lymphatic spread and occasionally by inoculation at the time of surgery. Once in the peritoneal cavity, tumor may be disseminated over the serosal surface.

The gross appearance of secondary tumors varies widely according to the primary lesion and the mode of spread. The main features are ascites and tumor nodules. The ascitic fluid is usually bloody and often contains exfoliated tumor cells. The tumor ranges from minute deposits, a millimeter or less in diameter, studded diffusely over the peritoneal membrane, to large plaques and masses of neoplastic tissue. As a rule the tumor nodules are less than a centimeter in diameter. The omentum may be so infiltrated with tumor that it is converted into a hard mass. There may be "drop metastases" to the cul-de-sac, giving rise to a rectal shelf. Sometimes a reticular pattern of white lymphatics permeated with tumor is seen beneath the serosa. It may be difficult to distinguish grossly the nodular lesions of secondary peritoneal tumor from those of tuberculosis, fat necrosis or foreign body granuloma.

Not all secondary tumors are malignant. One benign secondary lesion is the "parasitic fibroid," a leiomyoma that has become detached from the uterus and taken up a new attachment on the peritoneal surface. Similarly dermoid cysts of the ovary sometimes become secondarily attached to peritoneum.

An interesting form of secondary tumor of the peritoneum is *pseudomyxoma peritonei* which arises from rupture of a pseudomucinous cystadenoma of the ovary or rarely of the appendix. A lesion of similar appearance may be produced by rupture of a mucocyst of the appendix, in which case the mucus acts as an irritant and produces chemical peritonitis. The mucus-producing cells become implanted on the peritoneum and continue to produce a gelatinous exudate causing the so-called jelly belly.

The patient with metastases to the peritoneum usually has advanced symptoms of the primary lesion. He may complain of pain, discomfort or cardiorespiratory symptoms from abdominal distention. Cachexia, pallor and ascites are often present when the patient is seen for the first time. One may palpate tumor tissue in the form of a primary lesion, a Virchow's node, liver nodules, an omental mass or a rectal shelf.

If there is no evidence of metastatic malignancy other than the ascites, abdominal fluid may be examined cytologically. This examination is subject to the limitations inherent in the cytologic method.

Treatment can only be symptomatic. Abdominal tapping may be done for the comfort of the patient. Instillation of colloidal Au¹⁹⁸ or radiomimetic drugs may reduce ascites when the tumor nodules are only a few millimeters in diameter and there is no block at the porta hepatis.

MESENTERIES AND OMENTUM ANATOMY AND PHYSIOLOGY

The stomach, jejunum, appendix, transverse colon, sigmoid colon, liver and spleen are attached to the posterior wall of the abdominal cavity by mesenteries which carry their respective blood vessels, lymphatics and lymph nodes. The gastrophrenic ligament is the ventral mesentery of the stomach and first part of the duodenum, but it is also in a sense a dorsal mesentery of the liver and gallbladder. The gastrosplenic ligament is continuous with the anterior layer of the greater omentum. The mesentery of the jejunum is described as fan shaped, averaging 12 to 25 cm. in height. Its junction with parietal peritoneum is only 15 cm. long, whereas its other border where it enfolds small bowel, is about 700 cm. in length. This marked disparity in the lengths of its two borders can only be reconciled by numerous folds toward the enteric border. All mesenteries consist of two layers of

mesothelium enclosing a variable amount of fatty and loose connective tissue, vessels and lymph nodes.

The mesenteries of the duodenum, pancreas, ascending colon and descending colon have disappeared in fetal life because of fusion with parietal peritoneum. Fortunately for the surgeon, these planes of fusion can usually be converted into planes of cleavage restoring the primitive state. The "mesentery" thus created has only one layer of mesothelium, the posterior parietal peritoneum, but it carries in its fatty areolar tissue the blood vessels, lymphatics and lymph nodes of the organ in question.

The greater omentum is a double fold of mesentery embryologically. One might expect it to contain four mesothelial layers but the central two have fused and vanished. The size and fat content of the greater omentum are highly variable. Its role as the "abdominal policeman" seems well supported by experimental and clinical evidence. The areolar tissue of the omentum is rich in macrophages. Bacteria or carbon particles injected into the peritoneal cavity are rapidly removed by the omentum and are subsequently seen to be situated in phagocytes beneath its mesothelium. The ability of the omentum to adhere to sites of inflammation and perforation has been mentioned in the discussion of peritonitis. Foreign bodies in the peritoneal cavity such as a bullet or a sponge, are often found completely wrapped in omentum.

The presence of the greater omentum is not always beneficial. Adhesions and bands between the omentum and either parietes or viscera, or openings made in the omentum at surgical operations may lead to intestinal obstruction. Once a patient has had some such difficulty originating in the omentum, surgical excision of the omentum will circumvent further complications.

MESENTERIC LYMPHADENITIS ACUTE NONSPECIFIC MESENTERIC LYMPHADENITIS

Over the past thirty years this disease has been delineated from tuberculous lymphadenitis and has gradually gained wide acceptance as a clinical entity. Its chief importance lies in the fact that it produces the picture of an "acute abdomen" and must be considered in the differential diagnosis of appendicitis.

Acute nonspecific mesenteric lymphadenitis is uncommon and in our experience the

incidence is diminishing. Most patients are between five and fifteen years of age and it is somewhat more frequent in boys.

The etiology is unknown and there may be more than one cause. Cultures from the enlarged mesenteric lymph nodes are usually negative. Associated low-grade inflammatory changes in the appendix and distal ileum have been reported in a large proportion of cases and may have a bearing on the etiology.

The appendix is grossly normal. There is discrete enlargement of the mesenteric lymph nodes in the ileocecal angle particularly those in the juxtaintestinal group. Lymph nodes in this region are prominent in children and normal lymph nodes must be distinguished from those which are pathologic. The nodes vary from soft and pink in the early stage of the disease to firm and white later. Microscopically the lymph nodes show only nonspecific changes: hyperplasia, edema and hyperemia.

The child with acute nonspecific mesenteric lymphadenitis usually presents with colicky abdominal pain vaguely situated about the umbilicus, right side or right lower quadrant. The pain is of variable intensity but in between the cramps the child feels fairly well. There is often a recent upper respiratory infection, which may still be present. There is a history of previous attacks, separated by intervals of several months, in over half the cases. Malaise, anorexia, nausea or vomiting is also frequently present or reported in earlier attacks.

The child does not look or act ill. There is fever usually under 100° F. The patient is flushed and there may be circumoral pallor. The pharynx is often injected and the cervical lymph nodes may be enlarged. Abdominal tenderness is higher more medial, less well localized and more variable from one time to another than in appendicitis. Shifting tenderness (i.e., shift of point of maximum tenderness to the left when child lies on left side) due to change in position of the mesentery and nodes is said to be characteristic but is often absent. The white blood count may be increased to 10,000 to 15,000 per cu. mm.

Indiscriminate appendectomy cannot be condoned. However if one suspects acute appendicitis the matter should be settled by laparotomy. On the other hand, if one can be reasonably confident of a diagnosis of mesenteric adenitis, a few hours of observation may be confirmatory. The signs and

symptoms of this latter disease will subside or improve in less than twenty-four hours.

If at laparotomy one finds a normal appendix and the characteristic lymphadenopathy in the ileocecal angle appendectomy should be carried out. It is not advisable to remove a lymph node for biopsy because bowel may become adherent to the site. Postoperative respiratory complications should be anticipated. A small proportion of these patients may have further attacks after appendectomy but there is an over-all tendency toward recovery.

TUBERCULOUS MESENTERIC LYMPHADENITIS

Involvement of mesenteric lymph nodes by tubercle bacilli is now uncommon. The disease may be primary, in which case the organisms usually, bovine, gain entry through intact mucosa to Peyer's patches and then reach the nodes. The disease may be secondary to tuberculosis of lungs or bowel.

The pathologic picture is that of tuberculous lymphadenitis elsewhere, with tubercle formation, caseation and eventually calcification. The disease may be complicated by peritoneal adhesions, tuberculous peritonitis or secondary pyogenic infection.

The disease is seen in acute and chronic forms and its manifestations are highly variable. The salient features are fever, general ill health, weight loss, diarrhea and cramping abdominal pain.

A ray of the abdomen in a clinically well person may show calcified lymph nodes in the right iliac fossa—the so-called chalky tombstones of tubercle bacilli, which may or may not be dead.

In general, the treatment should be conservative with antituberculous drugs and supportive measures.

MESENTERIC AND OMENTAL CYSTS

A variety of unusual cysts may be found in the mesenteries and omentum. These include (1) enterogenous cysts or congenital reduplications, (2) lymphatic cysts, either cavernous lymphangiomas or cystic lymphangiectasis, (3) dermoid cysts, (4) hydatid cysts.

The cysts may produce no symptoms or only abdominal enlargement and vague discomfort. Occasionally a patient presents with intestinal hemorrhage or obstruction. Cysts of the omentum may give rise to secondary torsion and the clinical picture of an "acute abdomen." The characteristic physical finding is a "floating tumor" one in the

mesentery have greater mobility from side to side than in a vertical direction.

If surgical intervention is necessary the ideal treatment is to shell out the cyst. Unfortunately this is seldom possible in the mesentery as vessels supplying the bowel are adherent to the cyst wall and it may be necessary to resect bowel. A cyst of the omentum is easily treated by excision. If a distal cyst may be treated by deep x-ray therapy or by excision with care not to spill the fluid, which would reinfect the patient or cause a severe anaphylactic reaction.

INFARCTION OF THE OMENTUM

Infarction of the greater omentum may be due to (1) torsion (2) thrombosis, (3) polyarteritis nodosa, (4) embolism (5) trauma, (6) unknown etiology.

Idiopathic segmental infarction of the omentum is rare, reports of only forty cases having been published. It is most often found in well-nourished men in the third decade. The cause is obscure but it is thought to be related to venous engorgement (sometimes seen after a full meal) and mild trauma or increase in intra-abdominal pressure, leading to thrombosis. Usually the right, lower free margin of the omentum is infarcted.

Patients present with steady severe right lower quadrant pain, the onset of which may be gradual or sudden. Nausea and vomiting are unusual. On examination the tenderness is usually higher than one would expect for appendicitis and a mass may be palpable. Cutaneous hyperesthesia is said to be characteristic.

The correct diagnosis has never been made preoperatively; most cases are called acute appendicitis. The finding of a normal appendix and some serosanguineous fluid in the peritoneal cavity should always direct one's attention to disease elsewhere. The infarcted area forms a firm, red to purplish-black mass. Treatment is wide excision of the involved omentum.

TORSION OF THE OMENTUM

Torsion of the omentum may be classified as primary and secondary. Secondary torsion is more common and is subdivided into bipolar and unipolar types depending on whether or not there is a secondary fixation of the omentum. Among the causes are hernias, adhesions, cysts and tumors.

In primary torsion of the omentum there is no apparent cause although vigorous exercise movement of the omentum by intestinal

peristalsis and hemodynamic forces have been blamed. The torsion is always unipolar and may be either complete or incomplete. In the complete type there may be up to six full turns. The omentum is usually quite large and fatty with a long pedicle and a narrow attachment. It is often the right free margin of the omentum that is involved in the torsion.

The clinical picture and treatment of torsion of the omentum are similar to those of infarction of the omentum.

APPENDICES EPILOICAE

The appendices epiploicae are fat-laden pouches of peritoneum which are found on the large bowel and rarely on the appendix. They are usually arranged in two rows: one medial to the taenia libera and one lateral to the taenia omentalis. These are the sites at which some of the vessels enter the bowel wall. One will recall that diverticula also are situated in these positions.

The appendices epiploicae may be the sites of a number of diseases, the most common of which is "epiploic appendicitis." This may be due to torsion or thrombosis and so may be regarded as a miniature counterpart of torsion and infarction of the omentum. Epiploic appendicitis is found chiefly in the sigmoid and cecal regions. Other disease processes in which the appendices epiploicae may be involved include (1) acute and chronic inflammation secondary to diverticulitis, (2) degeneration to form an intraperitoneal loose body, (3) intussusception, (4) incarceration in a hernia.

The presenting symptom of epiploic appendicitis is lower abdominal pain on either the right or left side. The correct diagnosis is seldom made preoperatively. Treatment is excision with care not to open up a diverticulum.

RETROPERITONEAL SPACE

ANATOMY

The retroperitoneal space from the surgeon's viewpoint is a potential space extending from the respiratory diaphragm above to the pelvic diaphragm below. Its posterior boundaries are the vertebral bodies, the psoas and quadratus lumborum muscles and the aponeurotic portions of the transverse abdominis muscles (Fig. 23). Below the iliac crests it is bounded by the sacrum and the psoas and piriformis muscles posteriorly and by the iliacus and obturator internus muscles laterally. The an-

terior boundary of the retroperitoneal space is chiefly the posterior parietal peritoneum, but in addition the anterior boundary is made up of the posterior surface of the liver the ascending and descending portions of the large intestine the retroperitoneal portion of the duodenum and the rectum.

The major organs of the retroperitoneal space are the kidneys and ureters the adrenals and the pancreas. The space is also occupied by the great systemic vessels and their branches, veins of the portal system, lymphatic vessels and lymph nodes somatic nerves sympathetic chains and autonomic plexuses and an abundance of fatty and areolar connective tissue.

RETROPERITONEAL HEMORRHAGE

Bleeding into the retroperitoneal space is a sequel to blunt trauma to the trunk, fractures of the pelvis or lumbar vertebrae especially of their transverse processes and lacerations of the kidney. Because the blood is irritating to both somatic and autonomic nerves the clinical picture of peritonitis may be simulated. There may be abdominal pain nausea vomiting abdominal guarding low-grade fever and leukocytosis. This syndrome may proceed to adynamic ileus characterized by a silent distended abdomen. Except for rupture of an aortic aneurysm and rare instances of severe trauma to the kidney or renal vessels, retroperitoneal bleeding seldom presents as an exsanguinating hemorrhage.

Retroperitoneal bleeding should be recognized early and the ileus and gastric dilatation treated by gastrointestinal decompression. The possibility of a more serious intra-abdominal lesion initially presenting with a similar clinical picture should be considered. The differential diagnosis is difficult and must occasionally be made at laparotomy.

RETROPERITONEAL TUMORS

It is customary to exclude tumors of the kidneys ureters adrenals pancreas and bowel as well as metastatic tumors to the retroperitoneal lymph nodes from the group of neoplasms designated as retroperitoneal tumors.

The most common of the primary tumors seen in the retroperitoneal space are the lymphomas i.e., lymphosarcoma, Hodgkin's disease and reticulum cell sarcoma. The second most common is the liposarcoma—a noteworthy oddity since elsewhere in the body benign fatty tumors are quite common and malignant fatty tumors are almost un-

known. The remaining tumors are those which arise from the mesodermal tissues in the retroperitoneal space. The majority (80 per cent) are malignant, and one-third have metastasized at the time they are diagnosed. Those which are benign tend to recur after removal.

Because the retroperitoneum is a distensible space these tumors may reach very large size before they are diagnosed. About half the patients present with pain and one-third with an abdominal mass. Gastrointestinal symptoms backache pain and swelling in a leg, genitourinary symptoms and fever are occasionally early symptoms.

The most common physical finding is a fixed or movable mass usually nontender. Varicocele edema or varicosities of a leg, a forward protrusion of the liver lumbar dullness on percussion and occasionally a neurologic deficit, especially with dumbbell tumors, may be found.

The chief laboratory aid is the x-ray and the most important investigation is the pyelogram with anteroposterior and lateral views. The ureters which are adherent to the posterior parietal peritoneum are seen to be displaced forward or laterally. The function of each kidney should be assessed, as one kidney may need to be sacrificed at operation in order to encompass the lesion. The next most important radiologic procedure is a gastrointestinal series. Presacral oxygen insufflation may be useful to outline the contents of the retroperitoneal area. Aortography and venography may be useful, as the great vessels are often displaced.

At the time of operative intervention most retroperitoneal tumors will have surrounded or invaded major organs and vessels. Curative surgery is possible in about 20 per cent and the operative mortality ranges from 10 to 25 per cent.

Operation is required to assess the tumor and establish a diagnosis. A proportion of the tumors are amenable to surgical treatment, but each case must be individually evaluated. Close cooperation between the surgeon and the pathologist is necessary in order to determine the curability of the lesion and the extent of surgical extirpation which is justified. It may be necessary to sacrifice one kidney or the vena cava below the renal veins. If complete excision is impossible the residual tumors will be radio-sensitive to some degree in about three-quarters of the cases. These remaining patients should be treated with radiation or radioimimetic drugs.

READING REFERENCES

- Ackerman, L. V.: *Surgical Pathology*. St. Louis, C. V. Mosby Company 1933.
- Ald, J.: *Companion in Surgical Studies*. 2nd ed. London, E. & S. Livingstone 1937.
- Bardeen, C. R., and Lewis, W. H.: The Development of the Limbs, Body-Wall and Back in Man. *Am. J. Anat.* 77 1901-02.
- Baxter, E. H.: Congenital Absence of the Abdominal Muscles. *Ohio M. J.* 28:810, 1932.
- Berens, J. J., Gray, H. A., and Dockerty, M. B.: Subphrenic Abscess. *Surg. Gynec. & Obst.* 96 103, 1953.
- Blades, B.: Subphrenic Abscess (Editorial). *Surg. Gynec. & Obst.* 103:785 1956.
- Bremer, J. L.: *Congenital Anomalies of the Viscera Their Embryological Basis*. Cambridge Mass., Harvard University Press, 1937.
- Brodel, M.: Lesions of the Rectus Abdominalis Muscle Stimulating an Acute Intra-Abdominal Condition. I. Anatomy of the Rectus Muscle. *Bull. Johns Hopkins Hosp.* 61:295 1937.
- Collen, T. S.: *Embryology Anatomy and Diseases of the Umbilicus Together with Diseases of the Urachus*. Philadelphia, W. B. Saunders Company 1916.
- Collen, T. S.: Lesions of the Rectus Abdominalis Muscle Stimulating an Acute Intra-Abdominal Condition. II. Hemorrhage into or Beneath the Rectus Muscle Stimulating an Acute Abdominal Condition. *Bull. Johns Hopkins Hosp.* 61 317 1937.
- Davis, H. A.: *Surgical Physiology*. New York, Paul B. Hoeber 1937.
- Doehauser, J. L.: Primary Acute Mesenteric Lymphadenitis. *Arch. Surg.* 74:528, 1937.
- Fothergill, W. E.: Haematoma in the Abdominal Wall Stimulating Pelvic New Growth. *Brit. M. J.* 1:411 1929.
- Fountain, E. B.: Injuries to the Solid Abdominal Viscera. *Arch. Surg.* 75:740 1937.
- Gaston, B. H., and Mulholland, J. H.: Treatment of Penetrating Abdominal Wounds. *S. Clin. North America* 33:463, 1933.
- Gross, R. E.: *Surgery of Infancy and Childhood*. Philadelphia, W. B. Saunders Company 1933.
- Hertler, A. E.: *The Peritoneum*. St. Louis, C. V. Mosby Company 1919.
- Holmes, E.: Ipsilateral Spastic Rectus Abdominis in a Purely Thoracic Wound. *Surg. Gynec. & Obst.* 82:350, 1946.
- Jamieson, R. A.: Subcutaneous Rupture of the Muscles of the Abdomen. *Brit. J. Surg.* 36:434, 1948-49.
- Jones, F. W.: *Buchanan's Manual of Anatomy*. 8th ed. Baltimore, Williams & Wilkins Company 1950.
- Keith, A.: *Human Embryology and Morphology*. London, Edward Arnold, 1913.
- Lampe, E. W.: *Surgical Anatomy of the Abdominal Wall*. S. Clin. North America 32:545, 1932.
- Lee, C. M., Jr., Collins, W. T., and Largent, T. L.: A Reappraisal of Absorbable Glove Powder. *Surg. Gynec. & Obst.* 95 725, 1952.
- Levens, G. and Kaufman, S. A.: Roentgenologic Findings in Acute Diseases in the Abdomen. *M. Clin. North America* 41:1303 1957.
- Loda, F. L.: Historical Aspects of Penetrating Wounds of the Abdomen. *Internat. Abstr. Surg.* 67:321 1918.
- Mackenzie, W. C., and Small, J.: Primary Idiopathic Segmental Infarction of the Greater Omentum. *Canad. M. A. J.* 33:111 1916.
- Maignot, R.: *Abdominal Operations*, 3rd ed. New York, Appleton-Century-Crofts, 1935.
- Morse, S. W.: The Physiological Basis for Diagnostic Signs of an Acute Abdomen. *S. Clin. North America* 34 771 1938.
- Murray, S. D., and Burger, R. E.: Rupture of the Inferior Epigastric Vessels. *Ann. Surg.* 139:90 1931.
- Ochsner, A., and DeBakey, M.: Subphrenic Abscess. *Internat. Abstr. Surg.* 66:140, 1938.
- Pack, G. T., and Ehrlich, H. E.: Neoplasms of the Anterior Abdominal Wall with Special Consideration of Desmoid Tumors. *Internat. Abstr. Surg.* 79 177 1911.
- Patten, B. M.: *Human Embryology*. 2nd ed. New York, Blakiston Company 1933.
- Paul, M.: The Surgery of the Congenital Anomalies of the Midline Ventral Abdominal Wall. *Ann. Roy. Coll. Surgeons England* 13:313, 1933.
- Pearman, R. O., and Mayo, C. W.: Desmoid Tumors. A Clinical and Pathologic Study. *Ann. Surg.* 113 111 1912.
- Poirier, P.: *Traité d'Anatomie Humaine*. Ed. Charpy, A. and Nicolas, A. Paris, Masson et Cie 3me Ed., 1912.
- Pulaski, E. J., Noyes, H. E., and Brame, R. A.: The Influence of Antibiotics on Experimental Endogenous Peritonitis. *Surg. Gynec. & Obst.* 99 341 1954.
- Robt, C. G.: The Diagnosis of Abdominal Trauma in Warfare. *Surg. Gynec. & Obst.* 85 147 1947.
- Rouviere, H.: *Anatomy of the Human Lymphatic System* (Translated by Tobias). Ann Arbor Mich., Edwards Brothers, 1938.
- Schäfer, C. S.: *Rupture of the Rectus Abdominis Muscle and Deep Epigastric Arteries*. *Am. J. Surg.* 66 157 1933.
- Scott, J. E. S.: *Haemangiomas in Skeletal Muscle*. *Brit. J. Surg.* 41:496, 1937.
- Silverman, F. N., and Huang, N.: Congenital Absence of the Abdominal Muscles. *Am. J. Dis. Child.* 80 91 1950.
- Soutar, S. F., Douglas, D. M., and Dennison, W. M.: Patent Vitello-Intestinal Duct. The Risk of Obstruction Due to Prolapse. *Brit. J. Surg.* 45:617 1958.
- Stewart, D. E., Hay, L. J., and Varco, R. L.: Malignant Melanomas. 92 Cases Treated at the University of Minnesota Hospitals since January 1 1932. *Internat. Abstr. Surg.* 97 209, 1953.
- Thomas, P. A.: Abdominal Trauma. Injuries to the Hollow Viscera. *Arch. Surg.* 75 742, 1937.
- Trimingham, H. L., and McDonald, J. R.: Congenital Anomalies in the Region of the Umbilicus. *Surg. Gynec. & Obst.* 80 152, 1945.
- Welch, C. E., and Richardson, G. S.: Early Operative Treatment of Generalized Peritonitis Due to Appendicitis. In *Current Surgical Management*, edited by J. H. Mulholland, E. H. Ellison, and S. R. Friessen. Philadelphia, W. B. Saunders Company 1957.
- Wright, G. P.: *An Introduction to Pathology*. 2nd ed. London, Longmans, Green & Co., 1936.

HERNIA

By CHESTER B. McVAY M.D.

CHESTER BEDWELL McVAY left Yankton, South Dakota, his birthplace to receive his medical education, do graduate degree work in anatomy at Northwestern University and receive his surgical training at the University of Michigan. After military service in World War II he returned to Yankton to practice and is Clinical Professor of Surgery and Associate Professor of Anatomy at the University of South Dakota. His union of anatomy and surgery has made him an authoritative and stimulating investigator of the surgical problems of hernia.

Historically the development of hernia surgery is one of the most interesting chapters in the field of medicine since hernias have always been the most common visible affliction of man amenable to surgical treatment. Its common occurrence and the enigma of its etiologic background and the cure through the ages have made the hernia story an absorbing narrative which has few peers even in the field of fiction. The story in the past seventy years is a monument to the surgeons' ingenuity but the multiplicity of modern operations for inguinal and femoral hernias is a record of basic misunderstanding of the anatomy involved.

Definition. The classical definition of a hernia is "the protrusion of a viscus from its normal cavity through a congenital or an acquired aperture." While this definition suffices in a general way it requires some elucidation. For example, a patient may have a hernia consisting entirely of preperitoneal fat, without a peritoneal sac and without the protrusion of a viscus. This is quite common in epigastric and femoral hernias in the early stages. Occasionally in indirect inguinal hernias, the entire presenting mass is a pedunculated process of preperitoneal fat. These fatty protrusions are attached to the

parietal peritoneum and if the aperture through which the fat protrudes is not repaired at this stage, more and more fat gradually extrudes and eventually pulls a diverticulum of peritoneum with it. Into this peritoneal pouch, then, a viscus may herniate. This is the mechanism of the development of epigastric hernias and is referred to as the lipoma theory of Cloquet.

If one adheres to the classic definition, these fatty protrusions do not represent hernias nevertheless they produce a palpable mass which is usually tender and produces symptoms which bring the patient to the doctor. These fatty protrusions are usually irreducible and in the inguinal canal may not be differentiated from a bowel-containing process of peritoneum. Another aberration of this definition is the reducible hernia and the majority of hernias are reducible in their early stages of development. A patient who has had a loop of bowel protruding through an aperture in the abdominal wall, and then has this loop of bowel return to the abdominal cavity, certainly has a hernia from a practical standpoint, although the viscus no longer protrudes.

Many patients live their entire lives with a congenital processus vaginalis in the in-

guinal canal without bowel or omentum ever entering this preformed sac. Therefore from their standpoint or that of an examining physician, they have never had a hernia. A peritoneal sac is generally considered to be a component of a hernia but this is not necessarily so. Traumatic hernias of the diaphragm rarely have a peritoneal sac unless they begin as small ruptures of the musculoaponeurotic structure which leave the peritoneum and pleura intact. A wound dehiscence is certainly a protrusion of abdominal viscera without a peritoneal covering.

Thus it is apparent that the definition of a hernia is not such a simple matter. Further, more the mass or size of the hernia, whether it is contained in a peritoneal sac and whether it is reducible or irreducible are not the important considerations. The important point is the defect in musculoaponeurotic and fascial continuity or more simply the hole through the parietal abdominal wall (respiratory or pelvic diaphragms). It is the size of this defect which is the crux of the repair problem. The fixation and rigidity of the hernial ring are frequently important factors in reducibility and the incidence of strangulation. The size of the hernial sac is of no particular consequence from the standpoint of repair except in the very large variety where the replacement of viscera long outside the abdominal cavity may present a spatial problem.

Therefore, perhaps a better definition of a hernia would be one directed at the defect to wit: *An abdominal hernia is a defect in the normal musculoaponeurotic and fascial continuity of the abdominal wall (respiratory or pelvic diaphragms) either congenital or acquired which permits the egress of any structures other than those which normally pass through the parietes.* Thus, intermittent protrusion, peritoneal sac or not, viscous or fat, would not confuse the definition. If one could diagnose a peritoneal sac with relative certainty as in cryptorchidism or intermittent hydrocele, a diagnosis of a hernia could then be made even though there had never been an intra-abdominal viscous in the hernial sac. Hernias of the brain, lung, muscle and joint synovia occur but to include them in a consideration of abdominal hernias would require a much broader definition and would needlessly complicate the picture.

Classification. There are many descriptive adjectives used in any discussion of hernias. *Topographic* refers to the regional location of the hernia, e.g., inguinal, femoral, um-

bilical or epigastric. *Congenital or acquired* hernias would seem to be clear-cut terms and as the words imply divide all hernias into those present at birth and those which come on in later life but some hernias are not categorized that easily. While there is not much doubt about absent segments of the respiratory diaphragm and omphalocele being congenital hernias, certain hernias considered to be acquired have a congenital predisposition to the development of the hernia. For example the indirect inguinal hernia that develops in adulthood has had the peritoneal sac since the descent of the processus vaginalis which preceded the descent of the testis. *Reducible or irreducible (incarcerated)* refers to whether or not the contents of the hernia can be pushed back into the abdomen. Incisional hernias frequently have both reducible and irreducible components. *Strangulated* hernia is an irreducible hernia with compression obstruction of the blood supply to the incarcerated loop of bowel. If this is not relieved surgically in a few hours, the strangulated hernia becomes *gangrenous*. An *incisional* hernia occurs through a surgical incision or scar is man made and in this instance there is no question that the hernia is acquired. *Recurrent* hernia is one which recurs following an operation for a similar type of hernia. In the inguinal region, this becomes a moot point. After repair of an indirect inguinal hernia, if the patient develops a direct inguinal hernia or a femoral hernia, does this represent a recurrence or is it a separate subsequently acquired hernia? Was this second hernia present all the time and simply missed at the original operation, or did the surgeon damage the posterior inguinal wall at the original operation and thereby lay the groundwork for the development of the second hernia? The term *double* usually refers to bilateral inguinal or femoral hernias but is also used to describe a direct-indirect inguinal hernia on a single side. A direct indirect inguinal hernia, with the double sac straddling the inferior epigastric blood vessels, is also known as a *pantaloon* hernia. Very occasionally one encounters a direct inguinal, an indirect inguinal and a femoral hernia on the same side. In this event it is known as a *triple* hernia. An *external* hernia is one that protrudes to the outside where it is visible and palpable. An *internal* hernia is one that remains within the confines of the body cavities. Diaphragmatic hernia is an example of an internal hernia. Occasionally one sees a hernia through the foramen

of Winslow into a deep peritoneal recess or pouch, or through a traumatic rent in the omentum or mesentery. However hernias of this type are extremely rare and most so-called internal hernias within the abdominal cavity represent congenital malrotations of the intestinal tract. A *sliding* hernia is one in which a viscus forms part of the wall of the hernial sac. *Hernia adiposa* is one in which the mass consists entirely of preperitoneal fat and is commonly seen in the epigastrium. *Littre's* hernia (Lavater's or Richter's hernia) is a type of incarcerated or strangulated hernia in which only a portion of the circumference of the bowel is caught in the hernial ring. *Spiegel's* hernia occurs in the linea semilunaris at or below the linea semicircularis but above the point at which the inferior epigastric vessels cross the lateral border of the rectus abdominis muscle. If it occurs below the inferior epigastric vessels then it must be termed a direct inguinal hernia. An *interstitial* hernia develops and enlarges between the musculo-aponeurotic laminae of the abdominal wall rather than in the subcutaneous fascia. *Complete* or *incomplete* usually refers to the indirect inguinal hernia and the extent of the congenital peritoneal sac. A complete indirect inguinal hernia is one in which no portion of the processus vaginalis has become obliterated so that the hernial contents extend into the tunica vaginalis of the testis (inset, Fig. 4 a). An incomplete indirect inguinal hernia may also extend into the scrotum, but the sac does not communicate with the tunica vaginalis of the testis because the lower portion of the processus vaginalis has become obliterated (inset, Fig. 2 a).

Etiology The etiologic basis of a given hernia may be an obvious single defect, but more commonly the cause is a combination of predisposing factors. The congenital hernias of infancy may be ascribed to a single etiologic factor which is either a congenital defect, such as omphalocele congenitalis, or failure of segmental development, as in the absence of a portion of the abdominal wall or respiratory diaphragm. An additional type of truly congenital hernia is the indirect inguinal hernia which appears at birth or soon thereafter. This distinction in timing in the development of the most common of all hernias is made to eliminate the contributing factors such as increased intra-abdominal pressure and muscle relaxation, which are etiologic in the development of the indirect inguinal hernia in

adulthood. Rarely trauma to the abdominal wall is the direct cause of a hernia, but, aside from penetrating wounds, the effect of trauma is usually transmitted to the weakest point and may be at some distance from the point of the injury. A blunt force to the central portion of the abdominal wall may result in the occurrence of a diaphragmatic hernia, a direct inguinal hernia, an indirect inguinal hernia or a femoral hernia. In the latter two examples, however congenital predisposition is also an important factor. For example unless there is a preformed sac in the inguinal canal no amount of trauma will produce an indirect inguinal hernia. In the case of the femoral hernia, the point is debatable. In an incisional hernia, the cause is the previous operative incision, although here again there are contributing factors such as hemorrhage, infection or increased intra-abdominal pressure in the immediate postoperative period.

Heredity or the *familial* tendency to hernia, is an obvious fact when one reviews the case records of many hernias. Persistence of the processus vaginalis in the inguinal canal, with an abdominal inguinal ring large enough to permit the development of a hernia, is certainly a familial tendency. Likewise the generalized weakness of connective tissue as seen in the asthenic individual with visceroptosis and varicose veins is hereditary and these individuals more commonly develop hernias than does the general population. Any statistical evaluation of the hereditary influence on the incidence of hernia (such as the figure 25 per cent) must, however be plotted against the overall incidence of hernia in the general population (variously estimated at between 1.8 and 1.15). Racial differences are probably more related to habits of nutrition and physical development than to any racial characteristics.

Age as an etiologic factor has two considerations. First, the incidence of all types of hernias as related to age and, second, age as related to a specific type of hernia. For example direct inguinal hernia is a disease of advancing years and is rarely seen in children. Indirect inguinal hernia is more common in persons in the younger years having its greatest incidence in the first year of life and a gradually decreasing incidence after that until the mid teens when there is a sharp rise again which lasts until the mid twenties. This is undoubtedly due to the period in which the most vigorous physical exercise occurs. The incidence of indirect

inguinal hernia then stays rather stationary until the sixth decade when it again falls off.

Sex as a determining factor in the development of a hernia is concerned with the type of hernia and developmental factors peculiar to the sex. The indirect inguinal hernia is nine times more common in the male than in the female because of the embryologic descent of the testes. Femoral hernia is three times more common in the female than in the male, presumably because of the difference in the inclination of the pelvis and the common occurrence of increased intra-abdominal pressure due to pregnancy. Adult umbilical hernia is two to three times more common in the female because of the element of pregnancy. Direct inguinal hernia is a rarity in the female for reasons unknown, but, from cadaver examination and examination of the posterior inguinal wall during the repair of indirect inguinal and femoral hernias in the female, it would appear that the posterior inguinal wall is a much heavier aponeurotic layer in the female than in the male.

Obesity in the young adult probably has very little effect upon the incidence of the various hernias, but in the older age groups it very definitely increases the incidence of epigastric and umbilical hernias, especially in the pregnant female. Obesity operates in two ways to increase the incidence of these two hernias: first, by increasing intra-abdominal pressure and, second, by forcing fat into tiny apertures, such as the perforating blood vessel foramina in the linea alba above the umbilicus (epigastric hernia) or through a persistent defect at the umbilicus (umbilical hernia). In the aged obese patient, one can observe fatty infiltration of the aponeurotic laminae with multiple herniae adiposae in the epigastrium. The incidence of incisional hernia is much greater in the obese patient.

Musculoaponeurotic deficiencies of the abdominal wall are an anatomic fact with considerable variation in the strength of the flat muscles of the abdominal wall. This undoubtedly is a primary factor in the development of the direct inguinal hernia. A congenitally narrow insertion of the transversus abdominis aponeurosis into Cooper's ligament may be the principal etiologic factor in the development of a femoral hernia.

Increased intra-abdominal pressure is the major etiologic factor in the development of all acquired hernias and is the precipitating cause in the development of the indirect inguinal hernia, in spite of the existence of

a congenital sac. Obesity has already been mentioned as a cause of increased intra-abdominal pressure. Other obvious causes are intestinal obstruction, urethral obstruction, cough, straining, trauma, heavy lifting and pregnancy. Gradually increasing and steady pressure is not so likely to produce a hernia as are repeated and sudden surges of increased pressure as in coughing. Many a patient can time the onset of his hernia to a sudden cough or to an extreme muscular effort especially when associated with the element of surprise. The shutter action of the abdominal inguinal ring and of the inguinal canal is a fact which largely explains why a physically active man does not develop an indirect inguinal hernia during his active years but suddenly develops the hernia in old age. The congenital sac was always there, but the abdominal inguinal ring was effectively closed by muscular effort until he reached the age of poor muscle tone and developed a chronic cough or suddenly engaged in a severe muscular effort. Occasionally one sees the first evidence of a small umbilical defect with the development of ascites in which event the fluid containing umbilical bulge becomes a barometer of the degree of ascites. Intra-abdominal neoplasms rarely cause a hernia because they develop so slowly that there is no significant increase in intra-abdominal pressure. This is due to the gradual stretching of the abdominal musculature.

Incidence. The relative frequency of the various common hernias has been tabulated many times but, rather than borrow from existing tables, it was decided to make a survey of the incidence in recent years. The total number of hernioplasties (3395) listed in Table I is too small to draw any definite conclusions. However it is interesting to note that the percentages have remained roughly the same in the past fifty years, with the exception that incisional hernias are increasing in numbers. This is a natural sequence to the increasing number of operations performed each year.

The first group of 1065 consecutive hernioplasties was performed on our service at Sacred Heart Hospital in Yankton South Dakota, between the years 1947 and 1954. This group represents the percentages in a general hospital in a rural area.

The second group of 1330 consecutive hernioplasties was performed in Passavant Memorial Hospital, Chicago Illinois in recent years. This group represents the percentages in a private city hospital.

Table 1 The Relative Incidence of the Common Hernias in Consecutive Hernioplasties

SOURCE AND NUMBER OF CASES	INDIRECT INGUINAL	DIRECT INGUINAL	FEMORAL	UMBILICAL	EPIGASTRIC	DIAPHRAGMATIC	DIAPHRAGMATIC
Sacred Heart Hospital Yonkers, N. Y. 1065 Hernias	658 61.8%	89 8.3%	61 5.7%	112 10.5%	18 1.7%	123 11.5%	4 0.5%
Parnassus Mem. Hospital Chicago, Illinois 1350 Hernias	816 61.3%	370 27.8%	23 1.7%	33 2.5%	8 0.6%	76 5.7%	4 0.3%
Johns Hopkins Hospital Baltimore, Maryland 1000 Hernias	573 57.3%	53 5.3%	29 2.9%	175 17.5%	27 2.7%	111 11.1%	32 3.2%
Total Hernioplasties 3395	2047 60.3%	512 15.1%	113 3.3%	320 9.4%	53 1.6%	310 9.1%	40 1.2%

The third group of 1000 consecutive hernioplasties was performed in Johns Hopkins Hospital, Baltimore, Maryland, between the years 1949 and 1951. This group represents the percentages from a city hospital with a large charity service.

Symptoms and Diagnosis. The subjective symptoms caused by a hernia are extremely variable and depend more upon the pressure exerted on the contents than upon the size of the hernia. A very large hernia containing several feet of intestine and a large wad of omentum may cause the patient only the vaguest of symptoms, whereas a small hernia tightly constricting a knuckle of bowel or omentum can cause severe local pain, referred pain and nausea. When a hernia becomes strangulated, then the symptoms are severe, progressive and, unless corrected promptly, lead to serious consequences and even death.

An indirect inguinal hernia containing incarcerated omentum may cause only slight discomfort locally but by traction on the omentum can cause a more distressing epigastric pain which may be mistaken for that due to a duodenal ulcer or gallbladder disease. The small epigastric or umbilical hernia can have all of the symptoms referred to the epigastrium by traction on the omentum.

A small and unrecognized femoral hernia may contain an incarcerated knuckle of urinary bladder and be attended with all the symptoms referable to the urinary system, i.e., frequency, urgency, terminal dysuria and even hematuria. An esophageal hiatus hernia may cause symptoms related to the heart (palpitation and substernal pressure) by pressure from the dilated stomach as it lies in the posterior mediastinum. A traumatic diaphragmatic hernia may have the

chief symptom of shortness of breath due to compression of the lung, pain in the base of the neck due to diaphragmatic irritation, or, if the parietal pleura is irritated, a typical pleuritic pain aggravated by deep breathing.

Whenever there is compression of the bowel lumen, there are the added and usually progressive symptoms of bowel obstruction. Even partial obstruction of the lumen, as in a Littre's hernia, can cause symptoms of bowel obstruction. When the blood supply to a viscus is compressed, one then sees the added signs and symptoms of a strangulating obstruction.

If the hernia is visible or palpable and the mass is painful, there is little doubt on the part of either the patient or the physician as to the diagnosis. Small epigastric hernias, especially in the obese, are difficult to detect. Hernias of the respiratory diaphragm are, of course, diagnosed with certainty only by an x-ray examination usually augmented by a contrast medium. Internal hernias are usually diagnosed only during an operation for intestinal obstruction.

Most hernias have a somatic component of local pain plus a referred element of visceral pain. It is in those cases, in which the patient's chief complaint is of visceral origin, that the diagnosis may not be entirely clear. Pain at the hernial site is directly related to the degree of irritation of the parietal peritoneum and adjacent somatically innervated structures. Pressure and inflammation are the precipitating factors. The pressure upon a contained viscus or its subsequent vascular injury is referred to the ganglia of the afferent visceral pain fibers, the superior mesenteric or celiac plexus, and is therefore felt by the patient as pain in the epigastrium. Some patients who have had a troublesome hernia for a long time learn to learn

to localize visceral pain and, hence have no referred symptoms.

Certainly the most common complaint of a patient with a hernia is a painful lump and it is the pain which usually brings the patient to the doctor. However concern over the presence of a tumor even when painless will usually cause the patient to seek medical advice. The pain is intermittent when the hernia is reducible. It is absent when the hernia is reduced and usually mild when the hernia is "out," if it is of long standing. Especially in hernias with small rings and in hernias that have just made their appearance, the pain is quite sharp when the hernia comes down and again at the moment when it reduces.

The objective characteristics of a hernia are the presence of an abnormal swelling which may be so soft as to feel fluctuant, firm or hard, depending upon the contents of the sac and the pressure exerted. Omentum has a rather characteristic doughy feel, but so does a loop of sigmoid colon containing feces. A loop of gas filled intestine may be very soft and compressible or very tense depending upon the degree of incarceration. If a loop of intestine is strangulated, it is very tense and exquisitely tender. Except when incarcerated, the mass of the hernia can be made to disappear with gentle pressure, especially with the patient in the supine position. The patient is frequently more adept at reducing the hernia than is the examining physician because of long practice with his particular hernia and the fact that his muscles are more relaxed when he is not afraid that the physician will hurt him.

Another classic characteristic of a hernia is the reappearance of the tumor with straining or coughing after it has been previously reduced. Associated with the reappearance of the hernia are the sensations imparted to the physician's examining finger. This sensation has been variously described but can most simply be expressed as a push or impulse. An incarcerated hernia or a strangulated hernia is fixed, will not reduce and, of course, has no impulse on palpation. Transillumination with a strong light in a darkened room is a useful aid in differentiating a hydrocele of the cord from an incarcerated indirect inguinal hernia. The hydrocele will transilluminate, whereas the hernia containing bowel or omentum will not because of its greater density.

Percussion of a hernial mass will serve to differentiate a gas-containing viscus from a

solid wad of omentum. Auscultation over the hernial mass will occasionally serve to confirm the impression of an incarcerated loop of intestine. Auscultation over the abdomen is more important in determining the degree or status of intestinal obstruction when an incarcerated hernia is present and there are other signs of intestinal obstruction. X-ray examination of the intestinal tract with a contrast medium is rarely necessary for the diagnosis of a hernia of the abdominal wall. Occasionally however the barium enema examination of the colon will furnish the only evidence of an incarcerated knuckle of sigmoid colon (Littre's hernia) in an indirect inguinal hernial sac.

Treatment. The treatment of a hernia is surgical correction of the defect in normal musculoaponeurotic structure. The operation should have as its principal objective the restoration of the hernial area to as near the normal status as is possible. Except in patients with large incisional hernias and in those with congenital absence of portions of the abdominal wall or respiratory diaphragm, normal anatomy can usually be restored.

Methods other than the surgical correction of the anatomic defect are occasionally indicated but are of secondary importance. The injection treatment of hernias was a popular method in Europe for years and gained its greatest popularity in this country in the 1930's but experience has shown that it rarely cures a hernia and it has been abandoned. In the first few years of its use, it apparently cured small indirect inguinal hernias but, eventually most of these hernias reappeared. The scarring incident to the injected sclerosing solution makes the surgical repair extremely difficult.

Some patients refuse operation even though the need is urgent in other instances there is a strong contraindication to subjecting the patient to the risk of an operation because of associated serious systemic disease. Such patients whose hernia is amenable to compression reduction should be fitted with an appropriate truss. However the decision as to the type of truss and the suitability of the hernia to truss treatment requires experience. An improperly fitting truss, or a truss applied to the wrong type of hernia, not only can be painful but can increase the likelihood of damage to the contents of the hernial sac. The small indirect inguinal hernia is the best type of hernia for the application of a truss. However it is also one of the easiest to repair surgically.

and, if local anesthetic is used, there are very few contraindications to the operative correction of this hernia.

The yarn truss which has been used for years in the management of the indirect inguinal hernia in the infant, still has a place in premature infants or the newborn in whom an anesthetic would carry an excessive risk. However in the past few years most surgeons now correct these hernias in the infant irrespective of age. Present-day anesthesia and knowledge of nutrition, electrolyte and fluid requirements for infants make the operation no more hazardous than for older children. In fact when properly managed, even the newborn withstands the operation well. Operation not only corrects the defect promptly but these children progress better in their development than those with recurrent bouts of trouble with a hernia. It also removes the hazard of strangulation which a yarn truss does not. In fact, one occasionally sees a vomiting infant with an incarcerated hernia and yet the mother thought that she had the yarn truss properly applied. At present we are still willing to procrastinate in the case of a small umbilical hernia up to one year in the hope that the ring will close with compression treatment. We now advise operation upon the infant with an indirect inguinal hernia as soon as the diagnosis is made.

In planning a hernia operation upon a given patient the surgeon should carefully consider the patient as a whole. This is especially true in persons of the older age groups. The cardiovascular and renal systems should be carefully evaluated, on our service a preoperative x-ray examination of the chest is mandatory. Even though the x-ray film of the chest shows no abnormality this evidence may be of the most vital importance in evaluating postoperative pulmonary complications. In the older group of patients who suddenly develop a hernia, causes of increased intra-abdominal pressure should be carefully searched for in the preoperative work up. The patient with an obstructing carcinoma of the colon or urethral obstruction due to benign prostatic hypertrophy or carcinoma should not be subjected to a hernia operation unless the hernia is strangulated. If at all possible the obese patient should be reduced to near normal weight before undergoing an elective hernia operation. The operation is not only more difficult technically but the incidence of recurrence and postoperative complications is much higher in the obese patient than in one of normal weight.

HERNIAS OF THE INGUINOFEMORAL REGION (GROIN HERNIAS)

This rather general heading is used so that inguinal and femoral hernias may be considered together. It has been customary in the past to discuss the femoral hernia as a separate entity but the point should be emphasized that a femoral hernia is in reality a type of inguinal hernia. Although the etiologic factors of the indirect inguinal hernia, the direct inguinal hernia and the femoral hernia are different, the effect of these hernias upon the posterior inguinal wall is so interrelated that they must be considered together if one is to understand, first, the altered anatomic structure and, second, the rational repair.

Although the sac of the femoral hernia lies in the fossa ovalis of the femoral region, the defect which allows the hernia to develop is just as truly inguinal in location as is the defect which permits the development of a direct inguinal hernia, or the enlarged abdominal inguinal ring of an indirect inguinal hernia even though the sac of the latter may extend into the scrotum. Therefore the femoral hernia will be presented as simply a variety of inguinal hernia.

INDIRECT INGUINAL HERNIA

Definition An indirect inguinal hernia is one which passes through the abdominal inguinal ring and follows the course of the spermatic cord in the inguinal canal and then emerges through the subcutaneous inguinal ring. A very small sac may be confined to the inguinal canal, whereas the complete congenital peritoneal sac extends into the scrotum to communicate with the tunica vaginalis of the testis (Fig. 4 a and inset).

Etiology The primary etiologic factor of the indirect inguinal hernia is the presence of a congenital diverticulum of peritoneum which passes through the abdominal inguinal ring and into the inguinal canal. This is due to failure of the processus vaginalis to obliterate after the descent of the testis in the male and after fixation of the ovary in the female. It may be a short sac or extend into the scrotum and communicate with the tunica vaginalis of the testis (insets Figs. 2 a and 6 a). A necessary contributing etiologic factor is increased intra-abdominal pressure sufficient to force a viscus into the preformed peritoneal sac.

Incidence. This is not only the most common type of groin hernia but the most common of all hernias. 1) The fact that it represents more

all hernias and over 80 per cent of the groin hernias very adequately expresses the importance of the proper understanding of the pathologic anatomy of this hernia and its operative repair. It is ten times more common in the male than in the female.

Anatomy The normal anatomic structure of the inguinofemoral region is presented in Figure 1 a and b. The distortion of the normal anatomic relationship by the presence of an indirect inguinal hernia can best be presented by dividing the indirect inguinal

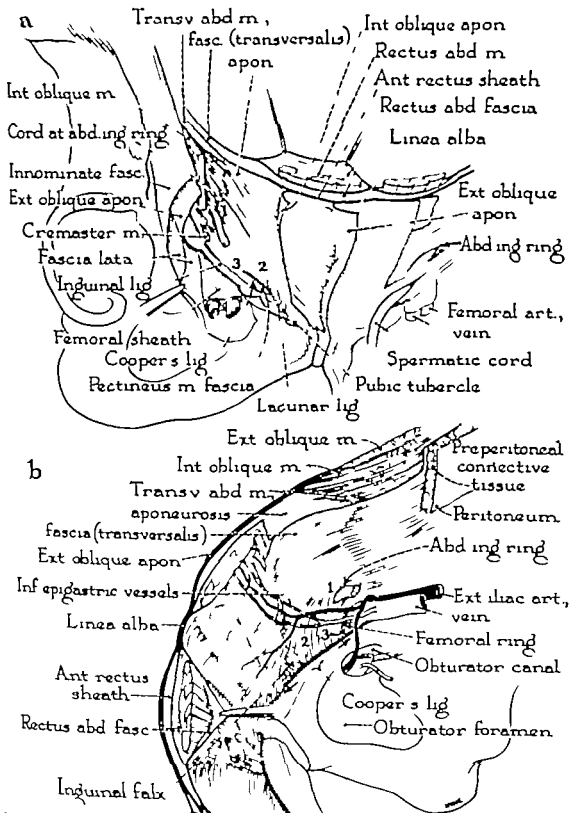


Figure 1 The normal anatomic structure of the inguinofemoral region. a, Anterior view b, Posterior view. The numbers 1, 2 and 3 represent the sites of origin respectively of the indirect inguinal, the direct inguinal and the femoral hernia.

hernias into three categories, depending upon the size of the dilated abdominal inguinal ring. The length of the sac or the quantity of the contents of the hernia is of no particular importance. The crux of the repair problem is the size of the abdominal inguinal ring and on this basis the pathologic anatomy of this type of hernia will be discussed under the headings of small medium and large indirect inguinal hernia.

Small indirect inguinal hernia (Fig. 2)
All indirect inguinal hernias are in this category at their outset. Irrespective of the length of the congenital peritoneal sac there is only very slight dilatation of the abdominal inguinal ring. Aside from the presence of the congenital peritoneal sac the only alteration of the normal anatomic state is the minimal stretching of the abdominal ring (Fig. 2 b and c). The posterior inguinal wall (transversus abdominis muscle aponeurosis and fascia) is intact, the femoral ring is small and even the continuity of the transversalis fascia into the anterior femoral sheath medial to the slightly dilated abdominal inguinal ring is normal. Therefore it should be apparent that the repair of this most common of all hernias should be a very simple problem.

Medium indirect inguinal hernia (Fig. 3)
The selection of this category is arbitrary to illustrate a point. As pointed out above the small indirect inguinal hernia has only the slightest alteration from the normal anatomic relationships. The large indirect inguinal hernia may cause complete destruction of the posterior inguinal wall and its repair involves the same problems as does the direct inguinal hernia. In between these two extremes there is great variation in the dilatation of the abdominal inguinal ring. As the ring is enlarged slowly by the presence of the hernia, it expands medially for the most part. Eventually a point is reached which determines whether the hernia can be repaired by simple closure of the dilated abdominal inguinal ring or whether the posterior wall must be reconstructed. From our experience it has been determined that this point is a line drawn cephalad from the femoral ring. If the dilated abdominal inguinal ring extends medially beyond this line the hernia must be repaired as for a large indirect inguinal hernia and, conversely, if it remains lateral to this line the hernia may be simply repaired by closing the dilated abdominal inguinal ring.

The term medium indirect inguinal hernia, then, refers to a hernia with an ab-

dominal inguinal ring which has dilated up to this line (Fig. 3 b and c). In this hernia, in addition to the congenital peritoneal sac, the pathologic anatomy consists of complete destruction of the continuity of the transversalis fascia into the anterior femoral sheath by the enlarged abdominal inguinal ring. However the posterior inguinal wall and its insertion into Cooper's ligament are undisturbed.

Two other variations of the normal anatomic relationship are common in both this size of indirect inguinal hernia and in the large variety protrusion of pedunculated processes of preperitoneal fat among the cord structures (Fig. 3 b) and the incorporation of a hollow viscus in the wall of the hernial sac, known as a sliding hernia.

Large indirect inguinal hernia (Fig. 4)
It should be understood that this hernia began as the small variety and in sequence was a medium indirect inguinal hernia before becoming a large indirect inguinal hernia. It therefore takes considerable time for the development of this type of indirect inguinal hernia. Any indirect inguinal hernia, with the abdominal inguinal ring dilated medially beyond the line drawn cephalad from the femoral ring, is termed a large indirect inguinal hernia. It should be realized that just as there is considerable variation between the size of a small and a medium indirect inguinal hernia, there is also a great variety of sizes of dilated abdominal rings between the medium indirect and the largest possible indirect inguinal hernia which are all designated large indirect inguinal hernias. In the very large variety the medial margin of the dilated abdominal inguinal ring extends to the lateral margin of the rectus abdominis muscle (Fig. 4 b and c). Anatomically this means that a posterior inguinal wall is non-existent.

In the slow transition of a small indirect inguinal hernia into the very large variety there are several features which are of importance. In the indirect inguinal hernia, the peritoneal sac is always on the anteromedial aspect of the spermatic cord as it lies in the inguinal canal, except in the recurrent indirect inguinal hernia, where it may bear any relationship to the cord structures. This constant position of the peritoneal sac goes back to the embryologic descent of the testis. Prior to the descent of the testis, the processus vaginalis of the peritoneum descends into the scrotum. The testis, which is a retroperitoneal structure slides into the scrotum behind and slightly ahead to the

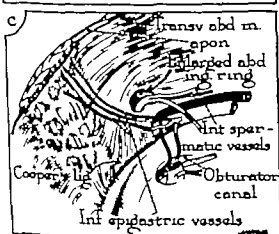
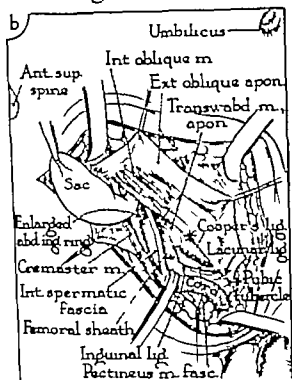
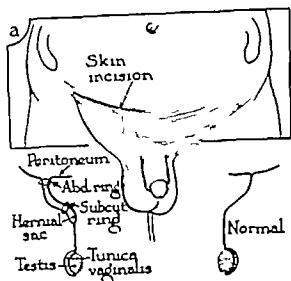


Fig. 2

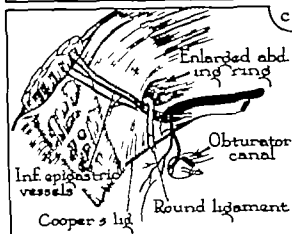
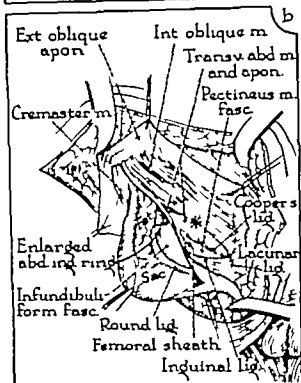
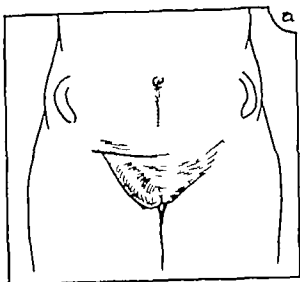


Fig. 3

Figure 2. a, Appearance of a small indirect inguinal hernia and skin incision used for all groin hernias. Insets. Left, an incomplete hernial sac, and, right, normal obliteration of processus vaginalis. b, Anterior view of the anatomy of a small indirect inguinal hernia. The line of separation of cremaster muscle from the internal oblique is denoted by —. c, Posterior view of the slightly dilated abdominal inguinal ring.

Figure 3. a, Appearance of a medium-sized indirect inguinal hernia in the female. b, Anterior view of the anatomy of a medium indirect inguinal hernia in the female. The line of separation of the cremaster muscle from the internal oblique is denoted by —. c, Posterior view of the moderately dilated abdominal inguinal ring.

peritoneal sac therefore, when the processus vaginalis fails to obliterate the indirect inguinal hernial sac is already in position waiting to receive a viscus and become an indirect inguinal hernia.

As the abdominal inguinal ring enlarges in the transition from a small to a medium to a large indirect inguinal hernia, this enlargement takes place predominantly in a medial direction. The origin of the lower fibers of the internal oblique and transversus abdominis muscles from the fascia lata lateral to the abdominal inguinal ring prevents any significant enlargement in this direction. Inferiorly or caudally the inguinal ligament over the iliac vessels and, more medially, the superior ramus of the pubic bone prevent enlargement in this direction. Superiorly there is some extension, but the abdominal musculature prevents much enlargement in this direction. Therefore with rather rigid restrictions superiorly laterally and inferiorly it can be seen that the abdominal inguinal ring enlarges medially at the expense of the posterior inguinal wall (transversus abdominis aponeurosis). As the posterior inguinal wall is stretched and attenuated, it is pushed outward and becomes part of the internal spermatic fascial investment of the hernial sac. The lateral border of the rectus abdominis muscle and tendon of origin stop the medial enlargement of the abdominal inguinal ring. When a ring of this size is reached, there is never any significant increase in size no matter how long the hernia persists or how many of the abdominal viscera descend into the hernial sac. In the enormous scrotal hernias there is some stretching of the musculature along the superior margin of the ring, but, for all practical purposes the rectus muscle is the final delimiting factor in the size of the ring.

Diagnosis. The diagnosis of this type of hernia is dependent upon the demonstration of the course of the hernial sac through the inguinal canal. The hernia begins at the abdominal inguinal ring, traverses the inguinal canal and emerges into the upper scrotum or greater labia at the subcutaneous inguinal ring. The descent of the sac into the scrotum is variable in degree and is dependent upon the original size of the congenital sac and upon the duration of the hernia. A scrotal hernia is invariably an indirect inguinal hernia, although rarely a direct inguinal hernia may extend well down into the scrotum, statements to the contrary notwithstanding. Usually the diagnosis of this type of hernia is made with ease by following a few simple

diagnostic maneuvers. In a slender individual observation will be sufficient to make the diagnosis because the bulge of the hernia can be seen to begin at the abdominal inguinal ring, follow the course of the inguinal canal and emerge at the subcutaneous inguinal ring. If the external oblique aponeurosis is a strong layer with heavy intercrural fibers the only bulge visible may be at the subcutaneous inguinal ring. In the obese patient or when the hernia is incipient, one must use palpation to make the diagnosis.

The classic maneuver of invaginating the scrotal skin with the index finger and then advancing the end of the finger through the subcutaneous inguinal ring helps to make the diagnosis in several ways. With the hernia reduced and the end of the examining finger through the subcutaneous inguinal ring, a sudden cough by the patient will demonstrate the oblique course of the hernia down the length of the inguinal canal. Sometimes it is difficult to tell whether the hernia is direct or indirect, especially when the subcutaneous inguinal ring is too small to admit the end of the examining finger. When this is the case digital pressure over the abdominal inguinal ring will prevent the descent of the hernia if it is of the indirect variety, whereas the bulge will still be felt if it is a direct inguinal hernia.

The patient should be examined in both the standing and the recumbent position for adequate evaluation of the hernia. Even in a patient having an obvious indirect inguinal hernia, the strength of the posterior inguinal wall should be evaluated before the operation because the patient can voluntarily contract his muscles. This is done with the patient in the recumbent position with the end of the examining finger through the subcutaneous inguinal ring and the indirect hernia reduced. The posterior inguinal wall is then indented by pressure posteriorly with the end of the finger and the patient asked to strain forcibly as when having a bowel movement. If the posterior inguinal wall is strong and intact, a firm, hard layer will be felt. If the hernia is direct or if there is a combined direct and indirect inguinal hernia, contraction of the abdominal muscles as in straining gives no sensation of a firm layer against the end of the examining finger. In fact, with the patient straining, the fingertip may be moved in and out with ease and the superior edge of the superior pubic ramus plainly felt. This is the location of Cooper's ligament. This last combined maneuver is an important of the pre-

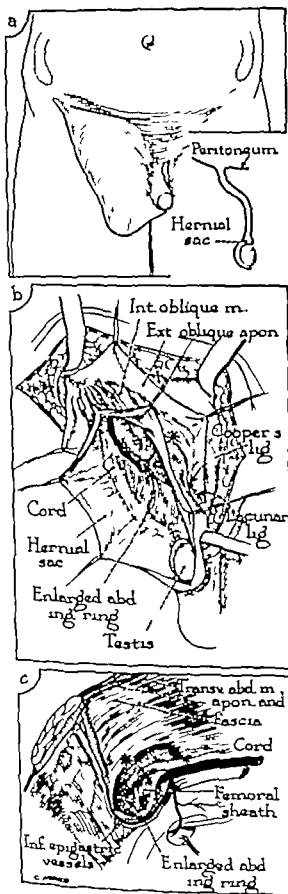
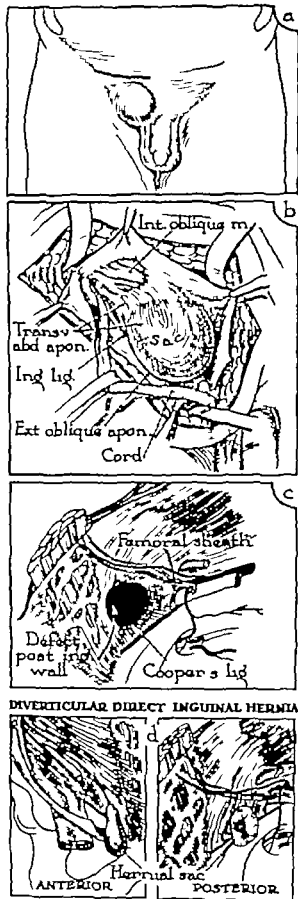


Fig. 4



DIVERTICULAR DIRECT INGUINAL HERNIA

Fig. 5

Figure 4. a, Appearance of a large indirect inguinal hernia. Inset: Complete, congenital hernial sac. b, Anterior view of the anatomy of a large indirect inguinal hernia. The function of weak and strong posterior inguinal wall is denoted by c. Posterior view of the greatly dilated abdominal inguinal ring. The edge of remaining strong posterior inguinal wall is denoted by d.

Figure 5. a, Appearance of a direct inguinal hernia. b, Anterior view of the anatomy of a direct inguinal hernia. c, Posterior view of the direct hernia defect in the posterior inguinal wall. d, Anterior and posterior views of the anatomy of the diverticular type of direct inguinal hernia.

operative examination especially for the beginner because at the operating table with the patient asleep it is sometimes difficult to evaluate the strength of the posterior inguinal wall.

As has been mentioned, a direct inguinal hernia may be confused with an indirect inguinal hernia or may coexist as a double or "pantaloon" hernia. A femoral hernia may curve cephalad over the inguinal ligament and superficially resemble an indirect inguinal hernia, but careful examination should serve to differentiate the two. Although the inguiofemoral group of lymph nodes lies below the inguinal ligament, these nodes do rise above or cephalad to the ligament when enlarged and in certain individuals may very closely resemble an incarcerated indirect inguinal hernia.

A large varicocele may resemble an indirect inguinal hernia, but it lacks the feel of a hernial sac, does not give the typical impulse and is irreducible. It may give a slight impulse with coughing due to increased venous pressure and can be decreased in size by compression, but it has a characteristic sensation to palpation often described as a "bag of worms."

An incompletely descended testis lying in the inguinal canal may feel like the present ing end of a hernial sac and is frequently seen in children. It is our belief that unless the testis has descended by the time the child reaches the age of six or seven years it should be placed in the scrotum surgically and that the use of Antultrin S is not warranted. In those few patients in whom it seems to effect a further descent of the testis it is likely that the effect is pure coincidence. Behind an incompletely descended testis one invariably finds a persistent processus vaginalis which, of course is the precursor of an indirect inguinal hernia.

Tumors of the testis and hydrocele of the tunica vaginalis of the testis should not cause confusion because the cord above the swelling is normal in size. Hydrocele of the cord, however particularly if it extends up through the subcutaneous inguinal ring, may simulate an incarcerated indirect inguinal hernia. Neither an incarcerated hernia nor a hydrocele gives an impulse, but the hydrocele will transilluminate and the incarcerated hernia will not. Occasionally one sees a hydrocele which disappears overnight or becomes soft and much smaller. This is direct evidence that a small communication exists through a persistent processus vaginalis and that a hernial sac exists. This type

of hydrocele should be treated surgically like an indirect inguinal hernia and the sac completely removed. In the presence of hydrocele of the cord, one should always look for a coexistent indirect inguinal hernia because whether there has ever been an actual hernia or not, a persistent processus vaginalis is commonly associated.

A hernia adiposa in the inguinal canal so closely resembles an incarcerated indirect inguinal hernia that the two cannot be differentiated. As a matter of fact, a hernia adiposa does constitute a true hernia even in the absence of a peritoneal sac. In any event, the protrusion of preperitoneal fat through the abdominal inguinal ring will have dilated the ring and, therefore, it needs surgical correction.

Treatment. In the small indirect inguinal hernia there is practically no alteration of the normal anatomic structure. The persistent processus vaginalis which is the hernial sac must, of course be removed and the adequate removal of this sac with high ligation has long been a standard procedure. After dissecting the peritoneal sac free from the cord structures well up through the abdominal inguinal ring, all that needs to be done to return the inguinal region to normal is to tighten the ring to normal size (Fig. 6 a and b). Any additional approximation or plication of the layers is not only unnecessary but may damage an otherwise normal posterior inguinal wall. After the abdominal inguinal ring is returned to normal size the spermatic cord is dropped back into its normal position and the external oblique aponeurosis is sutured together making a snug subcutaneous inguinal ring (Fig. 6 c).

After the peritoneal (hernial) sac is removed, the medial margin of the slightly dilated abdominal inguinal ring is delineated by sharp dissection. This is very simply accomplished by cutting the internal spermatic fascia at its point of origin from the transversalis fascia. This clearly demonstrates the margin of the medial half of the slightly dilated abdominal inguinal ring without disturbing the fascial and muscular attachments of the lateral half of the ring which need not be disturbed. It also demonstrates the fascial continuity of the transversalis fascia into the anterior layer of the femoral sheath. It is upon this anatomic fact that the rationale for the repair of this type of hernia is based. The dilated abdominal inguinal ring is reduced to normal size by suturing the transversalis fascia to the anterior layer of the femoral (Fig. 6 b).

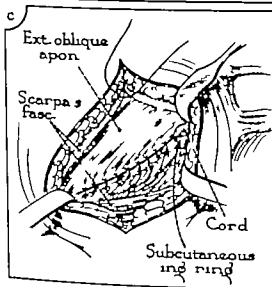
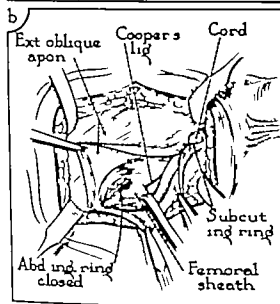
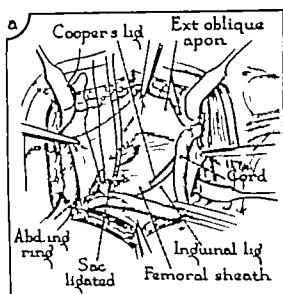


Fig. 6

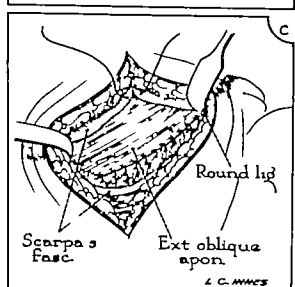
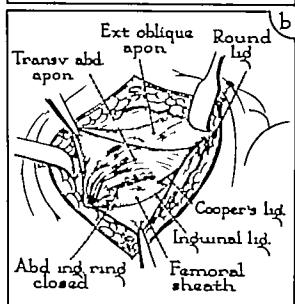
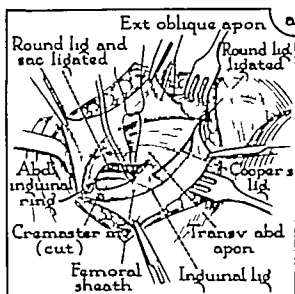


Fig. 7

Figure 6 Hernioplasty for small indirect inguinal hernia (viewed from right) a, b and c, Successive steps in the operation.

Figure 7 Hernioplasty for medium indirect inguinal hernia (viewed from right) in the female. a, b and c, Successive steps in the operation showing obliteration of the abdominal inguinal ring.

Frequently the external spermatic vessels do not pass through the abdominal inguinal ring but through a separate foramen more medially placed. If they cannot be pulled laterahward into the abdominal inguinal ring, they are cut between ligatures to avoid leaving an aperture through which fat might protrude.

The repair of a *medium indirect inguinal hernia* is essentially the same as that of the small indirect inguinal hernia except, of course, that the abdominal inguinal ring is larger and it takes more sutures to return the ring to normal size (Fig. 7 b). The principle of repair is identical in that the transversalis fascia is sutured to the anterior layer of the femoral sheath. When an indirect inguinal hernia is repaired in the female the round ligament is removed with the hernial sac which permits complete closure of the abdominal inguinal ring. This is the ideal hernia repair for the beginner to undertake because the continuity of layers is clear and one is not faced with the complicating feature of structures passing through the inguinal canal.

In addition to the presence of the congenital hernial sac a *large indirect inguinal hernia* because of the greatly enlarged abdominal inguinal ring, has destroyed the posterior wall of the inguinal canal. Therefore in accomplishing the repair of this type of hernia, one must not only remove the hernial sac and make a snug abdominal inguinal ring as in the two foregoing types of hernias, but one must also reconstruct a new posterior inguinal wall (Fig. 8).

Through the years innumerable devices have been used to obtain muscular, aponeurotic and fascial material to replace the posterior inguinal wall. Some of them have merit while others are based on false anatomical premises. Furthermore any hernioplasty which fastens the new posterior inguinal wall to the inguinal ligament is anatomically unsound. One should carefully study the normal anatomy of the inguinal region as presented in Figure 1 for it is upon a thorough understanding of the normal anatomy that the rational repair of groin hernias is based. In other words it should be the object of every hernia operation to return the region to the normal anatomic state.

When the posterior inguinal wall is destroyed in part or in toto by a large indirect inguinal hernia, aponeuroticofascial tissue must be borrowed somewhere to close the defect. The simplest device is to use the

aponeurosis and fused fasciae of the transversus abdominis immediately above and medial to the defect (Fig. 8). This aponeuroticofascial plate also contains the lowest aponeurotic fibers of the internal oblique muscle and, of course, where it lies over the rectus abdominis muscle it is known as the rectus sheath. This layer is the ideal material because it is primarily aponeurotic. The fasciae, of which the innermost layer is the transversalis fascia, serve to bind the aponeurotic fibers together into a firm and intact layer. Fascia or muscle does not meet the requirements for a satisfactory layer to close a hernial defect.

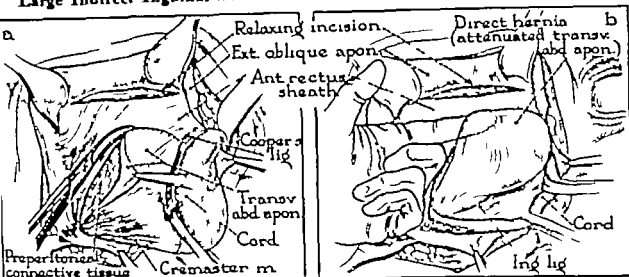
Another sound surgical principle is that layers should be approximated without tension. If one sutures the strong edge of the transversus abdominis aponeurosis to Cooper's ligament, there is considerable tension on the suture line. The same difficulty is encountered in the classic herniorrhaphies which use the inguinal ligament as the anchoring structure. To obviate tension, the "relaxing incision" (Fig. 8) should be used in every instance in which the posterior inguinal wall is reconstructed. This was first described by Halsted in 1911 and subsequently championed by Ridenhoff and Fultz. A modification of the relaxing incision is the turning downward of a triangular flap of the rectus sheath as described by Bloodgood and more recently by Farris. The "slide" of the rectus sheath made possible by the relaxing incision seems more physiologic than the flap method because the normal direction of musculoaponeurotic pull is maintained.

Preserved fascia, cutis grafts, osteoperiosteal grafts and flaps of fascia lata have all been used as patches for the posterior inguinal wall. While these methods have been used successfully by their proponents, we have never found it necessary to resort to transplanted material. However should it seem advisable to reinforce the posterior inguinal wall after its reconstruction by the method to be briefly described, it would seem preferable to use a wire mesh, as described by Koozts.

Before the new posterior inguinal wall can be transferred into position, all of the attenuated old posterior inguinal wall and hypertrophied cord fasciae must be excised (Fig. 8). The peritoneal hernial sac must be dissected out and the neck of the sac ligated as for any indirect inguinal hernia. For an accurate repair of this hernia, one must have an evenly cut margin of trans-

Large Indirect Inguinal Hernia

Direct Inguinal Hernia



RECONSTRUCTION OF THE POSTERIOR INGUINAL WALL

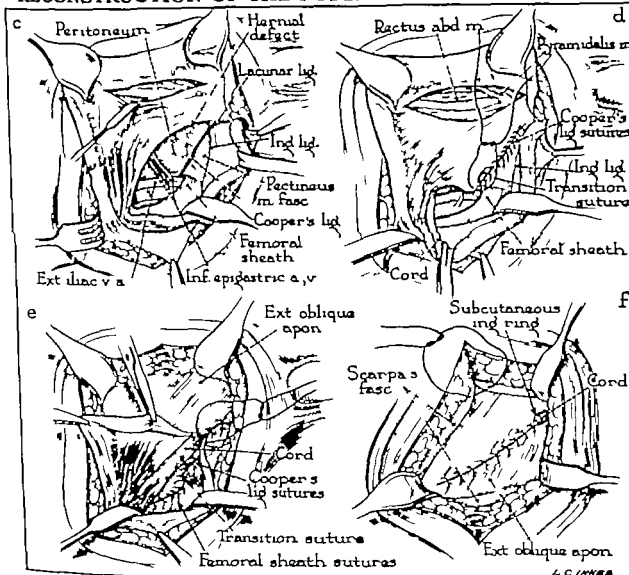


Figure 8 Hernioplasty for large indirect and direct inguinal hernias. a, Cutting out the attenuated portion of the posterior inguinal wall in a large indirect inguinal hernia. b, Attenuated posterior inguinal wall that is to be removed in a direct inguinal hernia. c, d, e and f Successive steps in the reconstruction of the posterior inguinal wall. Hernioplasty for both large indirect and direct inguinal hernias.

versus abdominis aponeurosis above, from abdominal inguinal ring to Cooper's ligament. Below the glistening margin of Cooper's ligament must be seen medially and the edge of the anterior femoral sheath laterally. When these margins have been carefully dissected and the peritoneal sac excised, one is then ready to repair the hernia (Fig. 8 c).

After making the relaxing incision (Fig. 8 a and b) the strong cut edge of the transversus abdominis aponeurosis is sutured to Cooper's ligament from the pubic tubercle to within a few millimeters of the external iliac vein (Fig. 8 d). This maneuver not only reconstructs the posterior inguinal wall, but it re-establishes a normally broad insertion into Cooper's ligament and thus obviates the possibility of the development of a femoral hernia. It should be noted that the relaxing incision (Fig. 8 d) is now a considerable defect in the rectus sheath, protected behind by the rectus and pyramidalis muscles and their fasciae.

The next suture is the "transition" suture which approximates the edge of the transversus abdominis aponeurosis to the medial wall of the femoral sheath and pectineus muscle fascia. This suture is necessary to close the angle and permit the line of closure to come up from the level of Cooper's ligament to the more superficial level of the anterior femoral sheath. This distance is represented by the diameter of the external iliac vein.

The remaining defect is closed by suturing the transversus abdominis aponeurosis or in case the layer is muscular at this point, the transversalis fascia, to the anterior layer of the femoral sheath. This re-establishes the normal continuity of transversalis fascia into the anterior femoral sheath. This line of sutures is continued laterally until a snug abdominal inguinal ring is made just as it is done for the repair of the small to medium sized indirect inguinal hernia. The spermatic cord is dropped in against the new posterior inguinal wall and the external oblique aponeurosis closed over it (Fig. 8 e and f) thus re-establishing the obliquity of the inguinal canal. The subcutaneous inguinal ring is snugly closed.

DIRECT INGUINAL HERNIA

Definition. A direct inguinal hernia is one that passes through the posterior inguinal wall medial to the inferior epigastric vessels in the area bounded by Hesselbach's triangle.

Etiology. The etiologic basis of the direct inguinal hernia is both congenital and acquired. The acquired factor in the development of this hernia is easy to understand because it is operative in the development of all hernias and is due to increased intra-abdominal pressure from whatever cause. Sudden or abrupt increase in the intra-abdominal pressure is more effective in producing a hernia than is a gradual increase in pressure.

The congenital predisposition to the development of a direct inguinal hernia is undoubtedly the most important etiologic factor. The robust common laborer is not prone to develop a direct inguinal hernia, nor is the asthmatic with paroxysms of violent coughing certain to develop this type of hernia. On the other hand, the asthenic individual with visceroptosis is not only the ideal candidate for the development of a direct inguinal hernia, but he is also more likely to have hemorrhoids and varicose veins than is a person of other body type. This suggests a congenital weakness of connective tissue throughout the body.

Direct inguinal hernia is a rarity in the female. This has been attributed to the flat, gynecoid pelvis of the female and a more even distribution of intra-abdominal pressures than in the male. While these anatomic facts may help to explain the low incidence of direct inguinal hernia in the female, it is also true that the posterior inguinal wall is almost always a heavy aponeuroticofascial plate in the female. Just why this is so, when as a rule the male has the better muscular development, cannot be explained, but it has been observed in the dissection of many cadavers and almost a thousand operations.

Irrespective of sex, body build, occupation or associated diseases that increase intra-abdominal pressure, the most important predisposing cause for the development of a direct inguinal hernia is a weak posterior inguinal wall. The posterior inguinal wall is the transversus abdominis aponeurosis with its investing fasciae and the strength of this wall is directly proportional to the number of aponeurotic fibers. The transversalis fascia is the innermost fascial layer and, by itself, has very little tensile strength. There is considerable variation in the strength of this layer even in those individuals who never develop a direct inguinal hernia.

There is also considerable variation in the internal oblique layer in the inguinal region which has been observed and recorded by a number of surgeons, but we now consider

this layer of secondary importance in the development of direct inguinal hernia.

Occasionally direct external trauma may be the etiologic basis of a direct inguinal hernia and, certainly injury to the posterior inguinal wall during the repair of an indirect inguinal hernia or a femoral hernia will cause a direct inguinal hernia. In this instance, it is usually a "diverticular" type of direct inguinal hernia (Fig. 5 d)

Incidence. This hernia represents about 20 per cent of the groin hernias and from 10 to 15 per cent of all parietal abdominal hernias. While it is readily seen that this is not as common as the indirect inguinal hernia, it is the direct inguinal hernia which has baffled surgeons in the past half century and it is the hernia that is most likely to recur. The conventional inguinal ligament herniorrhaphies have been notably unsuccessful in repairing the direct inguinal hernia, recorded recurrence rates varying from 8 to 50 per cent.

Anatomy. The normal anatomy of the inguino-femoral region is presented in Figure 1, in which the number 2 represents the site of direct inguinal hernia. Whatever combination of factors operate to cause a direct inguinal hernia, the immediate cause is, of course, a dissolution of the integrity of the posterior inguinal wall.

The most common type of direct inguinal hernia is not an actual hole through the posterior inguinal wall, but a gradual attenuation of an intact but congenitally weak transversus abdominis aponeurosis. It is weak because of a deficient number of aponeurotic fibers. As the bulge of the hernia becomes larger and larger the aponeurotic fibers are spread farther and farther apart. The fasciae, of which the innermost or transversalis fascia is the heaviest, stretch but maintain the continuity of this layer.

As the transversus abdominis layer gives way and bulges forward, the overlying internal oblique muscle is stretched and pushed superiorly. The spermatic cord, with its investing cremaster usually remains below the bulge of the hernia. This is due to the disruption of the delicate fascial connection between the lower edge of the internal oblique and the cremaster. A strong musculoaponeurotic internal oblique layer may slow down the development of a direct inguinal hernia, but it cannot prevent it if the underlying transversus abdominis layer gives way. The significance of the so-called inguinal triangle in the causation of a direct inguinal hernia is certainly questionable.

However this much is true—a deficient internal oblique layer in the inguinal region is frequently associated with a deficient transversus abdominis layer. The important point to understand is that the transversus abdominis aponeurosis with its investing fasciae is the key to the development of a direct inguinal hernia. If this layer is strong and intact a direct inguinal hernia will not develop even though the internal oblique and external oblique layers are very weak or deficient. On the other hand, if the transversus abdominis layer begins to attenuate and bulge the patient will develop a direct inguinal hernia in spite of a strong overlying internal oblique muscle.

The strength of the external oblique aponeurosis is of even less importance than the internal oblique muscle, for the developing hernia easily protrudes through this layer between the crura of the subcutaneous inguinal ring. This space between the aponeurotic crura of the external oblique aponeurosis is commonly referred to as a ring; actually it is a long triangle with the aponeurotic defect bridged by the innominate fascia. It is variable in strength, depending upon the number of intercrural fibers. When the hernia has passed through the external oblique layer it enlarges in size in the subcutaneous fascia in the vicinity of the subcutaneous inguinal ring.

Hesselbach's triangle bounded by the inguinal ligament, the inferior epigastric vessels and the lateral border of the rectus abdominis muscle, is a convenient descriptive area. Direct inguinal hernias must pass through this triangle. If a hernia occurs lateral to the inferior epigastric vessels it is an indirect inguinal hernia. If a hernia occurs below the inguinal ligament, it is a femoral hernia. If a hernia occurs above the inferior epigastric vessels in the linea semilunaris, it is a Spiegel's hernia. Occasionally the sac of a direct inguinal hernia will straddle the obliterated hypogastric artery to form a pantaloon type of sac. However this is a curiosity rather than of any particular significance and we place no emphasis upon whether the hernia has presented through the suprapubic fossa or the medial inguinal fossa.

The pathologic anatomy of the common type of direct inguinal hernia has been described. Rarely one sees a direct inguinal hernia with a small neck and these are described as a diverticular type of direct inguinal hernia (Fig. 5 d). This hernia proceeds through the layers of the abdominal wall in

versus abdominis aponeurosis above from abdominal inguinal ring to Cooper's ligament. Below the glistening margin of Cooper's ligament must be seen medially and the edge of the anterior femoral sheath laterally. When these margins have been carefully dissected and the peritoneal sac excised, one is then ready to repair the hernia (Fig. 8 c).

After making the relaxing incision (Fig. 8 a and b) the strong cut edge of the transversus abdominis aponeurosis is sutured to Cooper's ligament from the pubic tubercle to within a few millimeters of the external iliac vein (Fig. 8 d). This maneuver not only reconstructs the posterior inguinal wall, but it re-establishes a normally broad insertion into Cooper's ligament and thus obviates the possibility of the development of a femoral hernia. It should be noted that the relaxing incision (Fig. 8 d) is now a considerable defect in the rectus sheath, protected behind by the rectus and pyramidalis muscles and their fasciae.

The next suture is the "transition" suture which approximates the edge of the transversus abdominis aponeurosis to the medial wall of the femoral sheath and pectineus muscle fascia. This suture is necessary to close the angle and permit the line of closure to come up from the level of Cooper's ligament to the more superficial level of the anterior femoral sheath. This distance is represented by the diameter of the external iliac vein.

The remaining defect is closed by suturing the transversus abdominis aponeurosis or in case the layer is muscular at this point, the transversalis fascia, to the anterior layer of the femoral sheath. This re-establishes the normal continuity of transversalis fascia into the anterior femoral sheath. This line of sutures is continued laterally until a snug abdominal inguinal ring is made just as it is done for the repair of the small to medium sized indirect inguinal hernia. The spermatic cord is dropped in against the new posterior inguinal wall and the external oblique aponeurosis closed over it (Fig. 8 e and f) thus re-establishing the obliquity of the inguinal canal. The subcutaneous inguinal ring is snugly closed.

DIRECT INGUINAL HERNIA

Definition. A direct inguinal hernia is one that passes through the posterior inguinal wall medial to the inferior epigastric vessels in the area bounded by Hesselbach's triangle.

Etiology. The etiologic basis of the direct inguinal hernia is both congenital and acquired. The acquired factor in the development of this hernia is easy to understand because it is operative in the development of all hernias and is due to increased intra-abdominal pressure from whatever cause. Sudden or abrupt increase in the intra-abdominal pressure is more effective in producing a hernia than is a gradual increase in pressure.

The congenital predisposition to the development of a direct inguinal hernia is undoubtedly the most important etiologic factor. The robust common laborer is not prone to develop a direct inguinal hernia, nor is the asthmatic with paroxysms of violent coughing certain to develop this type of hernia. On the other hand, the asthenic individual with visceroptosis is not only the ideal candidate for the development of a direct inguinal hernia, but he is also more likely to have hemorrhoids and varicose veins than is a person of other body type. This suggests a congenital weakness of connective tissue throughout the body.

Direct inguinal hernia is a rarity in the female. This has been attributed to the flat, gynecoid pelvis of the female and a more even distribution of intra-abdominal pressures than in the male. While these anatomic facts may help to explain the low incidence of direct inguinal hernia in the female, it is also true that the posterior inguinal wall is almost always a heavy aponeuroticofascial plate in the female. Just why this is so when as a rule the male has the better muscular development, cannot be explained, but it has been observed in the dissection of many cadavers and almost a thousand operations.

Irrespective of sex, body build, occupation or associated diseases that increase intra-abdominal pressure, the most important predisposing cause for the development of a direct inguinal hernia is a weak posterior inguinal wall. The posterior inguinal wall is the transversus abdominis aponeurosis with its investing fasciae and the strength of this wall is directly proportional to the number of aponeurotic fibers. The transversalis fascia is the innermost fascial layer and, by itself, has very little tensile strength. There is considerable variation in the strength of this layer even in those individuals who never develop a direct inguinal hernia.

There is also considerable variation in the internal oblique layer in the inguinal region which has been observed and recorded by a number of surgeons, but we now consider

this layer of secondary importance in the development of direct inguinal hernia.

Occasionally direct external trauma may be the etiologic basis of a direct inguinal hernia and, certainly injury to the posterior inguinal wall during the repair of an indirect inguinal hernia or a femoral hernia will cause a direct inguinal hernia. In this instance, it is usually a "diverticular" type of direct inguinal hernia (Fig. 5 d).

Incidence. This hernia represents about 20 per cent of the groin hernias and from 10 to 15 per cent of all parietal abdominal hernias. While it is readily seen that this is not as common as the indirect inguinal hernia, it is the direct inguinal hernia which has baffled surgeons in the past half century and it is the hernia that is most likely to recur. The conventional inguinal ligament herniorrhaphies have been notably unsuccessful in repairing the direct inguinal hernia, recorded recurrence rates varying from 8 to 50 per cent.

Anatomy. The normal anatomy of the inguinofemoral region is presented in Figure 1, in which the number 2 represents the site of direct inguinal hernia. Whatever combination of factors operate to cause a direct inguinal hernia, the immediate cause is, of course, a dissolution of the integrity of the posterior inguinal wall.

The most common type of direct inguinal hernia is not an actual hole through the posterior inguinal wall, but a gradual attenuation of an intact but congenitally weak transversus abdominis aponeurosis. It is weak because of a deficient number of aponeurotic fibers. As the bulge of the hernia becomes larger and larger the aponeurotic fibers are spread farther and farther apart. The fasciae, of which the innermost or transversalis fascia is the heaviest, stretch but maintain the continuity of this layer.

As the transversus abdominis layer gives way and bulges forward, the overlying internal oblique muscle is stretched and pushed superiorly. The spermatic cord, with its investing cremaster usually remains below the bulge of the hernia. This is due to the disruption of the delicate fascial connection between the lower edge of the internal oblique and the cremaster. A strong musculoaponeurotic internal oblique layer may slow down the development of a direct inguinal hernia, but it cannot prevent it if the underlying transversus abdominis layer gives way. The significance of the so-called inguinal triangle in the causation of a direct inguinal hernia is certainly questionable.

However this much is true—a deficient internal oblique layer in the inguinal region is frequently associated with a deficient transversus abdominis layer. The important point to understand is that the transversus abdominis aponeurosis with its investing fasciae is the key to the development of a direct inguinal hernia. If this layer is strong and intact a direct inguinal hernia will not develop even though the internal oblique and external oblique layers are very weak or deficient. On the other hand if the transversus abdominis layer begins to attenuate and bulge the patient will develop a direct inguinal hernia in spite of a strong overlying internal oblique muscle.

The strength of the external oblique aponeurosis is of even less importance than the internal oblique muscle for the developing hernia easily protrudes through this layer between the crura of the subcutaneous inguinal ring. This space between the aponeurotic crura of the external oblique aponeurosis is commonly referred to as a ring, actually it is a long triangle with the aponeurotic defect bridged by the innominate fascia. It is variable in strength, depending upon the number of intercrural fibers. When the hernia has passed through the external oblique layer it enlarges in size in the subcutaneous fascia in the vicinity of the subcutaneous inguinal ring.

Hesselbach's triangle, bounded by the inguinal ligament, the inferior epigastric vessels and the lateral border of the rectus abdominis muscle, is a convenient descriptive area. Direct inguinal hernias must pass through this triangle. If a hernia occurs lateral to the inferior epigastric vessels it is an indirect inguinal hernia. If a hernia occurs below the inguinal ligament, it is a femoral hernia. If a hernia occurs above the inferior epigastric vessels in the linea semilunaris, it is a Spiegel's hernia. Occasionally the sac of a direct inguinal hernia will straddle the obliterated hypogastric artery to form a pantaloon type of sac. However this is a curiosity rather than of any particular significance and we place no emphasis upon whether the hernia has presented through the suprapubic fossa or the medial inguinal fossa.

The pathologic anatomy of the common type of direct inguinal hernia has been described. Rarely one sees a direct inguinal hernia with a small neck and these are described as a diverticular type of direct inguinal hernia (Fig. 5 d). This hernia proceeds through the layers of the abdominal wall in

versus abdominis aponeurosis above, from abdominal inguinal ring to Cooper's ligament. Below the glistening margin of Cooper's ligament must be seen medially, and the edge of the anterior femoral sheath laterally. When these margins have been carefully dissected and the peritoneal sac excised, one is then ready to repair the hernia (Fig. 8 c).

After making the relaxing incision (Fig. 8 a and b) the strong cut edge of the transversus abdominis aponeurosis is sutured to Cooper's ligament from the pubic tubercle to within a few millimeters of the external iliac vein (Fig. 8 d). This maneuver not only reconstructs the posterior inguinal wall but it re-establishes a normally broad insertion into Cooper's ligament and thus obviates the possibility of the development of a femoral hernia. It should be noted that the relaxing incision (Fig. 8 d) is now a considerable defect in the rectus sheath, protected behind by the rectus and pyramidalis muscles and their fasciae.

The next suture is the "transition suture" which approximates the edge of the transversus abdominis aponeurosis to the medial wall of the femoral sheath and pectineus muscle fascia. This suture is necessary to close the angle and permit the line of closure to come up from the level of Cooper's ligament to the more superficial level of the anterior femoral sheath. This distance is represented by the diameter of the external iliac vein.

The remaining defect is closed by suturing the transversus abdominis aponeurosis or in case the layer is muscular at this point, the transversalis fascia, to the anterior layer of the femoral sheath. This re-establishes the normal continuity of transversalis fascia into the anterior femoral sheath. This line of sutures is continued laterally until a snug abdominal inguinal ring is made just as it is done for the repair of the small- to medium-sized indirect inguinal hernia. The spermatic cord is dropped in against the new posterior inguinal wall and the external oblique aponeurosis closed over it (Fig. 8 e and f) thus re-establishing the obliquity of the inguinal canal. The subcutaneous inguinal ring is snugly closed.

DIRECT INGUINAL HERNIA

Definition. A direct inguinal hernia is one that passes through the posterior inguinal wall medial to the inferior epigastric vessels in the area bounded by Hesselbach's triangle.

Etiology. The etiologic basis of the direct inguinal hernia is both congenital and acquired. The acquired factor in the development of this hernia is easy to understand because it is operative in the development of all hernias and is due to increased intra-abdominal pressure from whatever cause. Sudden or abrupt increase in the intra-abdominal pressure is more effective in producing a hernia than is a gradual increase in pressure.

The congenital predisposition to the development of a direct inguinal hernia is undoubtedly the most important etiologic factor. The robust common laborer is not prone to develop a direct inguinal hernia, nor is the asthmatic with paroxysms of violent coughing certain to develop this type of hernia. On the other hand, the asthenic individual with visceroptosis is not only the ideal candidate for the development of a direct inguinal hernia, but he is also more likely to have hemorrhoids and varicose veins than is a person of other body type. This suggests a congenital weakness of connective tissue throughout the body.

Direct inguinal hernia is a rarity in the female. This has been attributed to the flat, gynecoid pelvis of the female and a more even distribution of intra-abdominal pressures than in the male. While these anatomic facts may help to explain the low incidence of direct inguinal hernia in the female, it is also true that the posterior inguinal wall is almost always a heavy aponeuroticofascial plate in the female. Just why this is so when as a rule the male has the better muscular development, cannot be explained, but it has been observed in the dissection of many cadavers and almost a thousand operations.

Irrespective of sex, body build, occupation or associated diseases that increase intra-abdominal pressure the most important predisposing cause for the development of a direct inguinal hernia is a weak posterior inguinal wall. The posterior inguinal wall is the transversus abdominis aponeurosis with its investing fasciae and the strength of this wall is directly proportional to the number of aponeurotic fibers. The transversalis fascia is the innermost fascial layer and, by itself, has very little tensile strength. There is considerable variation in the strength of this layer even in those individuals who never develop a direct inguinal hernia.

There is also considerable variation in the internal oblique layer in the inguinal region which has been observed and recorded by a number of surgeons, but we now consider

(indirect or femoral) sac which, of course, must be opened. This is known as the Hoguet maneuver.

Before beginning the reconstruction of the posterior inguinal wall (Fig. 8) it should be emphasized that the abdominal inguinal ring should be carefully explored from within to rule out the possibility of an incipient indirect inguinal hernia. Also the contents of the femoral canal should be examined to rule out the possibility of a femoral hernia. In the obese patient, there is frequently a large amount of preperitoneal fat down in the femoral canal and this is always removed before beginning the hernia repair.

After disposing of the hernial sac, or sacs, as the case may be, the reconstruction of the posterior inguinal wall is performed exactly as described for the large indirect inguinal hernia (Fig. 8). In repairing the diverticular type of direct inguinal hernia, most of the posterior inguinal wall can be saved, but, even so, the relaxing incision is made to avoid any tension on the suture line.

In the large indirect inguinal hernia there is frequently a small triangle of posterior inguinal wall which remains in the angle between Cooper's ligament and the lateral border of the rectus muscle. This represents the inguinal falx in many cases and the attachment of the new posterior inguinal wall can begin here. In the large direct inguinal hernia, however, the suturing must begin at the pubic tubercle.

FEMORAL HERNIA

Definition. A femoral hernia is one which passes through the femoral ring and down the femoral canal to become subcutaneous in the fossa ovalis. It is simply a third variety of inguinal hernia.

Etiology. The etiologic basis of the femoral hernia is undoubtedly a combination of both congenital and acquired factors, however we now consider the congenital predisposition to be the more important. The inclination of the female pelvis and increased intra-abdominal pressure due to pregnancy or to any other cause, certainly play an etiologic role in the development of this hernia. Obesity with an increase in fat in the preperitoneal layer and down into the femoral canal, may also play a role in the development of a femoral hernia, but a congenital peritoneal sac is not a factor.

The transverse diameter of the femoral ring in 100 consecutive cadavers was measured and found to vary from 5 mm. to 16

mm. An additional observation was that the larger the femoral ring, the narrower the attachment of the posterior inguinal wall into Cooper's ligament. Conversely when the femoral ring was narrow in transverse diameter the attachment of the posterior inguinal wall to Cooper's ligament was broad. In three of the cadavers a femoral hernia was present with a very broad femoral ring and a narrow insertion of the posterior inguinal wall into Cooper's ligament such as is illustrated in Figure 9 c. While this is not conclusive evidence of the etiologic basis of a femoral hernia, it is proof that there is considerable variation in the breadth of attachment of the posterior inguinal wall into Cooper's ligament with a corresponding inverse size of the femoral ring. A congenitally narrow insertion of the transversus abdominis aponeurosis (posterior inguinal wall) into Cooper's ligament may well be the principal cause of femoral hernia.

Incidence. The femoral hernia represents about 7.5 per cent of the groin hernias and between 3 and 4 per cent of all parietal abdominal hernias (Table 1). In our experience, it is four times more common in the female than in the male. It is most commonly seen in persons of middle age, but one of our patients with a bilateral femoral hernia was a two-month-old male infant.

Anatomy. The normal anatomy of the inguinofemoral region is presented in Figure 1. The number 3 represents the site of the femoral ring and is, of course, where a femoral hernia begins. There are several features of the normal anatomy of this region which are worthy of special mention. The broad attachment of the transversus abdominis aponeurosis (posterior inguinal wall) and the narrow femoral ring as shown in posterior view (Fig. 1 b) are the normal state and prevent the development of a femoral hernia. It should also be noted that the site of origin of a femoral hernia is as truly inguinal as either the indirect inguinal or the direct inguinal hernia—numbers 1 and 2 respectively (Fig. 1 a and b). The reason for emphasizing the inguinal origin of the femoral hernia is so the student will understand why the inguinal approach is the only rational route for the repair of this hernia.

Anatomy texts state that the medial wall of the femoral ring is the lacunar ligament. By comparing the anterior and posterior views (Fig. 1 a and b) it will be readily apparent that this is not true. The medial wall or margin of the femoral ring is the lateralmost attachment of the posterior in-

a similar manner and pushes out through the subcutaneous inguinal ring. It may also extend into the scrotum. This type of hernia has been seen following a previous repair of an indirect inguinal hernia and probably represents a tear in the posterior inguinal wall at the time of surgery. When it develops without previous surgery it is probably on the basis of the lipoma theory of Cloquet for on a number of occasions we have noted small protrusions of preperitoneal fat through the aponeurosis of the transversus abdominis while repairing an indirect inguinal hernia. When they were multiple, we have repaired the posterior inguinal wall as for a direct inguinal hernia. When single and small, we have simply reduced the fat and closed the tiny rent with a single fine silk suture.

Diagnosis. The diagnosis of a direct inguinal hernia is usually made with ease. The presence of a globular mass over the subcutaneous inguinal ring (Fig. 5 a) which reduces readily with recumbency immediately suggests the diagnosis. However one cannot be certain without palpation. Compression over the abdominal inguinal ring does not prevent the appearance of the hernial bulge with standing or straining, as is the case in the small to medium sized indirect inguinal hernia. While this is also true in the case of a large indirect inguinal hernia, the large indirect variety is always completely scrotal. When a direct inguinal hernia extends into the scrotum it is only into the upper scrotum and the spermatic cord and testis can be separated from the mass of the hernia.

With the examining finger pushing in vaginated scrotal skin through the subcutaneous inguinal ring, it is readily apparent that the posterior inguinal wall is gone. When the patient strains against the examining finger there is no feeling of resistance and, in spite of the muscular effort, the finger can palpate Cooper's ligament on the superior pubic ramus with ease and even feel the posterior aspect of the pubic bone.

The inexperienced may confuse a femoral hernia with a direct inguinal hernia, especially when the femoral hernia turns back upon itself and comes to lie over the inguinal ligament near the pubic tubercle. Careful digital examination, however, will demonstrate the neck of the sac and a portion of the body of the sac lying below the inguinal ligament. In the case of a direct inguinal hernia, the hernial mass is principally above the inguinal ligament. Other patho-

logic conditions in the groin are quite readily differentiated from the direct inguinal hernia.

The typical direct inguinal hernia reduces with ease and does not become incarcerated. This is due to the large size of the ring or defect in the posterior inguinal wall. For this reason, this hernia is usually painless although it may cause a heavy feeling or be described by the patient as a dragging sensation. The obese patient is the most likely to complain of discomfort because of the pressure of a large quantity of preperitoneal fat pushing through the defect. There may be enough pressure to cause urinary symptoms because the urinary bladder invariably forms a portion of the medial wall of the hernial sac. The uncommon diverticular type of direct inguinal hernia not only frequently incarcerates but may strangulate; this is due to the small rigid ring through the posterior inguinal wall. Whereas the direct inguinal hernia with a large defect will not get the patient into serious difficulty and operation is not urgent, the diverticular type is potentially dangerous and should be corrected surgically without delay.

Treatment. The rational repair of a direct inguinal hernia must include the replacement of the destroyed posterior inguinal wall. Various methods of accomplishing this have been discussed under the treatment of the large indirect inguinal hernia and the same principles apply to the direct inguinal hernia. In general, the only difference between the two hernias as regards the technique of repair is the different management of the peritoneal sac and the fact that the direct inguinal hernia usually destroys more of the posterior inguinal wall. Whereas dissection and high ligation of the peritoneal sac are mandatory in the operation for indirect inguinal hernia, the diffuse bulging of the direct inguinal hernial sac with its broad neck does not necessitate even opening the peritoneum. The sac is simply pushed in and held with a retractor while the posterior wall is reconstructed. However in the rare diverticular type of direct inguinal hernia, or if there is any question about adherent viscera to the larger sac, the sac is always opened, but it must be remembered that the urinary bladder forms its medial wall. In the event that there is an associated indirect inguinal or a femoral hernia, the excess of peritoneum in the direct inguinal hernial sac is pulled into the other

guinal wall into Cooper's ligament. The lacunar ligament lies more medially by at least 1 cm. and in a more superficial plane. It should also be noted that the inguinal and lacunar ligaments cannot be seen in a posterior view of the inguinal region. Those figures commonly used in textbooks which show the inguinal and lacunar ligaments in a posterior view are artifactual in that the posterior inguinal wall has either been removed or else it is shown inserting into the inguinal ligament. In the presence of a femoral hernia, the medial wall of the neck of the hernia does abut against the lacunar ligament (Fig. 9 b and c) but this is a pathologic state. Furthermore the inguinal and lacunar ligaments are not the primary ring of the hernial defect, although they are the final restraining structures. When it is necessary to cut the inguinal ligament to reduce a femoral hernia, it can be plainly seen that the true ring of the femoral hernial defect is the femoral sheath and the aponeuroticofascial fibers of the posterior inguinal wall en route to an insertion into Cooper's ligament (Fig. 9 b and c).

Irrespective of the number of factors which combine to permit the development of a femoral hernia, several progressive changes take place as the hernia enlarges. This hernia begins as a plug of fat in the femoral canal which gradually enlarges and eventually pulls a diverticulum of peritoneum through the femoral ring and down into the femoral canal. As more and more peritoneum pushes out through the femoral ring, the urinary bladder invariably becomes a part of the medial wall of the hernial mass. The hernial sac characteristically has a liberal covering of preperitoneal fat and the bladder on the medial wall is not immediately apparent (Fig. 10 a and b). The course of the developing hernia is down the femoral canal where it is forced out of the fossa ovalis by the vessels and the deep fascia. It becomes subcutaneous, pushing the cribriform fascia ahead of it, and at this point the hernia is femoral in location and appears as illustrated in Figure 9 a. As the hernia increases further in size, it turns cephalad and may eventually lie in part above the level of the inguinal ligament.

The most important anatomic change takes place at the femoral ring, for as the ring enlarges it pushes the lateral attachment of the transversus abdominis aponeurosis into Cooper's ligament medially there by progressively narrowing this tendinous insertion (Fig. 9 c) until the more super-

ficially placed lacunar ligament is reached. This is the final and greatest size of the femoral ring. It is a rigid and unyielding ring and accounts for the high incidence of incarceration and strangulation in this type of hernia. The other boundaries of the femoral ring are correctly recorded in all textbooks of anatomy and need not be elaborated upon, except to state that the immediate anterior boundary of the femoral ring in both normal anatomy and in the presence of a femoral hernia is the anterior femoral sheath, reinforced more superficially by the inguinal ligament. The direct inguinal hernia and the large in direct inguinal hernia destroy the posterior inguinal wall completely (Fig. 4 c and 5 c). It should be noted that a femoral hernia destroys the lateral half of the inferior portion of the posterior inguinal wall (Fig. 9 c).

Diagnosis. The diagnosis of a femoral hernia is usually not difficult but to superficial inspection a femoral hernia may closely resemble a direct inguinal hernia when it curves upward over the inguinal ligament. After the pubic tubercle and inguinal ligament are identified, the neck of the hernia can be felt below the inguinal ligament. In the slender patient, palpation is easy but in the obese female it may be difficult for the palpating fingers to become oriented. A direct inguinal hernia reduces with ease the diverticular type excepted, but a femoral hernia is usually incarcerated. An indirect inguinal hernia that just protrudes through the subcutaneous inguinal ring may superficially resemble a femoral hernia, but careful palpation will serve to differentiate the two. Varicocele of the upper scrotum or labia and a saphenous vein varix may slightly resemble a femoral hernia and it must be remembered that a sacular varix in this location also imparts an impulse on coughing. An enlarged subinguinal lymph node in the fossa ovalis, especially in the presence of lymphadenitis or metastatic neoplasm, may exactly mimic an incarcerated or strangulated femoral hernia. When this differential diagnosis presents itself a careful search should be made for wounds or other inflammatory lesions on the corresponding lower extremity, genitalia and rectum. A very trivial inflammatory lesion between the toes frequently associated with a fungus infection, can cause severe femoral lymphadenitis, a small fissure in ano can do the same. Subinguinal lymphadenitis and a strangulated femoral hernia are both exquisitely

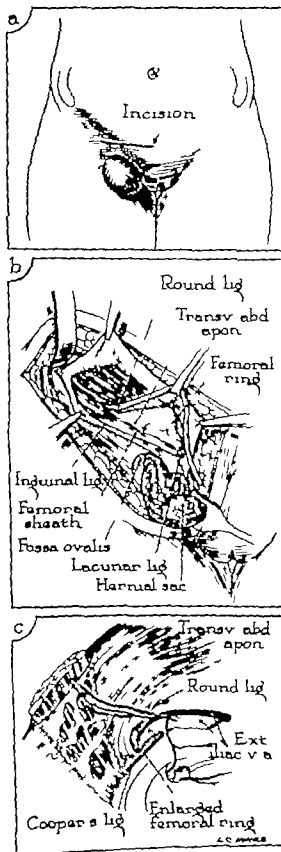


Fig. 9

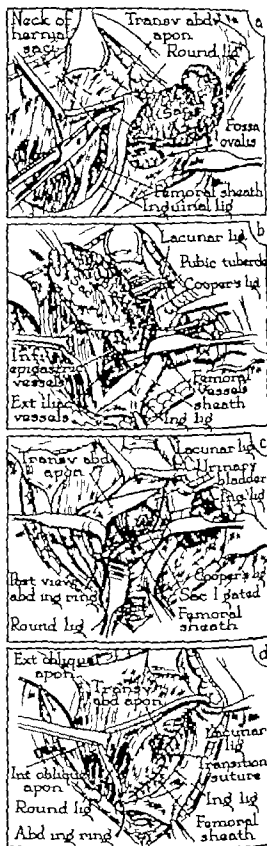


Fig. 10

Figure 9. a, Appearance of a femoral hernia with location of skin incision. b, Anterior view of the anatomy of a femoral hernia. c, Posterior view of the dilated femoral ring.

Figure 10. Successive steps in the femoral hernioplasty (viewed from right). a, Opening of the lower inguinal region to expose the neck of the femoral hernia. b, Conversion of the femoral hernial sac into the position of a direct inguinal hernial sac. c, Sac excised and the anatomy of the area prior to the hernioplasty; relaxing incision not necessary. d, Femoral hernioplasty; note similarity to Figure 8, d and c.

small retractor is used to elevate the inguinal ligament so that the constricting ring of the femoral sheath may be cut. For most femoral hernias this maneuver suffices for the reduction of the hernia. However if by a combination of gentle traction above and pressure from below the hernia cannot be reduced, the lacunar portion of the inguinal ligament is cut part way through or if necessary completely divided. After the sac has been pulled out of the femoral canal, it is in the position of a diverticular direct inguinal hernia (Fig. 10 b). A femoral hernial sac must always be opened because it frequently contains adherent omentum or viscera, the operator bearing in mind that the urinary bladder is part of the medial wall of the fatty envelope which surrounds the peritoneal sac (Fig. 10 b). With the sac opened, one should pass a finger into the abdominal cavity and feel for the opening of an indirect inguinal hernia sac, combining this with direct inspection of the abdominal inguinal ring. The inside of the abdominal inguinal ring can be easily inspected by retraction (Fig. 10 c) if for no other reason than to familiarize oneself with the construction of a normal abdominal inguinal ring. The hernial sac is next ligated at its base and the excess excised, again remembering the position of the urinary bladder.

The hernia is then repaired by suturing the cut edge of the posterior inguinal wall to Cooper's ligament up to within a few millimeters of the vein (Fig. 10 d). This not only reconstructs a normally attached posterior inguinal wall but closes the femoral ring and thus corrects the defect which permitted the hernia to develop. The relaxing incision is not necessary. The transition suture (Fig. 10 d) picks up the fragmented end of the anterior femoral sheath and a liberal bite of the pectineus muscle fascia and approximates them to the posterior inguinal wall. This suture not only re-establishes the medial wall of the femoral sheath but is necessary to close the angle between the level of Cooper's ligament and the more superficial anterior layer of the femoral sheath. The operation is completed by approximating the transversalis fascia to the anterior layer of the femoral sheath (Fig. 10 d). The external oblique aponeurosis is closed over the cord or round ligament and the subcutaneous fascia and skin are sutured as for the other groin hernia.

SLIDING HERNIA

The sliding hernia is not a special type of hernia but rather represents a complication

in the management of the hernial sac in several types of hernias. A sliding hernia is most commonly encountered in groin hernias. The entity sliding hernia, is most easily explained by describing the mechanism of its development. It occurs most commonly in the indirect inguinal hernia because this is the most common of all hernias but it should be understood that there is a sliding hernia of the urinary bladder in almost all femoral hernias (Fig. 11 c) and direct inguinal hernias.

In the early stages of an indirect inguinal hernia, the hernial sac consists solely of the congenital diverticulum of peritoneum and the hernia may enlarge considerably by stretching of the sac. However when the hernia is of long standing and because of the pressure of its contents, further enlargement of the hernial sac comes from a sliding downward of parietal peritoneum. The peritoneum proximal to the orifice of the hernia is least tightly fixed in the iliac fossa. As this peritoneum is pulled into the hernial sac, the cecum (Fig. 11 a) or sigmoid colon (Fig. 11 b) with a short or absent mesentery becomes part of the posterolateral wall of the hernial sac. The bowel is outside the lumen of the hernial sac and presents an additional problem in the high ligation of the sac. The peritoneum cannot be dissected off the viscus because it is visceral peritoneum. Therefore the viscus is simply cut out of the side of the hernial sac which, of course, leaves a rent in the wall of the sac. The management of this is a technical problem which is easily handled. Care must be exercised in dissecting the viscus away from the hernial sac because of the mesenteric vessels.

Another mechanism for the development of a sliding type of hernia is seen in complete descent of the testis or in the abnormal descent of an ovary into the inguinal canal (Fig. 11 d). In the former this is simply an arrest in the normal mechanism of the descent of the testis into the scrotum. In the latter the mechanism is the same although it is an abnormality for the ovary to descend into the inguinal canal. When either of these two abnormalities exists there is invariably an indirect inguinal hernia associated, although the peritoneal sac may not contain a viscus.

In a diaphragmatic hernia, a variety of retroperitoneal viscera may "slide" into the thoracic cavity. We have observed the spleen, liver, kidney, adrenal and tail of pancreas in a left-sided diaphragmatic hernia, in addition to the several hollow viscera.

tender which makes diagnostic palpation more difficult.

Occasionally premonitory urinary symptoms such as dysuria, hematuria and frequency will precede any palpable evidence of a femoral hernia by months. This is due to a knuckle of the urinary bladder being caught during the early development of the hernia. A Littre's hernia, in which only a portion of the circumference of the small intestine may be incarcerated or strangulated in the ring, is especially common in a femoral hernia. A femoral hernia should always come to mind when one is confronted with a case of unexplained intestinal obstruction. The appendix, ovary or Meckel's diverticulum is occasionally found in a femoral hernia. In this event, it is usually strangulation of the contained viscus which brings the patient very promptly to the physician.

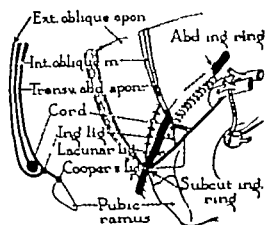
Treatment. The variety of operations described for the repair of a femoral hernia almost equals the number recorded for the repair of indirect and direct inguinal hernias and, in like measure, reflects basic misunderstanding of the pathologic anatomy. To review the many operations for femoral hernia would only serve to cause confusion and, as a matter of fact, the majority of these operations are better left unlearned except for those interested in the history of surgery in this region. The femoral approach is anatomically unsound. The many techniques in which an attempt is made to suture the inguinal ligament to Cooper's ligament even by the inguinal approach are likewise an anatomic mistake. The inguinal ligament has mobility in a cephalad direction, but it is a taut cord when attempts are made to displace it posteriorly and approximate it to Cooper's ligament as far laterally as the femoral vein. In any event, suturing the inguinal ligament to Cooper's ligament by either the femoral or the inguinal approach is an anatomic and unsound surgical procedure, for the defect is more deeply placed. The defect in a femoral hernia, irrespective of the causative factors, is a narrowing of the insertion of the transversus abdominis aponeurosis and its fused fasciae (posterior inguinal wall) into Cooper's ligament. The repair of a femoral hernia, after removal of the hernial sac, is correctly accomplished by broadening this insertion so as to obliterate the femoral ring. This concept was correctly understood many years ago by Lotheissen and in recent years by others. A femoral hernioplasty is one of

the nicest operations in the entire field of surgical anatomy when the surgeon understands both the normal and pathologic anatomy of this region. The aponeurotic fascial layers are invariably strong and, in the absence of associated inguinal hernias, it offers the surgeon an unparalleled opportunity to observe normal inguinal anatomy.

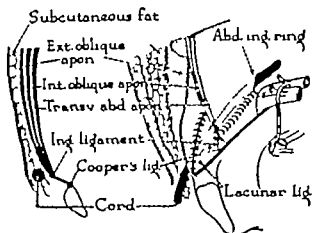
The skin incision for a femoral hernioplasty is the same as that for the other inguinal hernias (Fig. 9 a). The external oblique aponeurosis is incised and the inguinal region exposed by elevating the spermatic cord or the round ligament exactly as for an inguinal hernioplasty (Fig. 9 b). At this point, the posterior inguinal wall and the abdominal inguinal ring are carefully inspected for associated hernias. The abdominal inguinal ring is inspected again later from within. The hernial sac is shelled out of its subcutaneous femoral position (Fig. 10 a) until the neck of the sac is reached. If there is any question of non-viable contents, the sac should be opened at this time for careful inspection before releasing the constriction of the femoral ring. It is a disquieting experience to have a loop of bowel disappear into the abdominal cavity before it is examined. Should this happen, one is then obliged to inspect the adjacent colon and all of the small bowel with a mesentery sufficiently long to permit its entrance into the hernial sac. This precaution, as regards releasing the constriction at the neck of the hernia, applies to all types of hernias with questionably strangulated contents.

An incision is next made in the femoral sheath immediately above the inguinal ligament and carried over the bulge of the neck of the hernia and down to Cooper's ligament (Fig. 10 a). This allows access to the neck of the hernia and, later for the repair of the hernia. When this incision is made, one has, of course, entered the preperitoneal space and the next step is to separate the neck of the hernia from the external iliac vein. This is easily accomplished, as there is always a nice plane of cleavage due to the areolar tissue surrounding the vein. Tributary blood vessels must be searched for carefully doubly ligated and cut. This is especially true when an aberrant obturator artery has been pushed forward by the hernia. Aberrant obturator veins are commonly seen. The small artery and vein to the pyramidalis muscle, which course along the edge of Cooper's ligament, are invariably present.

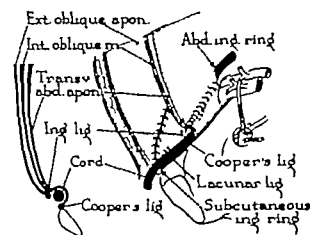
After the external iliac vein is identified a



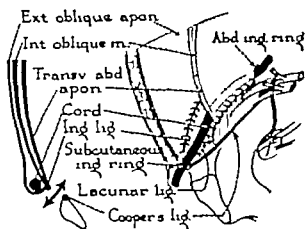
a BASSINI



b HALSTED I

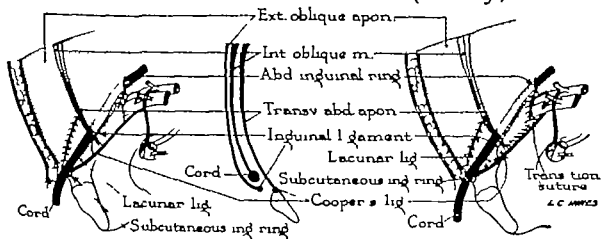


c FERGUSON



d BASSINI in DIRECT HERNIA

e
ABDOMINAL INGUINAL
RING REPAIR (Mc Vay)



f
RECONSTRUCTION OF
POSTERIOR INGUINAL
WALL (Mc Vay)

Figure 12. A diagrammatic comparison in sagittal and oblique posterior views of several methods of inguinal hernioplasty. Crosses indicate the continuation of the suture line. a, Bassini operation for a small indirect inguinal hernia. b, Halsted I operation for a small indirect inguinal hernia. c, Ferguson operation for a small indirect inguinal hernia. d, Bassini operation for a direct inguinal hernia. e, Author's method of repairing small- to medium-sized indirect inguinal hernias (see also Figure 6 a and b). f, Author's method of repairing all large indirect inguinal, direct inguinal and femoral hernias.

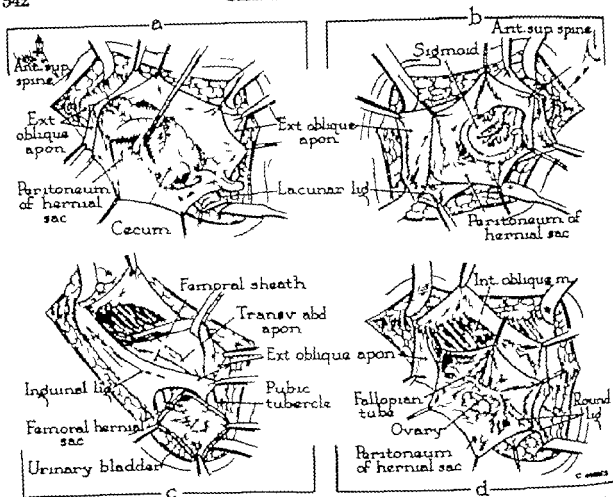


Figure 11 Varieties of sliding hernia. a, Cecum in a right indirect inguinal hernia. b, Sigmoid colon in a left indirect inguinal hernia. c, Urinary bladder in a femoral hernia. d, Fallopian tube and ovary in a right indirect inguinal hernia.

On the right side the liver, adrenal gland and even the superior pole of a kidney have been found in a posterior diaphragmatic hernia through the pleuroperitoneal foramen of Bochdalek. On one occasion a portion of the right adrenal was inadvertently removed in the repair of this hernia before the structure was recognized.

COMPARISON OF SEVERAL METHODS OF INGUINAL HERNIOPLASTY

Although we are opposed to the use of the inguinal ligament in the repair of any groin hernia, the approximation of the "conjoined tendons" to the "shelving border" of Poupart's ligament is so widely accepted that every surgeon should be conversant with certain operations which use the inguinal ligament as the anchoring structure. Although the number of these operations recorded is in excess of seventy they are all variations of three classic procedures and the difference between these three operations is the position in which the spermatic cord is placed. Figure 12 was prepared in

semidiagrammatic form to show the fundamental features of these three operations and then, for comparison what we believe should constitute an adequate hernia repair.

Before studying these diagrams reference should be made to Figure 1 to become familiar with the normal anatomy of the inguinal region. It is readily seen that the posterior inguinal wall does not insert into, or attach to the inguinal ligament, except that near the pubic tubercle the lacunar portion of the inguinal ligament and the posterior inguinal wall have a common insertion into Cooper's ligament. It is upon this anatomic fact, plus the inadequacy of the inguinal ligament as a substitute that we believe the principles for the repair of the groin hernias should be based. Should it quite justifiably be pointed out that the inguinal ligament hernioplasty has stood the test of time with a very favorable cure rate, it should be clearly understood that the majority of inguinal hernia operations (Table 1) are for the relatively small indirect inguinal hernia, where success will attend al-

a, b and c with o) All of the diagrams in Figure 12 a, b c and o represent techniques for the repair of the small indirect inguinal hernia and in all of these procedures the posterior inguinal wall is perfectly intact. The crux of the matter is this: when the pathologic condition is simply a small congenital indirect inguinal hernial sac with only slight dilatation of the abdominal inguinal ring, suturing of the posterior inguinal wall to Poupart's ligament (Fig. 12 a, b and c) is unnecessary. Not only is it unnecessary but there is danger of tearing this wall with subsequent herniation through the defect. Very adequate repair of this most common of all hernias is illustrated in Figure 12 c and consists of simply tightening the abdominal inguinal ring to normal size by suturing the transversalis fascia to the anterior layer of the femoral sheath. In children, one suture frequently suffices and not three as depicted. It should also be noted that the spermatic cord (Fig. 12 o) is in the normal position as it is in the Bassini operation (Fig. 12 a).

For this comparative study the Bassini operation was selected as an example of inguinal ligament hernioplasty for the repair of a direct inguinal hernia (Fig. 12 d). Whereas it was pointed out that suturing the posterior inguinal wall to the inguinal ligament was unnecessary in the small indirect inguinal hernia, it is equally important to note that it is anatomically incorrect to suture the posterior inguinal wall to the inguinal ligament in a direct inguinal hernia. When the posterior inguinal wall is destroyed, as in the case of a direct or a large indirect inguinal hernia, the reconstruction of a new posterior inguinal wall should include the attachment of this wall to its normal insertion, which is of course, Cooper's ligament (Fig. 12 f). The normal attachment of the posterior inguinal wall is accurately shown in Figure 1 a and b and diagrammatically demonstrated in Figure 12 a, b c and e. When the inguinal ligament is used as the anchoring structure in patients in whom the posterior inguinal wall has been destroyed, it leaves an anatomic fault (Fig. 12 d, double arrow) which is, in effect, a large femoral hernial ring. Some recurrent hernias are of this variety. More important, the parallel and easily separated fibers of the inguinal ligament are not a suitable anchoring structure for the repair of the large groin hernias. The normal insertion (Cooper's ligament) is readily available and is a very strong, unyielding structure.

HEINIAS OF THE LINEA ALBA AND THE LINEA SEMILUNARIS

These hernias are quite naturally grouped together since they have many features in common and occur along lines of aponeurotic fusion. The principles involved in their surgical repair are also very similar. Figure 13 is a posterior view of the abdominal wall showing the aponeurotic defect in an epigastric, an adult umbilical and a semilunar (Spiegel's) hernia. Opposite each hernia and indicated by the arrow is a transverse section through the abdominal wall to show the relationships of the various layers from skin to peritoneum.

UMBILICAL HERNIA

Definition. An umbilical hernia is one that passes through the umbilical ring and includes three types: omphalocele or fetal umbilical hernia (Fig. 14 a) which is a failure of a portion of the intestinal tract to return to the abdominal cavity during early fetal development; infantile umbilical hernia (Fig. 14 b) which is a failure of the umbilical ring to obliterate; adult umbilical hernia (Fig. 14 c) which is the acquired dilatation of an imperfectly closed umbilical ring.

Etiology. Omphalocele is due to the failure of complete return of the eviscerated organs to the abdominal cavity. This, in turn, is due to a spatial problem in that the abdominal cavity has not enlarged enough to accept all of the intestinal tract that has grown and rotated during its extrusion into the base of the umbilical cord. The size of an omphalocele varies from a single loop of small intestine to a large sac containing most of the abdominal viscera. An infantile umbilical hernia is due to faulty closure of the umbilicus as the umbilical vessels become obliterated. There seems to be a familial tendency in that several children in one family will have this type of hernia while none of the children in an equally large family presents this abnormality. From a racial standpoint, this hernia is much more common in members of the Negro race than in those of the white race. An adult umbilical hernia has a twofold etiologic basis in that there must be a congenital defect in the umbilical scar with the superimposed element of increased intra-abdominal pressure. A completely closed umbilicus presents a very dense interdigitation of aponeurotic fibers scarred together by the healing of the umbilical wound. No degree of intra-

most any operation that includes high ligation and excision of the peritoneal hernial sac. The large indirect inguinal hernia, the direct inguinal hernia and the femoral hernia have always had a high recurrence rate with inguinal ligament types of repair but the preponderance of small indirect inguinal hernias in any series has permitted a favorable and honest report of success. In the future the statistical analysis of the results in a given series of hernioplasties should break down the groin hernias into their respective types so that a true picture of hernia recurrence may be obtained.

The semidiagrammatic drawings (Fig. 12) represent a posterior view of the inguinal region in several types of hernioplasty with the medial portion of the posterior inguinal wall and the rectus abdominis muscle cut away. To the left of each figure and between the two bottom figures is a diagrammatic sagittal section at the level of the cut back in the main figure to show the attachment of the layers and the relative position of the spermatic cord. The small crosses on the inguinal lacunar ligament (Fig. 12 a, b c and d) and on Cooper's ligament (Fig. 12 f) represent the continuation of the line of sutures to the pubic tubercle. The peritoneum is not shown for it is presumed that the hernial sac has been adequately resected in all instances.

Credit for the first satisfactory inguinal hernia operation properly belongs to H. O. Marcy of this country as pointed out by Zimmerman and Anson. However the first satisfactory hernia operation to be widely accepted was described in 1889 by Edoardo Bassini. In addition to dissection and high ligation of the sac, the lower edge of the internal oblique muscle and the posterior inguinal wall (conjoined tendon) were sutured to the inguinal ligament from the pubic tubercle to the abdominal inguinal ring. The spermatic cord was placed against this wall and the external oblique aponeurosis closed over the cord, reconstructing the subcutaneous inguinal ring in its normal location (Fig. 12 a). In a general way this operation is the most rational because it reinforces the posterior inguinal wall and re-establishes the obliquity of the inguinal canal. Through the years the majority of operations have been based upon this principle with minor variations ad infinitum.

Almost simultaneously William S. Halsted, in this country in 1890 described an operation which has subsequently become known as the Halsted I (Fig. 12 b). Hal-

sted subsequently abandoned this technique and adopted a procedure essentially similar to the Bassini operation. In this country it became known as the Halsted II. The Halsted I operation also included high ligation of the peritoneal sac and the approximation of the conjoined tendon to Poupart's ligament. With the idea of making a stronger posterior inguinal wall the external oblique aponeurosis was closed deep to the cord, usually by plication, thus obliterating the inguinal canal. This procedure superimposes the subcutaneous inguinal ring upon the abdominal inguinal ring and leaves the spermatic cord in the subcutaneous fascia (Fig. 12 b). Although this principle is still widely used, it is faulty because it destroys the obliquity of the inguinal canal and the so-called shutter action of the muscles. It is attended by a high incidence of recurrence directly through the superimposed rings.

The third operation of classic significance is known as the Ferguson procedure although it was described many years before by Bull and Colley. Originally it was used to reinforce a supposed inadequacy in the origin of the internal oblique and transversus abdominis muscles from the inguinal ligament. The inguinal portions of these muscles do not originate from the inguinal ligament as described, but from the fascia lata immediately beneath. In later years this procedure has been advocated in patients with cryptorchidism to gain additional length of the spermatic cord. The procedure represents a third alternative for the placement of the spermatic cord (Fig. 12 c). After ligation of the hernial sac, the cord is pushed inward and the conjoined tendon sutured to Poupart's ligament over the cord. The external oblique aponeurosis is then closed over this wall. This technique places the abdominal inguinal ring just lateral to the pubic tubercle and immediately beneath the subcutaneous inguinal ring. The Ferguson operation also destroys the obliquity of the inguinal canal with its shutter action and is attended by a high incidence of recurrence just lateral to the pubic tubercle which is in effect a direct inguinal hernia.

If the Bassini, the Halsted I and the Ferguson operations (Fig. 12 a, b and c) are carefully compared, it will be apparent that the approximation of the layers is identical and that the difference lies in the placement of the spermatic cord. Of the three it is our opinion that the Bassini operation is the best. However at this point a very important fact should be observed (Fig. 12, compare

abdominal pressure will disrupt such an umbilicus. However, a small defect in the umbilicus, with increased intra abdominal pressure due to such conditions as pregnancy, intestinal obstruction, chronic cough and ascites, can cause an adult umbilical hernia without antecedent history of such a hernia. Obesity is associated with an umbilical hernia so commonly in the adult as to suggest the lipoma theory of Cloquet as a contributing etiologic factor.

Incidence. Omphalocele is a relatively rare congenital anomaly reported to occur about once in every 5000 to 6000 births. Infantile umbilical hernia is of very common occurrence, especially in members of the Negro race with the reported incidence varying from 1 to 10 per cent in white children and up to as high as 50 per cent in Negro children. Adult umbilical hernia is not uncommon but is less frequently seen than the infantile type. An accurate incidence of infantile and adult umbilical hernias is difficult to determine because unless these hernias are symptomatic, patients will frequently not consult a physician. However, these two types of umbilical hernias compose a substantial proportion of any series of hernias for which operations are performed (Table I average 9.4 per cent).

Anatomy. Normally at birth the umbilical defect is small and just accommodates the umbilical arteries, the umbilical vein, the urachus and a small amount of embryonic connective tissue. After ligation of the umbilical cord, the blood vessels become thrombosed back to their nearest collateral and eventually become fibrous cords. The umbilical vein becomes the round ligament of the liver lying in the free margin of the falciform ligament of the liver. The umbilical arteries become the obliterated hypogastric arteries which elevate the peritoneum into the lateral umbilical folds. The urachus obliterates and becomes the middle umbilical ligament over which the peritoneum folds as the middle umbilical fold.

The defunctionalized umbilical vessels and urachus apparently cannot keep the umbilicus open or stated another way it is possibly the pressure of the circulating blood in these vessels which prevents the umbilicus from closing prematurely with disastrous results for the fetus. It is also likely that the contraction of the abdominal musculature after birth exerts a shutter-like action at the umbilicus. In any event, irrespective of where the cord is ligated, the slough extends down to the aponeurotic aperture and,

in studying the disposition of the aponeurotic fibers about the umbilicus (Fig. 13) it is easy to understand how lateral muscles pull tends to close the umbilicus and tightly constrict the structures passing through it. When the cord sloughs, a granulating defect is left which adds scar tissue to the approximated aponeurotic fibers and forms a very dense composite wall that no amount of pressure will disrupt to result in an umbilical hernia. The umbilical skin grows in circumference under the umbilical eschar which accounts for the fixation of the umbilical skin to the linea alba.

Aberrations in this pattern of closure result in umbilical herniation. First, is the fetal umbilical hernia (Fig. 14 a) or omphalocele in which the defect cannot close because one or more viscera have been protruding through the umbilicus since the sixth week of intrauterine life and prevent normal development of the aponeurotic elements of the lateral musculature. The size of this defect depends upon the bulk of the protruding viscera. Ordinarily the viscera which protruded into the umbilical cord have returned by the tenth or eleventh week of intrauterine life have grown considerably and have completed the rotation that brings the transverse colon over the superior mesenteric vessels. When the viscera fail to return completely into the abdominal cavity, it is because the cavity has not enlarged sufficiently to receive them. When a child is born with an omphalocele, a situation exists which is not present in any other hernia and it demands prompt correction. The viscera lying in the umbilical cord are not covered with skin but by a very thin, translucent membrane which consists of fused peritoneum and amniotic membrane. With exposure to air it very rapidly dries and becomes necrotic so that peritonitis supervenes in a matter of hours.

The second type of umbilical hernia is the infantile umbilical hernia (Fig. 14 b) which makes its appearance either shortly after birth or in the first year or two of life, usually following an episode of increased intra abdominal pressure, which can be an almost hourly event attending crying, straining and coughing. This type of hernia is covered with the everted umbilical skin. The skin at the apex of the umbilical dimple is densely adherent to the linea alba at the umbilicus. On the posterior aspect of the umbilicus the peritoneum is equally adherent. Therefore there is peritoneum, umbilical scar and skin without the interposition of adipose tissue

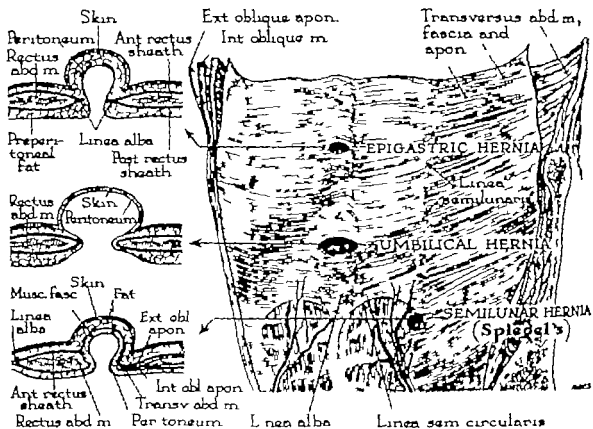


Figure 13. Posterior view of the anterior abdominal wall showing the musculospondylotic defects in an epigastric, an umbilical and a semilunar (Spiegel's) hernia. On the left are transverse sections through these hernial defects with the peritoneum and preperitoneal connective tissue added.

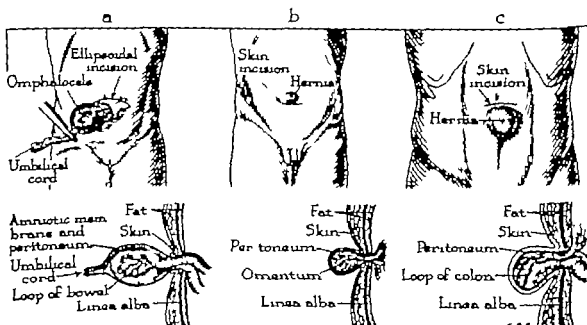


Figure 14. The appearance of the three types of umbilical hernias in anterior view and sagittal section. a, Fetal umbilical hernia or omphalocele. b, Infantile umbilical hernia. c, Adult umbilical hernia.

toms, vague upper gastrointestinal symptoms due to traction on the omentum are common. As the hernia enlarges and loops of intestine enter the sac partial intestinal obstruction with its associated symptoms appears. The transverse colon is usually the first hollow viscus to enter the hernia and is literally pulled in by the omentum. Strangulation of the contents of this hernia is very common because of the rigidity of the aponeurotic ring. Although this hernia is always unilocular rents in the incarcerated omentum make secondary constricting apertures which greatly aggravate the symptoms and may well be the precipitating element in strangulation.

Treatment. The fetal umbilical hernia, or omphalocele, demands immediate surgical treatment, which means within the first few hours after birth. In the small omphalocele, this presents no special problem because, properly managed, the infant withstands an operation well. The loop of intestine is easily reduced because there is no distention of the intestine with food and air. The repair of the aponeurotic defect is the same as for the infantile umbilical hernia and need not be elaborated upon. The only difference is that no attempt is made to fashion an umbilical dimple out of the cuff of periumbilical skin. This cuff of skin is excised as an ellipsoidal segment and removed with the omphalocele sac (Fig. 14 a). For the management of the large omphalocele, or when the abdominal cavity is too small to comfortably accommodate the exteriorized viscera, the two-stage procedure of Gross is certainly the operation of choice.

The infantile umbilical hernia which appears shortly after birth does not need immediate surgical correction unless it becomes incarcerated but this is a rare occurrence. Many of these hernias gradually disappear without any treatment owing to the gradual constriction of the defect by aponeurotic pull across the midline. In most instances it is wise to keep the hernia reduced, either by the use of tape and a block of sponge rubber or by the method of infolding the adjacent skin as described by Gross. Any hard object such as a coin button or marble should not be forced into the defect to keep the hernia reduced. A mechanical truss will not stay in place on a squirming infant. After the infant is approximately one year of age, taping the hernia is not effective and should be abandoned. If the hernia persists and is symptomatic after one year it should be repaired. If it persists and is asymptomatic, an elective operation can be performed at any time. A large umbilical hernia in an infant should be repaired without delay.

The infantile umbilical hernia is repaired quite simply through a small transverse incision either just above or just below the umbilicus (Fig. 14 b). The umbilical skin is carefully dissected off the hernial sac and the sac dissected free through the aponeurotic defect. After the sac is opened to be sure that there are no incarcerated contents, the neck of the sac is ligated with a fine silk suture (Fig. 15 a). The defect is then closed in the transverse plane as illustrated in Figure 15 b. Although closure of this defect in the vertical plane has given good results in reported series the transverse

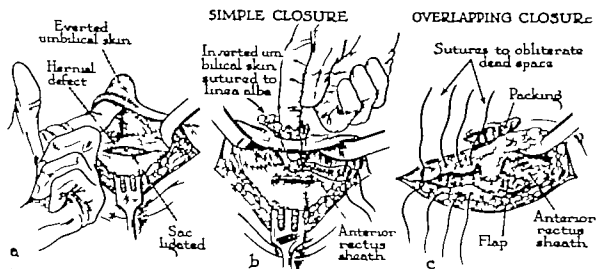


Figure 15. Umbilical hernioplasty. a, The sac ligated and the aponeurotic margins of the hernial defect ready to close. b, Simple transverse linear closure with the umbilical skin inverted and sutured to the linea alba. c, An alternative method in larger umbilical defects. The overlapping, vest-over-pants or Mayo operation.

and this accounts for the thin covering of these hernias when a defect persists in the umbilicus. Between the peritoneum of the hernial sac and the everted umbilical skin there is only the thinnest of areolar tissue layers. The healing of the umbilicus is essentially the same in all infants with approximately the same amount of scar tissue incident to the healing process. The arrangement that permits this hernia to develop must, therefore, be an abnormality in the disposition of the aponeurotic fibers about the fetal umbilical defect. When muscle fibers contract, the corresponding aponeurotic fibers attempt to make a straight line from their point of insertion (Fig. 18 c and d). The interdigitation of aponeurotic fibers about the umbilicus in three different planes corresponding to their origin from the muscle fibers of the two obliques and one transverse anterolateral abdominal muscle would tend to close effectively an aperture at the umbilicus. The protrusion of extraperitoneal fat is not an etiologic factor here as it is in epigastric and other hernias; neither are there perforating blood vessels other than the fetal ones. The foregoing is not advanced as the absolute cause of this type of hernia, but, like many others, through the years we have attempted to reach a rational explanation of the enigma of why some infants do and others do not, develop this hernia.

The third type of umbilical hernia is the adult umbilical hernia (Fig. 14 c). It is most commonly seen in persons of middle age, although not uncommonly in the obese multiparous female in her early twenties. Although increased intra-abdominal pressure is the precipitating cause of this hernia, the underlying etiologic condition is the persistence of a small defect from birth. As has been mentioned, the normally healed umbilicus will not develop a hernia. Small asymptomatic defects in the umbilicus, without a sac and without an impulse are a fairly common finding in routine physical examinations. Another common finding is a small pea-sized irreducible and nontender lump in the umbilical dimple. This is incarcerated omentum, as can be demonstrated by intra-abdominal exploration at the time of celiotomy performed for some other reason.

With a small defect containing a tag of omentum, it is not difficult to see that this is the entering wedge for the development of a typical umbilical hernia in the adult. Increased intra-abdominal pressure from whatever cause gradually pushes more and more

omentum into the defect and, eventually, transverse colon enters the hernia as the first viscus. Pregnancy is the most common etiologic factor. Obesity is an important contributing factor and ascitic fluid in an umbilical hernial sac may be the first indication that the patient has a hernia. Once established, the hernia progressively enlarges in size unless the cause of the pressure is relieved. It is not uncommon for an umbilical hernia to decrease in size and become asymptomatic after a woman has delivered. In this event, the woman with a previously painful umbilical hernia may refuse operation only to find that with the next pregnancy the hernia becomes larger and more symptomatic.

Diagnosis. The diagnosis of an umbilical hernia presents no problem even to the uninitiated, although occasionally a very small defect strangulating a tiny tag of omentum will be exquisitely tender and be difficult to feel in an obese patient. An epigastric hernia occasionally occurs just above the umbilicus, but, although it may bulge into the umbilical skin from above the apex of the umbilical dimple, remains fixed to the linea alba. Confusion on this differential point is of small consequence for the principles of repair of the aponeurotic defect are identical. A sebaceous cyst or urachal cyst may superficially resemble an umbilical hernia.

Symptomatically the omphalocele does not bother the newborn until peritonitis supervenes and this may be within a few hours. If the omphalocele sac is torn during delivery, peritonitis begins at once. An omphalocele should be immediately apparent, although a small loop of intestine may not be noticed unless the base of the umbilical cord is carefully inspected. To include a loop of small intestine in the ligature of the umbilical cord is to give the infant a strangulating obstruction with its attendant mortality.

The infantile umbilical hernia rarely incarcerates and is rarely symptomatic in the first year of life, although larger hernias in older children cause local pain and digestive disturbances. The mother invariably makes the diagnosis before the doctor sees the child.

The fully developed adult umbilical hernia is usually painful, frequently incarcerated and danger of strangulation is imminent. Omentum is usually the first organ to enter the hernial sac and, because of repeated trauma, shortly becomes adherent to the sac (incarcerated). At this stage, even though there are no significant local symp-

woven pattern closely covered by the fasciae which has led surgeons to refer to the sheaths loosely as "fasciae" and to cut them indiscriminately. The aponeurotic fibers in the rectus sheaths maintain their various directions as they mingle in their mutual intersection at the midline and form the linea alba. By careful dissection, and sometimes with the use of a magnifying glass it is possible to trace a given aponeurotic fiber through the linea alba into the opposite rectus sheath. It is undoubtedly this characteristic which led some of the earlier anatomists to consider the six anterolateral abdominal muscles as three digastric muscles. This, at least, serves a useful purpose from a teaching standpoint to remind the surgeon that midline hernias are in effect hernias through the tendons of the digastric muscles and that the repair of the hernia should take into consideration the lines of musculoaponeurotic pull.

If one carefully examines the linea alba in a large number of cadavers it will be noted that there is considerable variation in the concentration of the aponeurotic fibers from level to level. It is possible that a congenitally weak area is responsible for the development of an epigastric hernia. However it can also be observed that there are occasional blood vessels which also pass through the linea alba (Fig. 13) either in the midline or eccentrically placed next to borders of the rectus muscles. It is our opinion that it is the tiny foramina through which these blood vessels pass that provide the opening for the beginning of an epigastric hernia. This opinion is strengthened by the observation that even a very small epigastric hernia (hernia adiposa) is accompanied by one or more small blood vessels which will bleed significantly if the neck of the protruding fat is not ligated.

There is rarely any significant amount of

preperitoneal fat in the posterior aspect of the upper abdominal wall and the peritoneum is closely adherent to the transversalis fascia. However even in a slender individual there is always some adipose tissue between the folds of the falciform ligament of the liver. It is this fat that is the presenting part of an epigastric hernia and, in the small hernia adiposa that represents the entire hernial mass. It is this characteristic of the epigastric hernia which justifies the lipoma theory of Cloquet as the explanation of the cause of this hernia. It is certain that all epigastric hernias begin by the protrusion of a small lobule of fat which, in most instances at least, pushes through a perforating blood vessel foramen. As more fat is pushed out through the defect, the attached peritoneum is eventually pulled through and then a hernia with a peritoneal sac exists. As the hernia enlarges further omentum and the hollow viscera enter the sac. Most of these hernias are repaired when quite small because they are painful and the patient seeks help early in their development. However they can attain large size with marked displacement of the corresponding rectus muscle. We have recently repaired such a hernia which measured 10 cm. in diameter at the level of the aponeurotic defect. The hernial sac contained the transverse colon, omentum, gallbladder and several loops of small intestine.

Most epigastric hernias occur either roughly half the distance between the xiphoid process and the umbilicus (Fig. 16 a) or immediately above the umbilicus. It is important to inspect the linea alba just above the umbilicus when repairing an umbilical hernia because, occasionally, one will find an incipient epigastric hernia in this location. It is also important in repairing these hernias to examine carefully the linea alba within the limits of the incision because

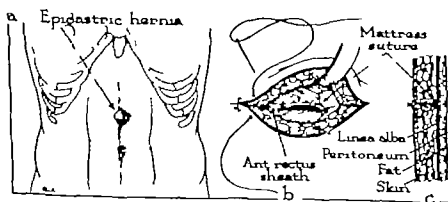


Figure 16. Epigastric hernioplasty a, Appearance of an epigastric hernia. b, Repair of the aponeurotic defect by simple transverse linear closure. c, Sagittal section showing repair of the several layers.

closure is under less tension and should have a lower incidence of recurrent herniation. The umbilical skin is then inverted and the apex of the cone is sutured to the linea alba (Fig. 15 b). The subcutaneous fascia is next closed so that dead space is obliterated around the new umbilicus (Fig. 15 c). In applying the dressing, a stent of fine-mesh gauze or silver foil is packed into the umbilical funnel so that a normal appearing umbilicus is present after the wound has healed. It is important in children to preserve the umbilicus for in general, children are quick to observe anatomic aberrations and they just as quickly ask questions or make derogatory comments.

The only suitable treatment for an adult umbilical hernia is surgical repair. Although an occasional patient can wear a truss with a soft cushion if the hernia is reducible in general a truss should not be worn, first, because it will not stay in place and, second, because the pressure is painful and dangerous. The small variety of adult umbilical hernia is repaired in exactly the same manner as the infantile umbilical hernia (Fig. 15 b). The large umbilical hernia presents some technical problems because to gain mobility of the rigid aponeurotic flaps, it is necessary to cut into the anterior and posterior rectus sheaths on either side. The Mayo overlapping (vest-over pants) closure (Fig. 15 c) is better than simple linear closure for the large umbilical hernia. An umbilical dimple should also be reconstructed in the adult.

EPIGASTRIC HERNIA

Definition. Strictly speaking, any hernia which occurs in the topographic designation of the epigastrium would be an epigastric hernia. However for practical purposes, this is a hernia of the linea alba anywhere between the xiphoid process and the umbilicus. Except for incisional hernias in the epigastric region hernias other than those in the linea alba are an extreme rarity.

Etiology. As in most other hernias, the cause of this hernia is a combination of a congenital aponeurotic defect and an acquired element associated with increased intra-abdominal pressure and muscular effort. Although complete proof is lacking, it is believed that these hernias have their origin in the apertures of perforating blood vessels and that, in this type of hernia, the increased intra abdominal pressure is due to muscular effort rather than to sources of increased pressure within the abdominal cavity.

Incidence. This is one of the least common of the parietal abdominal hernias, accounting for only 1.6 per cent in an average series of hernioplasties (Table 1). It is found almost entirely in the middle-aged male who is doing manual labor.

Anatomy. These hernias occur almost entirely in the linea alba between the xiphoid process and the umbilicus. Rarely a hernia is seen in the linea alba just below the umbilicus and, while this hernia occurs in the linea alba, it can hardly be designated as epigastric hernia because of its topographic location. By common usage however a hernia in the linea alba just above the umbilicus is called an epigastric hernia even though it lies within the topographic limits of the umbilical region. Hernias through the rectus sheath, or the linea semilunaris or the epigastrium, are so rare that they do not warrant discussion.

Surgeons frequently fall into the error of referring to the rectus sheaths as fasciae. While it is true that the laminae of the rectus sheaths are invested by fascial layers, the fasciae are very thin and densely adherent to the main structure of the sheaths which is, of course, the aponeurotic or tendon fibers of the anterolateral abdominal musculature. The fasciae by themselves have very little intrinsic strength and from a practical surgical standpoint serve only to bind the aponeurotic fibers more closely together. If one dissects the innominate fascia off the external oblique muscle toward the midline, it becomes difficult grossly to differentiate the fascia from the epitendineum. This is similarly true with the transversalis fascia on the posterior surface of the abdominal wall. So with the clear understanding that the rectus sheaths are aponeurotic plates, the next step is to realize that the linea alba is also an aponeurotic structure and not just a fusion of fascial layers. It represents the mutual insertion of six flat muscles and, quite understandably it is a very heavy aponeurotic plate. It is much thicker than either rectus sheath and mathematically should be four times as thick and strong as one layer of the rectus sheath. Because of the different course of the musculoaponeurotic fibers of the external oblique, the internal oblique and the transversus abdominis muscles, there is considerable criss-crossing and actual interdigitation of aponeurotic fibers within the laminae of the rectus sheaths. While there is considerable variation from level to level and between the anterior and the posterior rectus sheath in the degree of this in

It is this

aponeurotic fibers of the posterior rectus sheath change over into the anterior rectus sheath. Weakness in this angle as evidenced by separation of the aponeurotic fibers, was repeatedly observed and especially in those cadavers in which the change-over occurred at several levels so as to give, in effect, two and sometimes three margins that could be termed *linea semicircularis*. A protrusion of preperitoneal fat between the aponeurotic fibers of the transversus abdominis (*hernia adiposa*) due to an aponeurotic defect is the etiologic basis of this hernia whatever the contributing causes may be such as increased intra-abdominal pressure and degenerative changes. Perforating blood vessels in this angle have not been observed although a small vein which runs parallel to the lateral border of the rectus abdominis muscle is a constant finding at this level. Small protrusions of preperitoneal fat through the transversus abdominis aponeurosis are occasionally seen above the area of a direct inguinal hernia and so this region should be carefully inspected whenever a direct inguinal hernia is being repaired. Their simple reduction and closure of the aponeurotic defects with fine silk sutures seem to be sufficient. Should the hernial defect occur below the point at which the inferior epigastric vessels cross the *linea semilunaris* it must be termed a diverticular direct inguinal hernia. The variation in the position of the *linea semicircularis* allows considerable variation in the vertical location of this hernia.

From the few Spiegel's hernias we have observed, the presenting mass is preperitoneal fat and it remains for a long time as a vague and slightly tender lump in the interstitial position beneath the external oblique aponeurosis. Fully developed hernial sacs with incarcerated contents have, of course, been reported, but in our four subjects the hernial mass consisted of preperitoneal fat and a small empty hernial sac. The repair of this hernia, after the fat and the peritoneal sac are disposed of in a conventional manner is of the greatest simplicity. The transversus abdominis aponeurotic fibers, along with the overlying and interdigitating internal oblique aponeurotic fibers, are closed transversely. The overlying external oblique aponeurosis is closed in the oblique direction of its fibers.

LUMBAR HERNIAS

Two types of lumbar hernias have been described—one through the inferior lumbar

triangle of Petit and the other through the superior lumbar triangle of Grynfelt.

The inferior lumbar triangle of Petit is bounded by the superior margin of the posterior crest of the ilium, the anterior border of the latissimus dorsi muscle and the posterior border of the external oblique muscle. The roof of the triangle is the continuity of the muscle fascia and the superimposed subcutaneous fascia. The floor of the triangle consists of the internal oblique muscle fibers originating from the iliac crest and beneath, the similar fibers of origin of the transversus abdominis muscle. An inferior lumbar triangle of variable size is present in three-fourths of dissected cadavers. Therefore, the presence of this triangle does not constitute a predilection for the development of a lumbar hernia, although an inferior lumbar hernia must of necessity present through this triangle. Fundamentally this hernia must be caused by a defect in the transversus abdominis layer first with subsequent changes taking place in the overlying musculofascial layers. We have noted the absence of segments of the transversus abdominis muscle immediately above the iliac crest, but with the continuity of the layer maintained by the transversalis fascia and supported strongly by the overlying internal oblique muscle layer. While segmental congenital absence of layers undoubtedly accounts for an inferior lumbar hernia present at birth, and a deficient muscular floor in the triangle could explain the appearance of the hernia in an adult, a constant vascular lacuna at the site of the development of this hernia might also explain its origin. The rarity of the hernia, however, attests to the competency of this foramen.

The iliohypogastric nerve and the fourth lumbar artery and vein perforate the transversus abdominis muscle just above the iliac crest and lateral to the quadratus lumborum muscle (Fig. 17) at the point where inferior lumbar hernias occur. It is possible that this hernia begins with the protrusion of retroperitoneal fat into the neurovascular foramen in the same manner of origin as that for some of the epigastric hernias.

The superior lumbar triangle of Grynfelt is bounded by the inferior border of the twelfth rib, the lateral border of the sacrospinalis muscle and the posterior border of the internal oblique muscle. The roof of the triangle is the latissimus dorsi muscle and the floor the transversus abdominis muscle. As in the inferior lumbar triangle, the dimensions of this triangle vary considerably but

small defects with *hernia adiposa* are frequently found adjacent to the main hernial defect. This is especially true in patients in the older age groups in whom fatty infiltration or replacement affects the *linea alba* as it does many other parts of the body.

Diagnosis. The diagnosis of a fully developed epigastric hernia is easy but these hernias give symptoms early in their development and can be very difficult to feel, especially in the obese patient. At the stage when the hernia is only a 3- or 4-mm. *hernia adiposa*, it may give sharply localized tenderness and pain and a referred pain that can mimic that due to duodenal ulcer or gallbladder disease. The sharp localization of tenderness in the contracted abdominal wall helps to differentiate this condition from more deeply placed visceral disease. When the hernia enlarges to the point that there is a peritoneal sac containing viscera, then other symptoms appear associated with omental traction or partial intestinal obstruction just as in any other parietal hernia. The outstanding characteristic of this hernia is the *symptomatology* which is out of all proportion to its size. The tiny aperture of the early hernia is even more tightly squeezed by muscular contraction, which in turn makes the decussating aponeurotic fibers about the aperture more taut. Parietal somatic nerves and the sympathetic fibers accompanying the blood vessels are tightly compressed and explain the local and referred pain.

Treatment. The only treatment for the epigastric hernia is surgical. The pressure of any type of truss is intolerable because most of these hernias are irreducible and tender. A transverse incision is made over the hernial mass and the subcutaneous fascia is dissected off the hernia and the surrounding *linea alba*. The hernia is usually tightly incarcerated so that the *linea alba* must be incised on one or both sides to accomplish reduction. A small lobule of fat may be simply pushed in, but larger lobules should be inspected for a peritoneal sac. If a sac is present, it should be opened to rule out the possibility of incarcerated omentum or viscera. Pedunculated processes of fat should be ligated at their base before excision because of the blood vessels. As a rule a defect that is no broader than the *linea alba* can be repaired by simple linear closure in the transverse plane (Fig. 18 b). The method of closure or the type of suture material is of less importance than tight closure in preventing fat lobules from pushing out

between the sutures in the postoperative period, because this will inevitably manifest itself by recurrent epigastric herniation. Epigastric hernias, with an aponeurotic defect larger than the width of the *linea alba*, which extend into one or both rectus sheaths, present technical problems in repair. The surgical principle that the subcutaneous fascia should be carefully closed to avoid leaving a dead space applies to any incision and is simply illustrated in Figure 18 c.

Two features in the repair of this hernia are worthy of emphasis. First is the careful search for adjacent incipient epigastric hernias which may be separately repaired or included in the main closure when possible. The sutures should be carefully placed so that a suture hole does not become the site of a recurrent epigastric hernia. The second important feature is closure of the defect in the transverse plane. Without belaboring the point, enough has been said to present the realization that the direction of musculo-aponeurotic pull in the *linea alba* is transverse and that a closure in the vertical plane is subject to powerful disruptive forces.

SEMILUNAR (SPIEGEL'S) HERNIA

The extreme variation in the position and formation of the *linea semicircularis*, along with variation in the structural strength of the *transversus abdominis* aponeurosis, is the key to the development of this rare type of hernia. Variations in the internal oblique and external oblique layers while common, are of lesser importance. In reviewing statistical data obtained from observations upon the structure of the anterior abdominal wall in 250 cadavers it is amazing that clinical evidence of herniation through or near the *linea semilunaris* in the lower abdomen is not common. It is possible, since most cadavers are the bodies of aged persons, that these defects in aponeurotic structure represent a degenerative process. It is also likely that protrusions of preperitoneal fat through the aponeurotic fibers of the *transversus abdominis* layer are very slow in their development into discrete hernias.

Although hernias in the *linea semilunaris* above the level of the *linea semicircularis* have been described, aponeurotic deficiency at this level has not been observed, on the contrary, this line of aponeurotic fusion is extremely dense and strong. They most represent hernias through blood vessel foramina (Fig. 13). The angle where the *linea semicircularis* joins the *linea semilunaris* is the point at which the *transversus abdominis*

Every surgeon has had the disquieting experience of seeing a hernia develop in some of his surgical wounds and even though it is due to circumstances beyond his control it is a tragedy for the patient. The surgeon with a thorough knowledge of the anatomy of the abdominal wall who understands the importance of the transversus abdominis layer and makes his incisions accordingly and practices sound principles of surgical technique, will only occasionally be confronted with this difficult problem.

We favor the transverse abdominal incision, which preserves the greatest number of musculoaponeurotic fibers since this incision carries the lowest incidence of incisional herniation. In certain patients the additional exposure afforded by a long vertical rectus incision makes the increased chances of the development of an incisional hernia a justified risk. In the hands of a competent surgeon and in a slender patient with healthy tissues, the vertical wound heals well with a low incidence of herniation. However the surgeon today is faced with the problem of many seriously ill patients with various degrees of malnutrition whose wounds heal poorly under optimum conditions. In this group, and in the obese with their added problems of wound tension and incidence of infection, the transverse incision that carefully preserves the integrity of the transversus abdominis layer carries a much lower incidence of wound hernia.

Every surgeon should be familiar with the direction of the fibers of the external oblique, internal oblique and transversus abdominis muscles. The anterior and posterior rectus sheaths represent an intermingling and interdigitation of the aponeurotic fibers of insertion of these three muscles as they approach a mutual insertion in the linea alba. The interdigitation of these aponeurotic fibers within the rectus sheaths becomes so complex at times as to suggest the warp and woof of a carpet. The weave is further strengthened by the epitendineae and the fascial laminae, so that at a casual glance the rectus sheath appears to be a dense tightly woven structure which can be cut in any direction with impunity. However incisions in the rectus sheath should take into consideration the lines of force or musculoaponeurotic pull.

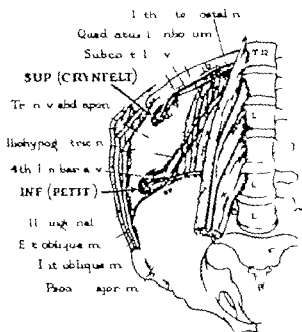
Above the level of the linea semicircularis the aponeurotic fibers of the transversus abdominis and internal oblique muscles in the posterior rectus sheath have a

predominantly transverse course toward their insertion in the linea alba. As the costal margin is approached these fibers course in a slightly oblique direction cephalad as they pass from the lateral edge of the sheath to the linea alba. The number of internal oblique aponeurotic fibers gradually decreases until in the upper epigastrium the posterior rectus sheath consists entirely of the transversus abdominis musculoaponeurotic fibers. Therefore in the posterior rectus sheath (Fig. 18 a and b) there can be no question about the direction that a surgical incision should take. A cholecystectomy incision (Fig. 18 b) in the transverse plane can extend from the linea alba to the costal margin by simply separating parallel musculoaponeurotic fibers in the posterior rectus sheath. In a gastrectomy incision these fibers can be separated from one costal margin to the other. If the linea alba and the posterior rectus sheath or sheaths are carefully approximated in the wound closure the patient has in effect a normal posterior layer for the abdominal wall, since no musculoaponeurotic fibers have been cut. As a matter of fact, the contraction of the transversus muscle in the postoperative period only tends to approximate more tightly the aponeurotic fibers which have been separated during the operation.

The musculoaponeurotic dynamics in the anterior rectus sheath are not quite so simple. The external oblique aponeurotic fibers course obliquely downward and the aponeurotic fibers of the internal oblique in the anterior rectus sheath course obliquely upward. As their respective aponeurotic fibers enter into the formation of the anterior rectus sheath, there is an intimate intermingling and interdigitation. Because these aponeurotic fibers are rather firmly united, they act as a unit. The predominant force of their combined pull is still in the transverse plane, although some fibers of the external oblique will tend to pull the superior margin of the incision cephalad and some fibers of the internal oblique will tend to pull the inferior margin of the incision caudad. While a transverse incision in the anterior rectus sheath is not ideal because aponeurotic fibers are cut, it is the best available direction for cutting the anterior rectus sheath.

The ideal abdominal incision is the small muscle- and aponeurosis-splitting right lower quadrant incision used for an appendectomy. The external oblique aponeurosis is divided obliquely in the direction of its fibers and the edges are retracted. At this

LUMBAR HERNIAS



Figures 17 Anterior view of the right posterior abdominal wall to show the points of origin of the superior and inferior lumbar hernias. The peritoneum, retroperitoneal connective tissue and the transversalis fascial layer have been removed. The course of the nerves and vessels illustrated represents one theory of the etiologic basis of these hernias

the bulwark against herniation is the integrity of the transversus abdominis musculospondyrotic layer. It is interesting to observe that the subcostal artery and vein and the twelfth intercostal nerve perforate the transversus layer in the floor of this triangle (Fig. 17) at the point of origin described for superior lumbar hernia. Again, it is suggestive evidence that a neurovascular lacuna is the origin of this hernia. Whatever technique of hernia repair is used, reconstruction of the transversus abdominis layer should be the primary objective.

INCISIONAL HERNIA

Definition. An incisional hernia is one which develops in the scar of a surgical incision.

Etiology Irrespective of the many contributing causes to the development of an incisional hernia, it is fundamentally a failure of the approximated musculoaponeurotic and fascial layers to remain in apposition.

Wound infection is probably still the most important factor in the development of an incisional hernia. Suppurative necrosis destroys the approximated margins of the aponeuroticofascial layers irrespective of the type of suture material used, and if catgut suture has been used, it undergoes dissolu-

tion and fragmentation at a very rapid rate.

Drains through the abdominal wall greatly increase the incidence of incisional hernia, as evidenced by the number of these hernias that develop at drain sites. This is due to incomplete closure of the wound plus the added element of infection. Other causes of incisional hernia may be grouped under several headings:

Faulty wound technique includes a host of errors of both omission and commission. The most important include inadequate hemostasis, avascular necrosis of the flaps due to sutures too tightly tied, faulty closure of the posterior rectus sheath or transversus abdominis layer, rough handling of tissue and wound contamination from several sources.

The *preoperative status* of the patient, which includes obesity, malnutrition, hypoproteinemia, vitamin deficiency (especially vitamin C) and tissue edema, may be a

The postoperative status of the patient, which includes abdominal distention, chronic cough and inadequate nutrition, may play an important role.

The type of incision also has some bearing upon the incidence of incisional herniation. All other factors being equal, there is not only a higher incidence of herniation through vertical incisions than there is through transverse muscle-splitting incisions, but hernias through vertical surgical scars are more difficult to repair than those through transverse scars.

Incidence. It is impossible to ascertain the true incidence of incisional herniation because no long-term follow-up studies are available on a large series of cases. However the percentage of incisional hernioplasties in recent series of consecutive hernioplasties would indicate that the incidence of this hernia is increasing (Table 1). This percentage of course is no index of the incidence of incisional hernia after celiotomy. It is also true that the number of operations performed is increasing year by year. Whatever the true incidence of incisional hernia may be as related to the total number of celiotomies, it is distressingly high when one considers the almost innumerable difficulties encountered in the repair of many of them. The surgeon should thoughtfully consider the fate of his surgical incision as he dramatically cures the patient of some fatal abdominal disease.

Anatomy The pathologic anatomy of the incisional hernia is a story of local failure

the integrity of the more superficial musculoaponeurotic layers. On the other hand if the transversus abdominis layer remains intact, a hernia will not develop even though the more superficial musculoaponeurotic layers are imperfectly approximated or subsequently become separated in the postoperative period. The rectus abdominis muscle can be sectioned within its sheath with impunity. If the epigastric blood vessels which course within the muscle substance are ligated to avoid a hematoma the severed rectus abdominis muscle heals as an auxiliary tendinous inscription.

The vertical rectus incision is by far the most common celiotomy incision in use today. It is an incision that is easily and rapidly made with a minimum of time spent in ligating blood vessels. The exposure is excellent when the incision is long enough and it is rapidly closed with a continuous catgut suture. When speed and wide exposure are needed, it is the incision of choice. However as an example of wide exposure using the transverse incisions it can be pointed out that a total colectomy and proctosigmoidectomy can be very nicely accomplished through a combination of upper and lower abdominal transverse incisions. This combined approach adds very little to the total operating time, gives excellent exposure from pelvis to subdiaphragmatic spaces and leaves the patient with a strong abdominal wall. A commonly cited advantage of the vertical incision is that it can easily be extended, either up or down, in the event that the pathologic process is not accessible through the original incision. One of the more common incisional hernias is in the vertical cholecystectomy incision which was converted into an appendectomy incision by caudal extension or in the appendectomy incision extended upward to include a cholecystectomy incision. This long tendon-severing incision, plus the high incidence of suppuration in the gallbladder and in the appendix, accounts for the excessive number of incisional hernias which occur in the right rectus incision. The advocates of "exploratory laparotomy" naturally favor the vertical incision because of the ease with which it can be extended. After careful study of the patient, a definite diagnosis should be made along with an appropriate transverse incision. Should the diagnosis and the surgical approach prove to be in error another transverse incision should be made at the proper site.

The vertical rectus incision cuts all of the

aponeurotic fibers in the rectus sheath for the length of the incision. As soon as the patient recovers from the anesthetic the contraction of the muscle fibers whose aponeurotic fibers have been cut puts tremendous tension upon the line of wound closure. By letting the patient come out of the anesthetic until he can close the glottis and strain we have found the power of the muscle pull to be as high as 40 pounds in a well-developed male. When a transverse incision is used the divided fibers of the transversus abdominis snap together and can be sutured without any tension. This of course, does not apply to the anterior sheath which is composed of internal oblique and external oblique fibers and a pull of up to 12 pounds has been necessary to approximate the anterior sheath with the glottis closed. However it is the transversus abdominis layer which is of the greatest importance in the prevention of wound hernia.

Figure 18 was prepared to emphasize some of the foregoing points. An incisional hernia in a vertical cholecystectomy incision (Fig. 18 a) is presented for comparison with an incisional hernia in a transverse cholecystectomy incision (Fig. 18 b). While the two hernias are not comparable in size, they do represent our average findings in repairing incisional hernias in cholecystectomy scars. We have no way of estimating the incidence of incisional hernia in the vertical cholecystectomy incision since we have never used this incision. However in over 400 cholecystectomies in which the transverse incision was used, we have had two incisional hernias develop in the lateral pole of the wound at the former drain site with a defect as shown in Figure 18 b. During the same length of time, we have repaired thirty-four incisional hernias in vertical cholecystectomy scars and observed an unrecorded large number of badly attenuated scars that are thin and bulging but without a discrete aperture that can be designated as a hernia. By comparing these two figures the myodynamics in the development of each hernia should be readily apparent. Whatever the original cause for the failure of the wound edges to stay in apposition (Fig. 18 a) the most important contributing cause is the muscular pull which can only be shown for the transversus abdominis musculoaponeurotic apparatus in this posterior view. The heavy arrows indicate the direction of muscular pull which is at right angles to the incision and is therefore maximal. One must take into consideration the

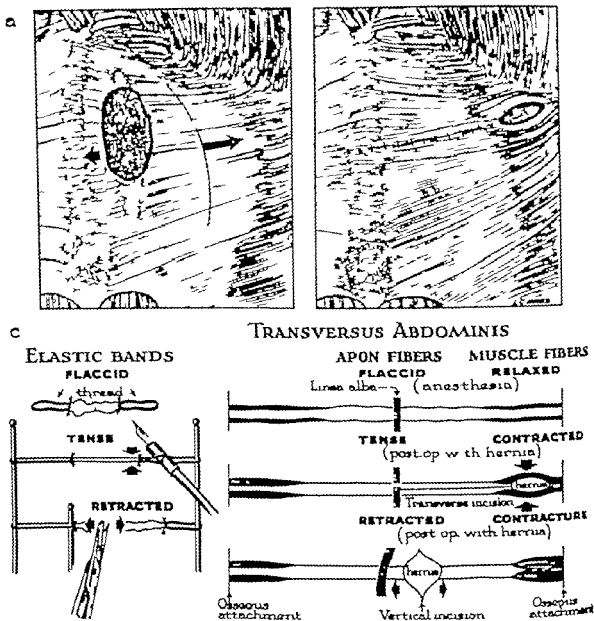


Figure 18. Incisional hernias. a, Posterior view of the anterior abdominal wall showing the sponenotic defect of a large incisional hernia in a vertical right rectus cholecystectomy incision. b, Posterior view of the anterior abdominal wall showing the usual musculoaponeurotic defect of an incisional hernia in a right transverse cholecystectomy incision. c and d, The thread and rubber band analogy between the transverse separation and the vertical section of the aponeurotic fibers of the transversus abdominis muscle.

level, the transversus abdominis and internal oblique musculoaponeurotic layers have an identical course and so these fibers are separated parallel with their course which is in the transverse plane. This incision, properly made, does not cut a single musculoaponeurotic fiber. If the transversus abdominis and its fascia, the transversalis fascia, are carefully closed so that preperitoneal fat cannot push out between the fibers this wound is as strong after the operation as a normal abdominal wall. The incision should never result in incisional herniation unless it has been necessary to leave a drain through the

abdominal wall. The larger transverse abdominal incision is a compromise to adequate exposure. More accurately it is a compromise only in regard to the anterior rectus sheath, for the posterior rectus sheath and the transversus abdominis muscle fibers can be separated from one side of the body to the other without cutting tendon fibers. It is worth re-emphasizing at this point that the transversus abdominis musculoaponeurotic layer is the most important bulwark against the development of an incisional hernia. If a defect develops in the transversus layer a hernia is born irrespective of

Diagnosis. The diagnosis of a fully developed incisional hernia presents no difficulties because it is obvious to the patient and the physician alike. However small or incipient incisional hernias may be difficult to feel in the surrounding scar and this is especially true in the obese patient. Until a defect can be felt with the examining finger along with an impulse on coughing, one cannot definitely diagnose an incisional hernia. Many surgical scars are tender for a long time after the operation with episodes of sharply localized pain and tenderness following strenuous muscular effort. These latter episodes probably represent tearing of scar tissue with small areas of hemorrhage and the acute symptoms will subside in a week or two. Broad and attenuated vertical abdominal scars are a common finding, but they do not constitute a true hernia unless there is a circumscribed defect through the scar. The surgeon should not contemplate repairing thinned-out scars unless it becomes necessary to operate upon the patient for adhesive intestinal obstruction, because there is no assurance that a better end result can be obtained.

It is difficult to describe the symptoms of an incisional hernia. An objectively severe incisional hernia may cause only the vaguest of symptoms, while an apparently insignificant defect may be very painful and be associated with the whole gamut of symptoms which accompany partial or complete intestinal obstruction. In general, the symptoms of an incisional hernia are mild, considering the rigidity of the defect and size of the hernial protrusion. These hernias are more often incarcerated than reducible and only a small percentage of them become strangulated. They gradually increase in size and become more difficult to repair with the passage of time; therefore nothing but increasing difficulty is to be gained by procrastination.

Incisional hernias frequently present multiple defects with septa separating the apertures. These septa represent portions of the wound closure that have held together. In addition to the aponeurotic defects, which represent separate hernial rings, the omentum that is invariably present and incarcerated within the hernial sac will make additional secondary hernial rings which account for the fact that these hernias are so frequently irreducible. It is also common to find a portion of the hernia regularly reducible while other segments cannot be reduced into the abdominal cavity. Frequent

findings at the time of operation are unsuspected interstitial components of the hernia which have dissected between the rectus sheath. Incisional hernias may attain enormous size and contain all of the viscera with a mesentery plus some of the so-called fixed viscera as components of a sliding hernia. While these patients are usually confined to a sedentary life, they are usually free of symptoms of intestinal obstruction.

The hernial sac of an incisional hernia which is fully developed lies immediately beneath the skin so that peristaltic movement can be plainly seen. At times the skin is so tightly stretched that it appears about to break. Trophic changes in the skin over a large incisional hernia are common along with ulcers that are due to simple avascular necrosis.

Treatment. The repair of an incisional hernia is a complex problem. This hernia is a perfect example of the old aphorism that an ounce of prevention is worth a pound of cure. Once a large incisional hernia has developed in a vertical rectus incision all methods of surgical repair are at best only makeshift and the incidence of recurrent herniation is appalling. The most important single feature in the prevention of an incisional hernia is the careful preservation of the integrity of the transversus abdominis muscle and its aponeurosis. This is accomplished by separating the musculoaponeurotic fibers in the transverse plane and not by cutting them in the vertical plane.

Trusses of the usual spring variety with a ball or cushion that presses against the hernial defect, cannot be used in incisional hernias which are incarcerated and cannot even be kept in place for the occasional reducible incisional hernia with a single discrete defect. An elastic support, such as a two-way stretch girdle, will afford considerable relief in some instances while in others even very slight pressure increases the discomfort. For very large incisional hernias, a cloth and elastic support which is in effect a sling or hammock can be worn with some amelioration of symptoms because it supports the weight of the hernia and reduces the traction upon the mesentery of the contained viscera.

In general, a small incisional hernia should be repaired by developing the layers of the three anterolateral abdominal musculoaponeurotic laminae and approximating each in the direction of its fibers, with special attention to the reconstruction of a firm transversus abdominis layer. When this is

muscle pull from the opposite side as well as the muscle pull on the side of the hernia. In the fully developed vertical incisional hernia of this size, two inches or more, almost adequate repair by reapproximating the edges of the hernial orifice in the vertical plane. First is the lateral resection of the aponeurotic fibers on the side of the operation with the conception of a new construction of the corresponding muscle sheet. This construction is described on the outside that the myoelectrostatic. The hernia has receded laterally. A similar but lesser, recurrent trans place in the contralateral side. Because of this construction the margins of such a defect can only be approximated in the vertical plane with extreme tension and with the aid of surgical assistance. It is not surprising that such a hernia repair is associated with an alarming recurrence rate. In such a hernia it is impossible to close the defect in the transverse plane and so the surgeon is left with several alternatives in the way of using materials to strengthen the closure of the abdominal wall, but none of them is entirely satisfactory.

By showing the incisional hernia in the transverse cross-section (Fig. 13 b) it can be seen that the problem is simply one of approximating myoelectrostatic fibers that have been laid apart by a crani and independent to the removal of the crani by the protruding mass. What it can not be fair to compare a small hernia in the lateral plane or a transverse incision to a large hernia in a vertical incision. These are the hernias which we have repaired in cholecystectomy scars. They serve to emphasize the magnitude of the problem in the transverse scar and the magnitude of the problem in the vertical scar, as well as the relative frequency of occurrence in the two scars. Therefore vertical incisional hernias is compared with two transverse incisional hernias. In our own cases we have had three large incisional hernias in transverse incisions for cholecystectomy that were one to massive wound infection. These were quite easily repaired without excessive tension on the suture line or simple approximating the used aponeurotic edges in the transverse plane again.

For a number of years we have used a very simple way of to stress the importance of preventing the myoelectrostatic structure of the transverse incisions after an using cellulose acetate. The two second rubber bands connected by two threads

Fig. 13 c represent myoelectrostatic fibers of the transverse abdominal wall placed under surgical tension. Fig. 13 c. When the rubber bands are made use by stretching the threads against the fibers and the curves of the rubber and elastic sheet, snap together to form parallel straight lines as the suture lines between two fixed points. The cords in the muscles. This corresponds to the same muscle contraction in the postoperative period but is of no consequence if the fibers have been separated in the transverse plane. When a crani is supported between a muscle sheet represented by a connective

Fig. 13 c. the displaced muscle sheet over a force that separates the crani. The Panniculus crani is removed within a few days so the defect snaps shut. However, if a large crani is used, it is not in part of any with the membrane in crani or if a large a space has been set around the crani so that constant sides in inside it and a hernia can develop. Fig. 13 d. It will be apparent that the repair of such a hernia is very simple. When the threads are

Fig. 13 e. the transverse incision crani and pull the severed ends of the wound apart. It will take some time to be the threads together again and even so the severed rubber bands continue to exert a pull upon the crani. This is analogous to the vertical incision when one of the aponeurotic fibers of the transverse abdominal muscle in the posterior rectus abdominis sutures to the posterior rectus abdominis and to such an incisional hernia again.

Fig. 13 f. There is another variation of the crani in muscle pull and on the one the patient feels that with his hernia or muscle fibers have become strengthened and represent a muscle connection. Fig. 13 d. Reapproximating the severed ends of the hernial defect always produces more tension than can be present at the time of an original wound closure. It is one wonder that the incidence of recurrent hernias is high.

Finally a bit more notes about the crani. It is not as accurate or as secure as a suture line is a wound tendon and can be the suture line be repaired, as it always come into standing tension in the wrist. Furthermore the suture tend to pull out from between the aponeurotic fibers of the rectus abdominis. The demarcation of the aponeurotic fibers and the adhesive qualities of the investing fascia are the two factors which prevent this from happening on.

Diagnosis. The diagnosis of a fully developed incisional hernia presents no difficulties because it is obvious to the patient and the physician alike. However small or incipient incisional hernias may be difficult to feel in the surrounding scar and this is especially true in the obese patient. Until a defect can be felt with the examining finger along with an impulse on coughing, one cannot definitely diagnose an incisional hernia. Many surgical scars are tender for a long time after the operation with episodes of sharply localized pain and tenderness following strenuous muscular effort. These latter episodes probably represent tearing of scar tissue with small areas of hemorrhage and the acute symptoms will subside in a week or two. Broad and attenuated vertical abdominal scars are a common finding, but they do not constitute a true hernia unless there is a circumscribed defect through the scar. The surgeon should not contemplate repairing thinned-out scars unless it becomes necessary to operate upon the patient for adhesive intestinal obstruction, because there is no assurance that a better end result can be obtained.

It is difficult to describe the symptoms of an incisional hernia. An objectively severe incisional hernia may cause only the vaguest of symptoms, while an apparently insignificant defect may be very painful and be associated with the whole gamut of symptoms which accompany partial or complete intestinal obstruction. In general the symptoms of an incisional hernia are mild, considering the rigidity of the defect and size of the hernial protrusion. These hernias are more often incarcerated than reducible and only a small percentage of them become strangulated. They gradually increase in size and become more difficult to repair with the passage of time; therefore nothing but increasing difficulty is to be gained by procrastination.

Incisional hernias frequently present multiple defects with septa separating the apertures. These septa represent portions of the wound closure that have held together. In addition to the aponeurotic defects which represent separate hernial rings the omentum that is invariably present and incarcerated within the hernial sac will make additional secondary hernial rings which account for the fact that these hernias are so frequently irreducible. It is also common to find a portion of the hernia regularly reducible while other segments cannot be reduced into the abdominal cavity. Frequent

findings at the time of operation are unsuspected interstitial components of the hernia which have dissected between the rectus sheath. Incisional hernias may attain enormous size and contain all of the viscera with a mesentery plus some of the so-called fixed viscera as components of a sliding hernia. While these patients are usually confined to a sedentary life, they are usually free of symptoms of intestinal obstruction.

The hernial sac of an incisional hernia which is fully developed lies immediately beneath the skin so that peristaltic movement can be plainly seen. At times the skin is so tightly stretched that it appears about to break. Trophic changes in the skin over a large incisional hernia are common along with ulcers that are due to simple avascular necrosis.

Treatment. The repair of an incisional hernia is a complex problem. This hernia is a perfect example of the old aphorism that an ounce of prevention is worth a pound of cure. Once a large incisional hernia has developed in a vertical rectus incision, all methods of surgical repair are at best only makeshift and the incidence of recurrent herniation is appalling. The most important single feature in the prevention of an incisional hernia is the careful preservation of the integrity of the transversus abdominis muscle and its aponeurosis. This is accomplished by separating the musculoaponeurotic fibers in the transverse plane and not by cutting them in the vertical plane.

Trusses of the usual spring variety with a ball or cushion that presses against the hernial defect, cannot be used in incisional hernias which are incarcerated and cannot even be kept in place for the occasional reducible incisional hernia with a single discrete defect. An elastic support, such as a two-way stretch girdle, will afford considerable relief in some instances while in others even very slight pressure increases the discomfort. For very large incisional hernias, a cloth and elastic support which is in effect a sling or hammock can be worn with some amelioration of symptoms because it supports the weight of the hernia and reduces the traction upon the mesentery of the contained viscera.

In general, a small incisional hernia should be repaired by developing the layers of the three anterolateral abdominal musculoaponeurotic laminae and approximating each in the direction of its fibers, with special attention to the reconstruction of a firm transversus abdominis layer. When this is

impossible, the fused layers should be approximated in the transverse plane with an overlapping closure similar to the Mayo operation for large umbilical hernias. When tension precludes such a vest-over pants closure, simple linear closure with less tension is the better procedure.

Unfortunately long vertical oval defects are commonly seen and for these there is no good solution. They cannot be approximated in the transverse plane and to reapproximate the edges forcibly in the vertical plane carries a very high incidence of recurrent herniation. It is for this reason that various prosthetic devices have been tried to strengthen the line of closure or to fill in the gap when the edges cannot be approximated. Without going into detail, some of the devices that have been used are broad homologous sutures of fascia lata, aponeuroticofascial flaps either turned with their blood supply preserved or transplanted from another part of the body, patches of ox fascia, patches of plastic cloth full-thickness or split thickness patches of skin and sheets of metallic wire mesh. None of them is a complete answer to the problem and all of them, with the exception of the musculoaponeurotic flap with its blood supply intact, eventually result in a layer of scar tissue as the sole bulwark against recurrent herniation. Scar tissue slowly stretches and the broader the amount of scar tissue in a wound, the greater will be the eventual attenuation of the hernial repair. While it is ideal to turn a musculoaponeurotic flap with its blood supply intact in the repair of a midabdominal defect, the technical problems are insurmountable.

Through the years, we have tried most of the described prosthetic devices, but recently we have relied entirely upon the wire mesh advocated by Koontz as being the simplest and most effective. At first we used the tantalum mesh, but lately we have used stainless steel wire mesh because it is less brittle and more easily handled in the wound. Stainless steel suture material is much easier to use than tantalum and, although the galvanic action of different metals in the same wound might not be important, we have never tried the use of more than one in a wound.

The fate of the wire mesh and the eventual strength of the wound have been interesting to observe. All metal has a fatigue point at which it breaks and the movement of the abdominal wall in breathing and bending eventually fragments the

wire mesh rather completely (Figs. 19 and 20). Although the wire mesh acts as a firm support in the first few months after the operation, the eventual support is only scar tissue. What happens after the wire mesh fragments is largely dependent upon the habits and habits of the patient. As an example the case histories of two patients will be compared.

The first patient (Fig. 19) had a cholecystectomy and appendectomy in 1933 through a long vertical rectus incision. Within a few months after the operation, he noticed a lump in the incision which gradually enlarged through the years. When first seen by us in 1951 he had a strangulated incisional hernia the size of a football with an aponeurotic defect that measured 10 cm. in diameter. He was obese (225 pounds) and the edges of the defect were approximated with considerable tension. A sheet of stainless steel wire mesh was sutured in place over the suture line. Following this operation, he seemed to have a very strong abdominal wall, but against our advice he remained obese, physically active and would not wear an elastic girdle. He was not seen from 1952 until 1955 when he returned with an incisional hernia through the fragmented

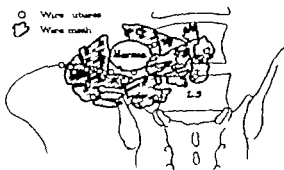


Figure 19 A sketch of an x-ray plate to show a recurrent incisional hernia through the fractured fragments of wire mesh.

wire mesh (Fig. 19) that was irreducible and with partial intestinal obstruction. It should be noted that the scar which incorporated the fragments of the wire mesh was exceedingly tough and yet a hernia had occurred through this scar. The patient was still obese and now had moderately severe myocardial damage. The margins of the hernia were defined after a difficult dissection, the hernia reduced and the dense margins including fragments of wire mesh, were simply approximated with wire sutures in the transverse plane.

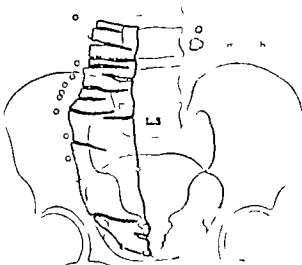


Figure 20 A sketch of an x ray plate to show the fractures in a large sheet of wire mesh that was used to repair the defect in a very large incisional hernia.

The second patient (Fig. 20) had a cholecystectomy and appendectomy in 1928 through an incision that extended vertically from the right costal margin to within 4 cm. of the pubic tubercle. When first seen in 1951 he had an enormous incisional hernia with recurrent episodes of intestinal obstruction. This patient was slender (126 pounds) but even so the edges of the defect could not be approximated. The fused flaps of the hernial sac were used to bridge the defect and supported with a large sheet of wire mesh. This patient has remained thin, worn an elastic two-way stretch girdle most of the time and has done no heavy work of any kind. He has continued his trade of repairing shoes. He was called in for an examination in May 1955 at which time the x-ray picture was taken from which the drawing shown in Figure 20 was made. Clinically the repair is solid although the wall is thin and some of the fractures in the wire mesh can be felt.

These two cases reports emphasize the fact that the cooperative slender patient can be given reasonable assurance that the repair of his large incisional hernia will be successful. The obese obdurate patient is doomed to failure of the repair and should not be operated upon unless intestinal obstruction makes it necessary. The obese patient with a large incisional hernia, who will not reduce his weight to normal and will engage in heavy physical work, had better kept his hernia, for the second recurrence will undoubtedly be more troublesome than the first.

HERNIAS OF THE RESPIRATORY DIAPHRAGM

Definition. A diaphragmatic hernia represents the protrusion of an abdominal viscus or viscera into the thoracic cavity through an aperture in the respiratory diaphragm.

Etiology. From an etiologic standpoint the diaphragmatic hernias may be divided into three groups: those due to congenital segmental absence of a portion of the diaphragm; those due to congenital weakness or imperfect fusion of the diaphragmatic segments; those due to traumatic perforations or rents in the diaphragm. The first group represents a defect in the embryologic development of the diaphragm and most commonly the left pleuroperitoneal segment of the diaphragm is involved. These hernias are present at birth and, unless they are very small, produce profound and immediate symptoms. The second group includes those hernias which are due to imperfect fusion of the various embryologic segments of the diaphragm. Esophageal hiatus hernias are also included in this group because most of them are due to an imperfect closure of the esophageal hiatus. Even those hiatus hernias which are due to a congenitally short esophagus have a developmental defect of the hiatus in that this structure fails to close properly because of the thoracoabdominal position of the stomach. Even the adult hiatus hernia probably has a congenital component in its etiologic background. The third group of diaphragmatic hernias includes all acquired hernias which develop after trauma, whether the trauma is due to direct perforation or to indirect force.

Incidence. It is difficult to determine the true incidence of hernias of the respiratory diaphragm. Wide discrepancies in the reported occurrence of this hernia (Table 1) reflect the source of the statistics. Because of the difficulties involved in the diagnosis and especially in the treatment of diaphragmatic hernias, the large medical centers report an actual as well as a relative increase in these hernias during the past twenty years. The relative incidence of the various diaphragmatic hernias will also depend upon the source of the material. Harrington's statistics from the Mayo Clinic show a great preponderance of esophageal hiatus hernias because his group of subjects is largely composed of adults. Gross statistics show a preponderance of hernias through the left pleuroperitoneal space, because his group represents the newborn and the infant por

tion of the population. If one were to attempt to compile statistics from the general population, they would also be in error because of the infants with diaphragmatic hernias who die shortly after birth with a diagnosis of "blue baby" due to a cardiac abnormality and those adults in whom the diagnosis is not established, either because the hernia is asymptomatic or because inadequate diagnostic studies are made. Consecutive hernioplasties will give varying percentages of diaphragmatic hernias which are dependent upon the source of the material. Asymptomatic esophageal hiatus hernias are a common finding in careful routine x-ray examinations of the upper gastrointestinal tract.

Anatomy All but the traumatic hernias have their inception in a developmental fault. A thorough knowledge of the normal anatomy of the diaphragm is essential for proper surgical repair of these hernias and likewise the origins of the various segments and their musculoaponeurotic composition and mutual insertion in the central tendon are essential information.

Complete absence of the respiratory diaphragm is a very rare autopsy finding in the newborn, but, by careful dissection, fragments of the septum transversum and a cuff of costal attachment can be found. From a practical standpoint, diaphragmatic hernias due to a developmental anomaly are usually concerned with a segmental failure of development in the posterior pleuroperitoneal component and are known as pleuroperitoneal hernias or hernias through the pleuroperitoneal foramen of Bochdalek (Fig. 21). This segment may be absent in part or completely absent. Occasionally the corresponding part of the septum transversum is also deficient so that the hemidiaphragm is totally wanting. Segmental absence of the diaphragm is six times more common on the left side than on the right, presumably owing to the presence of the large right lobe of the liver. When the pleuroperitoneal segment is missing, there is free communication between the abdominal cavity and the hemithorax, which means that there is no hernial sac of either peritoneum or pleura. In this type of defect, the hemithorax is completely filled with abdominal viscera and not only is the homolateral lung collapsed, but there is shift of the heart and mediastinal structures to the opposite side with partial compression of the opposite lung. This situation is not compatible with

life and unless corrected very shortly after birth results in death of the infant.

A common finding in cadaver dissection is a small cleft over the lateral lumbocostal arch in which the pleura is in direct contact with the retroperitoneal fat adjacent to the superior pole of the kidney (Fig. 21). This may be considered the final line of closure of the pleuroperitoneal segment. In between this small cleft, which may be considered normal, and complete absence of the pleuroperitoneal segment, various quantitative deficiencies exist. The larger ones do not have a pleuroperitoneal sac and are symptomatic immediately after birth. The smaller hernias of this type represent failure of the muscle to develop between the layers of the pleura and the peritoneum and they therefore have a combined pleural and peritoneal sac. More commonly they develop so far posteriorly that the retroperitoneal organs protrude into the thorax with pleura alone forming the hernial sac (Fig. 21). The smaller pleuroperitoneal defects prevent immediate herniation and the hernia develops very slowly so that symptoms, or diagnostic evidence of herniation, may not appear until adulthood or they may remain so small that they are an incidental autopsy finding.

Of the lesser apertures through the respiratory diaphragm, only one permits the development of a hernia. This is the cleft between the costal and sternal attachments of the diaphragm through which the superior epigastric vessels regularly pass. These vessels are surrounded by a small amount of fat and areolar tissue that is continuous above with the areolar tissue in the anterior mediastinum and below with the preperitoneal connective tissue. A hernia through this cleft is known as a parasternal hernia or hernia of Morgagni (Fig. 21). In any statistical analysis, this is a rare hernia and is due to deficient muscular development about the foramen of Morgagni. When discovered in infancy it must be due to a developmental defect for it has a peritoneal sac and may contain liver or a loop of a hollow viscus. In the adult, a congenitally large foramen of Morgagni must be the underlying etiologic factor but the secondary factors of obesity and increased intra-abdominal pressure also play a role, because the presenting mass of the hernia is preperitoneal fat with the peritoneum pulled through secondarily (Fig. 21). This hernia is never very large, but instances have been reported in which it protrudes into the pericardial sac.

The remaining type that is congenital in origin is the esophageal hiatus hernia (Fig 21). In adult statistics this appears as the most common of all the diaphragmatic hernias. Hernias through the two other major apertures, the aortic and inferior vena caval hiatuses have not been recorded. The pathologic anatomy of the esophageal hiatus hernia may be very simply described as a hiatus that is too large, so that a pouch of peritoneum and the adjacent fundus of the stomach are forced through the hiatus by the differential pressures within the abdomen and the thorax. The esophagogastric junction may remain fixed at its normal level below the diaphragm so that the hernia consists of fundus and eventually a portion of the body of the stomach or the esophago-

gastric junction may advance through the hiatus as the leading part of the hernia. When this situation prevails the hernia must be differentiated from an additional variety of esophageal hiatus hernia that is due to a congenitally short esophagus. Whereas congenital factors exist in both types of hiatus hernias, when there is a congenitally short esophagus the child is born with the upper portion of the stomach in the posterior mediastinum. When the esophagus is of normal length and the hernia develops because of a large hiatus, the hernia appears gradually after birth and the establishment of negative intrathoracic pressure. The rapidity with which an esophageal hiatus hernia develops is directly related to the size of the hiatus. Although the construction of the

HERNIAS OF THE RESPIRATORY DIAPHRAGM

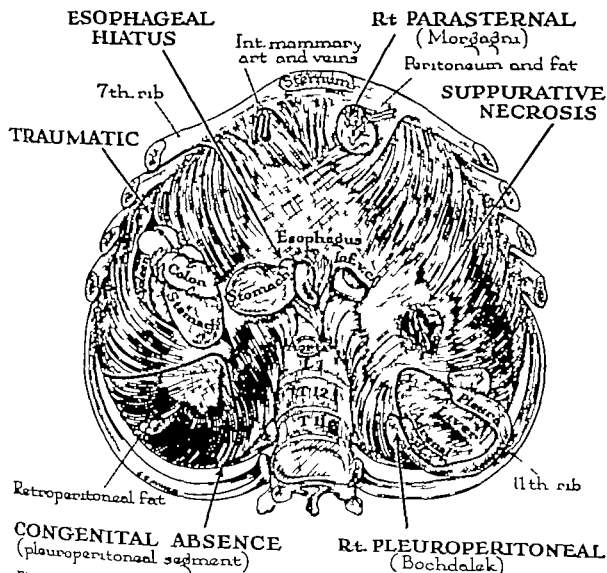


Figure 21 A view of the superior surface of the respiratory diaphragm to show the various types of diaphragmatic hernias.

esophageal hiatus by the crural fibers of the diaphragm is figured correctly in a general way there is considerable variation in the size of the hiatus as well as the strength of the muscular margins. The hiatus is never a fixed or rigid aperture as can be demonstrated during any upper abdominal celiotomy by the fact that one or two fingers may be passed into the hiatus along the side of the esophagus. If this were not so a large bolus of food could not pass through the distal esophagus. It is not possible to state just what the measurements of a normal hiatus should be because of the variation in size of cadavers that do not have an associated hernia. The frequency with which small hiatus hernias are found in the obese adult population suggests that there is an acquired element in the development of some of these hernias which is secondary to obesity and increased intra abdominal pressure. A roentgenologically demonstrable and symptomatic hiatus hernia in an obese patient or a pregnant woman will frequently become asymptomatic with weight reduction or after delivery.

Most traumatic diaphragmatic hernias involve the tendinous dome of the left hemidiaphragm (Fig. 21) because the right lobe of the liver will seal small perforating wounds and dissipate the indirect force so evenly that the right dome is rarely lacerated. A small perforating wound of the left hemidiaphragm, which permits a tag of omentum to enter the left pleural space is the beginning of a diaphragmatic hernia that may take a long time to develop into a symptomatic hernia. Large rents in the diaphragm due either to direct or indirect violence result in the immediate development of a hernia. The traumatic diaphragmatic hernia does not have a peritoneal sac, although adhesions may prevent open communication between the abdominal and pleural cavities. Aside from the perforating wounds so common in warfare crushing injuries are the most common cause of this hernia. The force may be due to abdominal trauma and the diaphragm may be torn by the force of the sudden increase in intra abdominal pressure. More commonly the tear is due to force applied directly to the circumference of the lower thorax. The fracture of a rib along the costal origin may tear the adjacent diaphragm or the sudden anteroposterior or lateral compression of the thorax with the diaphragm contracted may tear the central tendon without rib fracture. After a small rent in either hemidiaphragm,

the constant pressure differential after a time may result in bizarre findings at operation. For example we have seen half of the right lobe of the liver in the right pleural space connected to the infradiaphragmatic portion by a 3-cm. isthmus that represented the diameter of the defect in the diaphragm. On the left, we have observed the spleen as a bilobate organ with the edge of the hernial defect almost bisecting it. This was "oozing" of a solid viscus through an aperture much smaller than the organ itself we have also observed in a right pleuropulmonary hernia.

The final type of diaphragmatic hernia is that due to suppurative necrosis from a subdiaphragmatic abscess (Fig. 21). While the mechanism in the production of a diaphragmatic hernia has been described, suppurative necrosis of the diaphragm more common results in a fistulous connection with a lower lobe bronchus. This is because the diaphragmatic surface of the lower pulmonary lobe becomes adherent to the infected diaphragm long before an actual perforation exists.

Diagnosis. The incidence of the diagnosis of a diaphragmatic hernia is directly correlated with the incidence of suspicion of the examining physician that the patient may have a diaphragmatic hernia. With the exception of the large congenital defects in the newborn or the large traumatic defects in the adult with their dramatic symptoms and findings, the symptoms of a diaphragmatic hernia are usually vague. When the herniation is acute the symptoms simulate those due to acute disease of the heart, lung or gastrointestinal tract. The physical findings may be equivocal and, unless the physician suspects a diaphragmatic hernia and orders appropriate x-rays, the patient may be dismissed with some other diagnosis. The routine x-ray examination of the chest has disclosed many an unsuspected diaphragmatic hernia. Small hernias through the parasternal foramen or the pleuropulmonary foramen especially on the right side, may be totally asymptomatic and be incidentally discovered on a routine chest x-ray film. When this occurs they must be considered in the differential diagnosis of intrathoracic neoplasms.

The prompt diagnosis of a congenital diaphragmatic hernia in the newborn is of the utmost importance if the child is to survive. In every infant who has respiratory difficulty immediately after birth the possibility of a diaphragmatic hernia should be considered. The "blue baby" with a scaphoid abdomen,

a chest which is dull or flat to percussion on the left side associated with the heart shifted to the right has prima facie evidence of a congenital diaphragmatic hernia through the left hemidiaphragm. Air will not be present in the stomach or intestine immediately after birth to aid in the diagnosis, but a simple anteroposterior x-ray examination will usually clinch the diagnosis. If doubt exists, a small polyethylene tube may be passed into the infant's stomach and a small amount of Lipiodol injected for a contrast study. As there is danger that any ingested or injected material may be aspirated by the newborn the use of barium is contraindicated because of its irritating effect on the tracheobronchial tree.

Aside from the hernia occurring in the infant in whom the symptoms are resolved into objective findings, the symptomatology of the diaphragmatic hernias is so varied that no group of symptoms may be ascribed to a given diaphragmatic hernia. In general the symptoms may be divided into those referable to the gastrointestinal tract, the heart and the respiratory system, depending upon the size of the hernia, its location and the pressure it exerts upon its contents.

A loop of intestine constricted by the hernial ring produces signs and symptoms of intestinal obstruction which are no different from those presented by a constricted loop of bowel in an indirect inguinal hernia. However a host of symptoms frequently accompanies the diaphragmatic hernias that are not present in the common varieties of parietal abdominal hernias because of the frequent involvement of the stomach in the diaphragmatic hernia. Complaints referable to the stomach or of dysphagia are common symptoms with esophageal hiatus hernia. Constriction of the stomach where it passes through the hiatus, angulation of the cardio-esophageal junction and scarring secondary to peptic ulceration all cause mechanical obstruction with resultant dysphagia. A temporary obstruction may follow sudden distention of the thoracic portion of the stomach by swallowed liquid, food or air. This is relieved promptly by vomiting or gradually as the material passes into the stomach below the diaphragm. All of the symptoms of gallbladder disease and diseases of the stomach, duodenum and pancreas can be simulated by an esophageal hiatus hernia. Varices or ulceration of the herniated stomach may cause hematemesis. Carcinoma of the distal esophagus or cardia of the stomach may coexist with a hiatus hernia or may re-

semble it both subjectively and objectively. Constriction of the distal esophagus by an angulation may closely resemble carcinoma.

A diaphragmatic hernia which presses upon or displaces the heart may have many symptoms that appear to be cardiac in origin: tachycardia, substernal pressure or pain and a choking sensation due to extrasystoles. Even a small hernia that cannot possibly interfere with the heart will frequently give cardiac symptoms that must be reflex in origin. The same symptoms are reflexly produced by gaseous distention of the stomach or the splenic flexure of the colon. While these cardiac symptoms are usually of major concern to the patient, they are of no organic importance unless the heart is grossly displaced, as occurs in association with the congenital hernia of the newborn or large traumatic rents in the diaphragm. Even so the respiratory embarrassment is far more important, for the heart can work effectively in a variety of positions.

Diaphragmatic hernias which cause compression atelectasis of the ipsilateral lung will, at their outset, cause respiratory embarrassment in direct proportion to the amount of lung collapsed and the degree of mediastinal shift to the opposite side. A diaphragmatic hernia, due to trauma, that has been present for some months and has enlarged gradually may displace a considerable portion of the corresponding lung and yet the patient does not complain of shortness of breath. A compensatory mechanism has been established similar to that occurring in the pneumothorax patient whose respiratory complaints are minimal except with exertion. The mediastinum becomes fixed and does not move with changes in pressure. This same patient may have episodes of discomfort and dyspnea when the contained stomach or bowel is distended with air. The symptoms promptly disappear with belching or expelling flatus, but this sequence of events is also characteristic of many other intra-abdominal conditions.

Esophageal hiatus hernias frequently cause only the mildest of symptoms which are passed off by the patient as "acid indigestion," dyspepsia, heartburn, ulcers or gallbladder trouble. Because of the mildness of the symptoms, their intermittency and the prompt response to any of the antacids, the patient does not usually come to the doctor until the symptoms become more persistent and respond less readily to home remedies. The careful roentgenologic examination of this group of patients will reveal a high in-

cidence of esophageal hiatus hernias. These hernias, as well as the adult types of pleuroperitoneal and parasternal hernias are frequently asymptomatic and are accidentally discovered during a routine x ray examination of the chest or during upper gastrointestinal study. A presumptive diagnosis of a left sided diaphragmatic hernia may be made by auscultation if bowel sounds are heard high in the left side of the chest and breath sounds are absent. However a condition known as eventration of the diaphragm will give the same physical findings. In this latter condition the diaphragm is greatly stretched and attenuated contains very few muscle fibers and the dome lies high in the hemithorax. Motion is slight and paradoxical. The condition is symptomless, is not a hernia and does not require any type of treatment. Eventration is probably congenital in origin, although a similar picture is presented by a patient who has had the phrenic nerve interrupted years before. A very striking elevation of an atonic hemidiaphragm is seen in those patients who have artificial pneumoperitoneum in combination with phrenic nerve interruption for the treatment of pulmonary tuberculosis. It should be apparent that the final diagnosis is dependent upon x ray examination usually with the aid of a contrast medium in the intestinal tract. A right pleuroperitoneal hernia with a globular protrusion of the liver into the right costovertebral angle may be impossible to differentiate from an extrapulmonary tumor such as a neurofibroma, in this location.

Treatment. The asymptomatic esophageal hiatus hernia does not require treatment. A hernia aliposa of the parasternal foramen of Morgagni and a herniation of the liver through a right sided pleuroperitoneal foramen of Bochdalek do not need treatment when asymptomatic if one can be sure of the diagnosis. However they so closely resemble extrapulmonary but intrathoracic solid neoplasms that an operation is usually indicated. It is well to remember that paralysis of the corresponding hemidiaphragm by interruption of the phrenic nerve in the neck is occasionally indicated in the elderly poor risk patient and it may give palliative relief of symptoms. It may also greatly embarrass respiration in the patient with emphysema and minimal respiratory reserve. The obese patient with a symptomatic esophageal hiatus hernia may become completely asymptomatic with the proper weight reduction. Some subjects become asymptomatic by ob-

serving a few simple precautions such as remaining upright after a meal, avoiding over eating and the drinking of carbonated beverages eating slowly reducing their weight and avoiding heavy lifting. When these remedies fail, then surgical correction of the hernia is indicated.

The congenital diaphragmatic hernia in the newborn require immediate surgical correction. Nothing but increasing difficulties and disaster attends procrastination in infants with congenital, segmental absence of the diaphragm. Properly managed, the newborn withstands an operation very well and the technical problem of restoring the viscera to the abdominal cavity is simplified if operation is performed before the intestinal tract contains food and air. Because the affected hemithorax communicates freely with the abdominal cavity positive pressure anesthesia must be instituted before the abdominal cavity is opened. The anesthetic mixture must be high in oxygen content and for this reason cyclopropane is the anesthetic of choice. However for a variety of reasons, we have always used ether. A special infant anesthetic machine must be used because these patients cannot circulate the gases in an adult type of closed system as a result of their small respiratory excursion. A cut-down is established before the operation is started for the administration of water and electrolytes as well as for replacement of blood loss. Blood loss is carefully estimated during the operation because the infants circulating blood volume is small. The amount of blood lost by ordinary standards may be very minimal, but for the newborn infant it may prove to be exsanguinating. The abdominal approach is always used in this group of subjects for several reasons. The costal margin of the infant usually flares widely and is very resilient, which makes the diaphragm readily available for the repair. The abdominal approach permits the surgeon to examine the abdominal viscera for associated anomalies of development and the hernia is more easily repaired with the abdominal viscera delivered out onto the abdominal wall. The infant tolerates surgical evisceration very well, if the viscera are protected by a warm moist pack. This is our initial procedure in these patients, as well as in those with malrotation or intestinal atresia in whom early operation is mandatory.

In older children or in patients having an esophageal hiatus hernia with an associated short esophagus, the thoracic approach is

preferable. A congenital diaphragmatic hernia that has been present for some time may develop adhesions between the viscera and the pleura that are more easily severed by the thoracic approach. In any event time has ruled out associated congenital anomalies of the intestinal tract and the abdominal cavity is sufficiently large after a few months to accommodate the herniated viscera. If the child has survived for a time, the hernia cannot be massive and the quantitative replacement problem is not so great as in those subjects in whom immediate operation is required. In this group, the technical problems of repairing the diaphragmatic defect are more easily handled from above. In patients with a congenitally short esophagus with a thoracoabdominal stomach, the thoracic approach is always used. Whether one simply enlarges the hiatus and fixes the stomach, transplants the esophagus to a higher position on the diaphragm or gains increased length of the esophagus by dissection, the operation is almost impossible from the abdominal approach. This type of hernia does not give serious symptoms in the newborn and therefore the operation is performed upon older children and adults.

Preoperative and postoperative decompression of the gastrointestinal tract is a necessity in most types of diaphragmatic hernia. In recent years we have routinely done a gastrostomy after intestinal surgery on infants and used the gastrostomy tube for gastric suction, which, in turn can be used for trial feedings. If a long intestinal tube has been passed preoperatively the upper gastric and esophageal portion is simply pulled out of the gastrotomy wound and used in the same manner. This greatly reduces the problems in the postoperative period and is followed by a lowered incidence of tracheobronchitis and pneumonia. The few adults in whom we have used the gastrostomy tube for postoperative decompression of the upper gastrointestinal tract, as suggested by Farris, have counted the loss of the preoperative nasogastric tube as their greatest postoperative blessing.

These general principles apply to all adult types of diaphragmatic hernia and, in general, the thoracic approach is preferable. In the traumatic type of diaphragmatic hernia, the thoracic approach is always used unless it is advisable to explore all the abdominal viscera for associated injuries. If operation is delayed for some time after the injury the thoracic approach is mandatory because of the high incidence of very dense adhe-

sions frequently as high as the apex of the pleural space. Hemorrhage at the time of the injury may firmly encase an atelectatic lobe or lung in a fibrous envelope so that it will not expand without decortication and this can only be accomplished by the thoracic approach. Again the hernia repair is more easily accomplished from above.

Finally the repair of the diaphragmatic defect must adhere to sound surgical principles. The strong musculoaponeurotic margins should be approximated in the direction of the fibers of that portion of the diaphragm involved. When the margins of the defect cannot be approximated, several alternatives are available to the surgeon. The costal attachment of the diaphragm can be advanced superiorly to make a flat transverse diaphragm and thus gain additional musculoaponeurotic material for closure of the defect. The size of the hemithorax can be decreased at diaphragmatic level by resecting adjacent ribs. Although we have not had occasion to use it as yet, a sheet of wire mesh could be sewn into the defect and, by the time the mesh fragments, there should be a firm scar tissue lamina between the thoracic and abdominal cavities. Although this would undoubtedly stretch in time it should resemble an eventration rather than a true hernia.

HERNIAS OF THE PELVIS AND PELVIC DIAPHRAGM

Except for the enterocele, hernias of the pelvic wall and pelvic floor occur rarely. The obturator canal and the greater sciatic notch are so well protected by musculoaponeurotic and fascial laminae that it is not surprising that hernias through these naturally occurring channels are rarely seen. On the other hand, the musculoaponeurotic structure of the pelvic diaphragm is frequently observed to be deficient in cadavers dissected routinely; this observation coupled with the fact that the pelvic floor is subjected to severe stresses and strains, especially in the parturient woman, would seem to indicate the pelvic diaphragm as an ideal location for the development of a hernia. This is not the case; in fact, hernias through the pelvic diaphragm are more rarely encountered than are obturator or sciatic hernias. When one considers man's upright carriage, the frequent pressures exerted in the pelvis and the frequency of severe trauma including fractures of the pelvic bones, the extreme rarity of pelvic hernias leads one to the conclusion that these her-

nias have their origin in a congenital defect. This view is possibly strengthened by an occasional report of one of these hernias occurring in an infant.

Our experience with pelvic hernias is confined to protrusions of fat and small peritoneal sacs without contents which we have not infrequently encountered in the examination of cadavers. The sole exception has been a patient having an obturator hernia containing a knuckle of incarcerated and strangulated ileum (Richter's hernia). With the exception of the enterocele which is quite commonly observed, these hernias rarely reach a size at which they present a palpable tumor externally. Their diagnosis is usually established at operation for intestinal obstruction. Isolated instances have been reported for all of the pelvic hernias in which the sac was large and palpable externally.

The *obturator hernia* (Fig. 22) leaves the pelvis through the obturator canal along with the obturator nerve and blood vessels. The canal has very rigid margins and usually just accommodates the neurovascular bundle and a very small amount of areolar tissue that is continuous with the preperitoneal connective tissue. The superior portion of the canal is the obturator groove on the undersurface of the superior ramus of the pubic bone and the groove is converted into a canal by the tough obturator membrane. The canal is deepened and strengthened on either side of the membrane by the internal and external obturator muscles. It is inconceivable that a hernia could develop in the normal obturator canal. A congenitally large canal must be the etiologic basis. From cadaver observation, a pedunculated process of preperitoneal fat precedes and is the entering wedge for the peritoneal hernial sac. In the repair of this hernia, the peritoneal sac is extracted with ease and excised. The margins of the defect, however, cannot be approximated. A patch of adjacent internal obturator fascia can be turned as a flap to close the defect or a patch of wire mesh can be sewed circumferentially in place.

The *sciatic hernia* leaves the pelvis through the greater sciatic notch, either above the piriformis muscle or below it, in company with the corresponding group of gluteal blood vessels (Fig. 23). The *suprapiriformis* or *superior gluteal hernia* is said to be more common. The neck of the hernia is always intimately related to the trunks of the sacral plexus (Fig. 22). Here again, the

normal apertures for the egress of the gluteal vessels from the pelvis are so small that some congenital enlargement of these channels must be the underlying etiologic factor for development of sciatic hernias. If the inferior sciatic hernia follows the course of the internal pudendal artery it is then called a *pudendal hernia*. From cadaver dissections, it would not appear that an adequate fascial flap is available for the closure of these defects and that one must avoid incorporating the trunks of the sacral plexus in the repair. It would appear that a patch of wire mesh would be an ideal material for the closure of sciatic hernial defects.

The *perineal hernias* protrude through the pelvic diaphragm between the musculoaponeurotic fibers of the levator ani muscles. Anterior perineal hernias advance into or through the urogenital diaphragm and posterior perineal hernias expand in the ischio-rectal fossa. The frequent observation of deficiencies in musculoaponeurotic structure in cadavers makes one wonder why these hernias are not more common. A cleft between the pubococcygeus and the iliococcygeus segments of the levator ani is commonly encountered. We have not observed a perineal hernia following perforating wounds of the pelvic diaphragm or following pelvic fractures even with displacement of the bone fragments nor have we observed postoperative pelvic hernias following combined abdominoperineal resection of the rectum or pelvic eventeration. These facts also indicate a congenital defect as the cause of these hernias. After removing the peritoneal sac and reducing fragments of peritoneal connective tissue, the musculoaponeurotic defect should be easy to close from below if the line of closure follows the direction of the musculoaponeurotic fibers of the levator ani.

Enterocele is a true hernia and must be differentiated from protrusions into the vagina due to attenuation of the rectovaginal septum and the vesicovaginal septum known, respectively as *rectocele* and *cystocele*. In Figure 22, a small cystocele and a rectocele associated with an enterocele are shown for comparative study. Although an anterior enterocele occasionally occurs and begins in the uterovesical pouch, the most common enterocele is posterior and has its origin in the rectouterine pouch. The enterocele is not just a prolongation of the rectouterine peritoneal pouch but is a defect through it with the hernial sac dissecting downward in the rectovaginal septum. It bulges into the

posterior vaginal wall where it may be confused with a rectocele. The enterocele commonly occurs in conjunction with a rectocele; when thus associated it may be observed as a secondary bulge above the rectocele. If the enterocele pushes into the rectal ampulla instead of anteriorly into the vagina it is known as a *hidrocele*. The etiologic basis of the enterocele is a rent in the upper end of the rectovaginal septum into which a diverticulum of the peritoneum in the rectouterine pouch protrudes. The trauma incident to parturition is the usual precipitating cause although an enterocele occasionally occurs in a nulliparous woman.

The enterocele rarely becomes incarcerated and the symptoms are essentially those of the rectocele—a heavy feeling in the perineum or a dull, aching type of pain. The

symptoms are aggravated when there is associated uterine procidentia. The average enterocele does not interfere with defecation but a large rectocele usually results in difficulty in evacuating the rectal ampulla. The mechanical reason for this is demonstrated in Figure 22. The enterocele is a fairly common hernia; it is important that it be diagnosed correctly because the usual vaginal plastic operations will not effect a cure. A bulge in the mid or upper posterior vaginal wall that reduces with digital pressure only to reappear with a cough is presumptive evidence of an enterocele. The combined rectovaginal examination will easily differentiate it from rectocele. If a rectocele is present, the examiner's finger in the rectum will be seen in the vagina covered only by the thinned-out rectovaginal

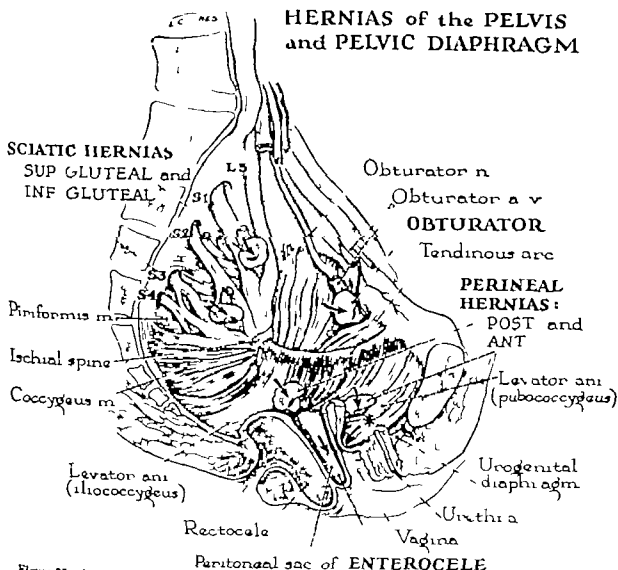


Figure 22. View of the left half of the pelvis after division in the midsagittal plane. The viscera have been removed except for the hollow tubes that perforate the pelvic diaphragm. Except for the peritoneal hernial sacs, all of the peritoneum, preperitoneal connective tissue and the muscular fasciae have been removed to show the apertures of the various pelvic hernias. The arrow with the *h* shows a small cystocele. The cystocele and rectocele are not true hernias but are shown in this figure for comparison.

nas have their origin in a congenital defect. This view is possibly strengthened by an occasional report of one of these hernias occurring in an infant.

Our experience with pelvic hernias is confined to protrusions of fat and small peritoneal sacs without contents which we have not infrequently encountered in the examination of cadavers. The sole exception has been a patient having an obturator hernia containing a knuckle of incarcerated and strangulated ileum (Richter's hernia). With the exception of the enterocele which is quite commonly observed, these hernias rarely reach a size at which they present a palpable tumor externally. Their diagnosis is usually established at operation for intestinal obstruction. Isolated instances have been reported for all of the pelvic hernias in which the sac was large and palpable externally.

The obturator hernia (Fig. 22) leaves the pelvis through the obturator canal along with the obturator nerve and blood vessels. The canal has very rigid margins and usually just accommodates the neurovascular bundle and a very small amount of areolar tissue that is continuous with the preperitoneal connective tissue. The superior portion of the canal is the obturator groove on the undersurface of the superior ramus of the pubic bone and the groove is converted into a canal by the tough obturator membrane. The canal is deepened and strengthened on either side of the membrane by the internal and external obturator muscles. It is inconceivable that a hernia could develop in the normal obturator canal. A congenitally large canal must be the etiologic basis. From cadaver observation, a pedunculated process of preperitoneal fat precedes, and is the entering wedge for the peritoneal hernial sac. In the repair of this hernia, the peritoneal sac is extracted with ease and excised. The margins of the defect, however, cannot be approximated. A patch of adjacent internal obturator fascia can be turned as a flap to close the defect or a patch of wire mesh can be sewed circumferentially in place.

The sciatic hernia leaves the pelvis through the greater sciatic notch, either above the piriformis muscle or below it, in company with the corresponding group of gluteal blood vessels (Fig. 22). The supra-piriformis or superior gluteal hernia is said to be more common. The neck of the hernia is always intimately related to the trunks of the sacral plexus (Fig. 22). Here again, the

normal apertures for the egress of the gluteal vessels from the pelvis are so small that some congenital enlargement of these channels must be the underlying etiologic factor for development of sciatic hernias. If the inferior sciatic hernia follows the course of the internal pudendal artery it is then called a *pudendal* hernia. From cadaver dissections, it would not appear that an adequate fascial flap is available for the closure of these defects and that one must avoid incorporating the trunks of the sacral plexus in the repair. It would appear that a patch of wire mesh would be an ideal material for the closure of sciatic hernial defects.

The perineal hernias protrude through the pelvic diaphragm between the musculoaponeurotic fibers of the levator ani muscles. Anterior perineal hernias advance into it through the urogenital diaphragm and posterior perineal hernias expand in the ischio-rectal fossa. The frequent observation of deficiencies in musculoaponeurotic structure in cadavers makes one wonder why these hernias are not more common. A cleft between the pubococcygeus and the iliococcygeus segments of the levator ani is commonly encountered. We have not observed a perineal hernia following perforating wounds of the pelvic diaphragm or following pelvic fractures even with displacement of the bone fragments nor have we observed postoperative pelvic hernias following combined abdominoperineal resection of the rectum or pelvic exenteration. These facts also indicate a congenital defect as the cause of these hernias. After removing the peritoneal sac and reducing fragments of peritoneal connective tissue, the musculoaponeurotic defect should be easy to close from below if the line of closure follows the direction of the musculoaponeurotic fibers of the levator ani.

Enterocele is a true hernia and must be differentiated from protrusions into the vagina due to attenuation of the rectovaginal septum and the vesicovaginal septum known, respectively as rectocele and cystocele. In Figure 23, a small cystocele and a rectocele associated with an enterocele are shown for comparative study. Although an anterior enterocele occasionally occurs and begins in the uterovesical pouch, the most common enterocele is posterior and has its origin in the rectouterine pouch. The enterocele is not just a prolongation of the rectouterine peritoneal pouch but is a defect through it with the hernial sac dissecting downward in the rectovaginal septum. It bulges into the

THE ALIMENTARY CANAL

Congenital Malformations

By THOMAS H. LANMAN, M.D.

THOMAS HINCKLEY LANMAN was born in Cambridge in the shadow of Harvard University where he obtained his education. Graduated in medicine from Harvard Medical School he was trained at the Massachusetts General Hospital and then became associated with the Children's Hospital in Boston where he became one of the pioneers in the surgery of children

The early diagnosis and the treatment of congenital malformations of the alimentary tract require a working knowledge of embryology. Without such knowledge the various malformations encountered, and there are a great variety, cannot be diagnosed early nor can they be properly treated.

CONGENITAL ATRESIA OF THE ESOPHAGUS AND TRACHEOESOPHAGEAL FISTULA

Malformations and maldevelopments of the esophagus and trachea have become more widely and promptly recognized in recent years. With early recognition, more satisfactory and safer methods of treatment have been established.

One of the most dramatic advances in surgery is the treatment of congenital atresia of the esophagus with or without associated tracheoesophageal fistula. In Figure 1 the various types of atresia are illustrated. One

can reconstruct the symptomatology of the various types, the most important differentiation being whether or not there is a fistula between the esophagus and the air passage. About 90 per cent of these malformations are of type C. In this type the upper esophagus ends blindly and the lower end communicates with the trachea, usually at about the level of the bifurcation. In such cases, saliva or any liquid given for feeding rapidly fills this upper blind pouch. It then overflows and is inhaled into the trachea. This explains the coughing, choking and cyanosis so characteristic in the newborn infant who has this anomaly. Any attempt at feeding the child will produce these symptoms. If the airway is cleared, the infant's condition improves and may appear relatively normal, but each successive attempt at feeding is immediately followed by increasingly severe respiratory symptoms. Symptoms of this sort in the newborn baby should at once direct

septum The enterocele can be felt as a thickening of the rectovaginal septum which disappears when the enterocele is reduced. The repair of the enterocele properly falls in the field of gynecology but, in general the neck of the peritoneal sac must be closed flush with the peritoneum of the cul-de-sac and the rent in the rectovaginal septum closed at the source of the hernia. This operation is usually combined with other plastic procedures performed by the vaginal route. If an enterocele is missed during a posterior vaginal wall repair it will reappear to bother both the patient and the physician.

READING REFERENCES

- Andrews, E., and Bassell, A. D. Direct Hernia. A Record of Surgical Failure. *Surg. Gynec. & Obst.* 58:753, 1934.
- Anson, B. J., and McVay C. B. The Anatomy of the Inguinal and Hypogastric Regions of the Abdominal Wall. *Anat. Rec.* 70:211, 1938.
- Anson, B. J., and McVay C. B. The Anatomy of the Inguinal Region. *Surg. Gynec. & Obst.* 66:186, 1938.
- Anson, B. J., Morgan, E. H., and McVay C. B. The Anatomy of the Hernial Regions. I. Inguinal Hernia. *Surg. Gynec. & Obst.* 89:417, 1949.
- Ashley F. L., and Anson, B. J. The Anatomy of the Region of Inguinal Hernia. II. The Parietal Coverings and Related Structures in Indirect Inguinal Hernia in the Male. *Quart. Bull. Northwestern Univ. M. School* 15:114, 1941.
- Bassini, E. Spora 100 casi di cura radicale dell'ernia inguinale operata col metodo dell'autore. *Arch. ed. art. d. Soc. ital. di chir.* 3:318, 1888.
- Bloodgood, J. C.: The Transplantation of the Rectus Muscle or Its Sheath for the Cure of Inguinal Hernia When the Conjoined Tendon Is Obliterated. *Ann. Surg.* 70:81, 1919.
- Cannaday J. E. Some of the Uses of Cutis Graft in Surgery. *Am. J. Surg.* 59:409, 1943.
- Cloquet, J.: Recherches anatomiques sur les hernies de l'abdomen. Paris, Thèse, 1817.
- Curtis, A. H., Anson, B. J. and McVay C. B. The Anatomy of the Pelvic and Urogenital Diaphragms, in Relation to Urotheciae and Cystocele. *Surg. Gynec. & Obst.* 68:161, 1939.
- Dickson, A. R. Femoral Hernia. *Surg. Gynec. & Obst.* 63:665, 1936.
- Fallis, L. S.: Direct Inguinal Hernia. *Ann. Surg.* 107:572, 1938.
- Farris, J. M., Ettlinger J. and Wernberg, J. A. Hernia Problem with Reference to Modification of the McVay Technique. *Surgery* 24:293, 1948.
- Ferguson, A. H. Oblique Inguinal Hernia, Typo Operation for its Radical Cure. *J.A.M.A.* 33:6, 1899.
- Gross, R. E. The Surgery of Infancy and Childhood. Philadelphia, W. B. Saunders Company 1963.
- Halsted, W. S.: The Radical Cure of Hernia. *Bull. Johns Hopkins Hosp.* 1:12, 1889.
- Halsted, W. S.: The Cure of the More Difficult as well as the Simpler Inguinal Ruptures. *Bull. Johns Hopkins Hosp.* 14:208, 1903.
- Harrington, S. W.: Various Types of Diaphragmatic Hernia Treated Surgically. *Surg. Gynec. & Obst.* 58:735, 1934.
- Hoguet, J. P. Direct Inguinal Hernia. *Surgery* 7:671, 1920.
- Laxon, A. H.: Hernia. Philadelphia, Blakiston Company 1941.
- Koonitz, A. R.: Dead (Preserved) Fascia Graft in Hernia Repair. *J.A.M.A.* 69:1230, 1927.
- Koonitz, A. R. Tantalum Mesh in the Repair of Ventral and Inguinal Hernias. *South. Surgeon* 11:43, 1950.
- Koonitz, A. R. Difficult Hernias. Use of Tantalum Mesh in Repair. *J. Internat. Coll. Surgeons* 18:63, 1951.
- Lothelmsen, G.: Zur Radikaloperation der Schenkelhernien. *Zentralbl. Chir.* 25:548, 1888.
- Marcy H. O.: The Cure of Hernia. *J.A.M.A.* 8:58, 1887.
- McVay R. W., and Lichtenstein, M. E.: Postoperative Hernia. *Am. J. Surg.* 18:60, 1932.
- McVay C. B. A Fundamental Error in the Bassini Operation for Direct Inguinal Hernia. *Univ. Hosp. Bull., Ann Arbor* 5:14, 1939.
- McVay C. B. Inguinal and Femoral Hernioplasty. *Anatomic Repair Arch. Surg.* 57:524, 1943.
- McVay C. B.: The Pathologic Anatomy of the Most Common Hernias and Their Anatomic Repair. Springfield, Ill., Charles C. Thomas, 1954.
- McVay C. B., and Anson, B. J.: Aponeurotic and Fascial Continuities in the Abdomen, Pelvis and Thigh. *Anat. Rec.* 78:213, 1940.
- McVay C. B. and Anson, B. J. Composition of the Rectus Sheath. *Anat. Rec.* 77:213, 1940.
- McVay C. B., and Anson, B. J. A Fundamental Error in Current Methods of Inguinal Hernioplasty. *Surg. Gynec. & Obst.* 74:748, 1942.
- McVay C. B., and Anson, B. J.: Inguinal and Femoral Hernioplasty. *Surg. Gynec. & Obst.* 63:47, 1949.
- Polya, E. Die Ursachen der Rezidive nach Radikaloperation des Leistenbruchs. *Arch. Klin. Chir.* 90:816, 1912.
- Rienhoff, W. F. The Use of the Rectus Fascia in Closure of the Lower or Critical Angle of a Wound in the Repair of Inguinal Hernia. *Surgery* 8:328, 1940.
- Seison-Jarochewitsch, A. J. Pathogenese und Behandlung der Leistenbrüche, anatomisch-klinisch beleuchtet. *Arch. Klin. Chir.* 144:352, 1927.
- Veal, J. R., and Baker D. D. Repair of Direct Inguinal Hernia by Osteoperitoneal Graft to the Peritoneal Line of the Pubis. *Surgery* 3:583, 1938.
- Wangsten, O. H. Repair of Recurrent and Difficult Hernias. *Surg. Gynec. & Obst.* 59:766, 1933.
- Watson, L. F.: Hernia, 3rd ed. St. Louis, C. V. Mosby Company 1948.
- Zimmerman, L. M. Recent Advances in Surgery of the Inguinal Hernia. *S. Clin. North America* 3:103, 1952.
- Zimmerman, L. M., and Anson, B. J.: The Anatomy and Surgery of Hernia. Baltimore, Williams & Wilkins Company 1953.

THE ALIMENTARY CANAL

Congenital Malformations

By THOMAS H. LANMAN, M.D.

THOMAS HINCKLEY LANMAN was born in Cambridge in the shadow of Harvard University where he obtained his education. Graduated in medicine from Harvard Medical School he was trained at the Massachusetts General Hospital and then became associated with the Children's Hospital in Boston where he became one of the pioneers in the surgery of children.

The early diagnosis and the treatment of congenital malformations of the alimentary tract require a working knowledge of embryology. Without such knowledge the various malformations encountered, and there are a great variety, cannot be diagnosed early nor can they be properly treated.

CONGENITAL ATRESIA OF THE ESOPHAGUS AND TRACHEOESOPHAGEAL FISTULA

Malformations and maldevelopments of the esophagus and trachea have become more widely and promptly recognized in recent years. With early recognition, more satisfactory and safer methods of treatment have been established.

One of the most dramatic advances in surgery is the treatment of congenital atresia of the esophagus with or without associated tracheo-esophageal fistula. In Figure 1 the various types of atresia are illustrated. One

can reconstruct the symptomatology of the various types, the most important differentiation being whether or not there is a fistula between the esophagus and the air passage. About 90 per cent of these malformations are of type C. In this type the upper esophagus ends blindly and the lower end communicates with the trachea, usually at about the level of the bifurcation. In such cases saliva or any liquid given for feeding rapidly fills this upper blind pouch. It then overflows and is inhaled into the trachea. This explains the coughing, choking and cyanosis so characteristic in the newborn infant who has this anomaly. Any attempt at feeding the child will produce these symptoms. If the airway is cleared, the infant's condition improves and may appear relatively normal, but each successive attempt at feeding is immediately followed by increasingly severe respiratory symptoms. Symptoms of this sort in the newborn baby should at once direct

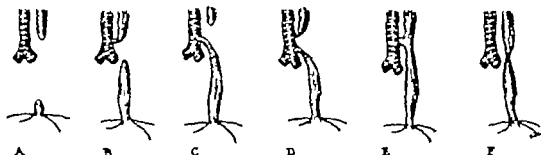


Figure 1 Types of congenital abnormalities of the esophagus. A, Esophageal atresia, no esophageal communication with the trachea. Under such circumstances the lower esophageal end is very apt to be quite short. B, Esophageal atresia, the upper segment communicating with the trachea. C, Esophageal atresia, the lower segment communicating with the back of the trachea. Over 90 per cent of all esophageal malformations fall into this group. D, Esophageal atresia, both segments communicating with the trachea. E, Esophagus has no disruption of its continuity but has a tracheoesophageal fistula. F, Esophageal stenosis. (Craw: The Surgery of Infancy and Childhood.)

the physician's attention to the possibility of this anomaly.

When the lower end of the esophagus communicates with the trachea, air readily finds its way into the stomach and bowel. This is easily demonstrated by a scout film of the chest and abdomen. The appreciation that about 90 per cent of these anomalies are of this type shows the futility of attempting, as was the case in earlier years to treat this anomaly by a "preliminary" gastrostomy, for by this procedure anything introduced into the stomach immediately finds its way into the lungs.

The diagnosis is easily established by passing a small no. 10 urethral catheter down the esophagus. Its passage is stopped at the level of the atresia. An x-ray film of the chest and abdomen with the catheter in place will give visual evidence of the level of the atresia and in this type air will be seen in the stomach and upper bowel, demonstrating the communication between the trachea and the lower end of the esophagus. Contrast media are sometimes of help but Iptodol should be used, never a barium mixture. A certain amount of spill-over of the opaque material is to be expected and, if barium is used, it will cause a severe pulmonary reaction.

Prompt recognition of these characteristic symptoms should lead to early diagnosis. It is gratifying to observe that this anomaly is more frequently diagnosed than in the past. In the years 1926 to 1936, there were twenty-two patients with this anomaly admitted to the Boston Children's Hospital. In the following ten years, 1936 to 1946, there were 144 patients. From the above, one might argue that this anomaly had increased by about 500 per cent in ten years. This, of course, is not true and the increase must be

accounted for by the more frequent recognition of the condition. It seems probable that from 1926 to 1936 there must have been a comparable number of infants born with this anomaly. The anomaly was not reported as such and the death certificate probably had the diagnosis "pneumonia, which, indeed, these infants had and from which they died.

In the Boston Children's Hospital series, there was a high incidence of prematurity in the infants who had this anomaly. Fifty-two of the 250 patients studied weighed 5 pounds or less at birth; this is significantly higher than the prematurity rate seen in association with other malformations. This observation merits further study. Associated with this condition there is also a rather striking incidence of anomalies elsewhere in the body many of which are of very serious consequence, particularly congenital heart disease, atresia or stenosis of the bowel and malformations of the anus and rectum. Some of these associated anomalies may require surgical treatment before or at the time of surgical repair of the tracheoesophageal anomaly. It is well, therefore, to consider the possibility of a coexisting abnormality in all these patients.

It is important to point out that the surgical repair of this anomaly should not be regarded as a true "emergency" procedure, for it has been found that spending twelve to twenty-four hours in overcoming the effects of dehydration and combating the varying degrees of pneumonitis which all these patients exhibit is useful and indeed essential. The infant should be placed in a semiprone position to minimize the amount of gastric secretions entering the trachea and the bronchial tree through the fistula from the lower esophagus. Constant suction of the

upper blind pouch by means of a small urethral catheter should be instituted to prevent the overflow of saliva from the upper blind pouch into the air passage. Intensive chemotherapy should be started at once. My colleagues and I have used a combination of penicillin and streptomycin. An oxygen tent is always helpful. It is of prime importance to have continuous nursing care, with particular attention to the maintenance of a clear airway.

As in any extensive surgical procedure in the young, means to maintain constant intravenous fluid administration should be established. This is best done by inserting a fine polyethylene tube through a cut-down incision over an ankle vein. This allows the administration of fluids and of blood as needed. It may be left in place for thirty six hours and should, of course, be inserted before the operation. Since there is definite danger of phlebitis if the tube is left in place for more than forty-eight hours, it is wise to use another vein if the intravenous route is still needed.

A skilled anesthetist is essential. It has been found that the use of the intratracheal tube may result in a disturbing and even dangerous irritation of the larynx or trachea in these small patients. Also the intratracheal tube may allow too high a pressure within the alveoli, leading to pulmonary edema in the postoperative period. Cyclopropane-oxygen administered through a properly fitting face mask is the anesthetic of choice.

A direct transpleural attack on this anomaly with closure of the fistula and a direct anastomosis of the two ends of the esophagus is the preferred procedure. In earlier years this method of treatment did not seem possible and in some subjects the fistula was closed and an exterior esophagus constructed. For a patient suffering from cancer the construction of an exterior esophagus is justifiable, but to commit an infant to such a life is most undesirable and this procedure has been abandoned entirely since 1945. All patients are now submitted to a one-stage repair with closure of the fistula and primary anastomosis of the esophagus.

After direct anastomosis, some of the patients may be fed by mouth from the start. However it is far safer to perform a temporary Stamm-type gastrostomy employing a self retaining catheter either at the time of the original operation or the next day. This procedure permits early and adequate feeding and allows better healing of the esophagus.

It is best done through an upper left rectus muscle-splitting incision.

Following surgery these infants need plenty of oxygen in a moist wet atmosphere. Suction should be available at all times. Chemotherapy is continued until all evidence of pulmonary infection has disappeared. Usually within a few days the child can swallow its own saliva. Feedings through the gastrostomy opening are started on the second day using a 10 per cent solution of glucose in appropriate amounts at short intervals and adding a milk formula by the fourth day. Caution must be taken not to give too large amounts at a time. The stomach in these patients is usually smaller than normal and any overdistention will cause regurgitation of the stomach contents up into the esophagus. The safest method is to give these feedings by the gravity drip method and to spend at least a half hour in each administration. Oral feedings may usually be safely started on the tenth postoperative day. By the end of three weeks the infant is able to take full feedings by mouth, at which time the gastrostomy tube is left in place but is clamped off. By the end of a month or six weeks, the gastrostomy tube may be safely withdrawn and the gastrostomy opening allowed to close spontaneously. The greatest postoperative dangers are the respiratory complications and until these are well under control the patient should remain in the hospital. However in our series of patients, about 75 per cent of those surviving operation were discharged with safety at the end of three weeks.

It is important to keep these patients under observation for a number of months and to remember that even though they may swallow liquid food, they may have some later difficulty with solid food. Dilation is needed only when there is clinical evidence of stenosis. X-ray evidence alone is not a reliable guide.

CONGENITAL SHORTNESS OF THE ESOPHAGUS

Congenital shortness of the esophagus is always associated with the presence in the lower mediastinum of a segment of the stomach lying in the same axis as the esophagus. This segment is usually cylindrical in shape and is therefore sometimes not recognized as stomach by the roentgenologist. When exposed at operation or autopsy however the condition is obvious. The esophago-gastric junction (cardia) in an individual

with this condition lies at a level above the diaphragm, sometimes as high in the mediastinum as the arch of the aorta. Although there is sometimes a small upward projection of the peritoneum to form a short sac along the anterior surface of the thoracic segment of stomach, the condition is not, strictly speaking, a true herniation through the esophageal hiatus. This distinction must be made in order to permit the surgeon to choose the correct procedure if an operation becomes necessary.

Although it is sometimes compatible with the enjoyment of a normal existence, patients who have this anomaly have a much greater than normal tendency to develop esophagitis in the segment of esophagus just above the cardia. This complication may not be encountered until the patient reaches adult years or even old age.

Ulcerations frequently develop and the ultimate result of the destruction of mucous membrane is healing by cicatrization with subsequent stricture formation. Treatment by bougienage is sometimes successful in maintaining an adequate lumen. In certain patients however resection of the diseased area must be resorted to and can be performed with relative safety and complete relief of the condition by a left side trans-thoracic approach. It is an interesting fact that a relatively large proportion of patients

with a short esophagus, thoracic stomach and esophagitis or ulceration near the cardia have coexisting duodenal ulcer. These patients may have a particular disposition to ulcer formation.

CONGENITAL OR REDUPLICATION CYSTS OF THE ESOPHAGUS

These are rare in occurrence but may become so large that dysphagia results. In an occasional patient such a cyst may become infected and produce serious difficulties. Occasionally a cyst empties by discharging its contents into the lumen of the esophagus. A cyst can usually be removed from the wall of the esophagus by local excision in such a manner that the defect can be repaired without the necessity for segmental resection.

CONSTRICTION OF THE ESOPHAGUS BY ANOMALOUS OR ABERRANT VESSELS IN THE MEDIASTINUM

Constriction of the esophagus and trachea by an anomalous double aortic arch or by an aberrant innominate artery which arises from the left side of the aortic arch and passes across the esophagus to the right is a rare cause of congenital dysphagia. Division of one of the vessels which make up the vascular ring of a double aortic arch brings about immediate relief (Fig. 3). Compression of the esophagus by an aberrant right



Figure 2. A roentgenogram made after ingestion of barium, showing a large, rounded filling defect in the lower portion of the esophagus produced by a congenital enterogenous cyst of the esophageal wall.

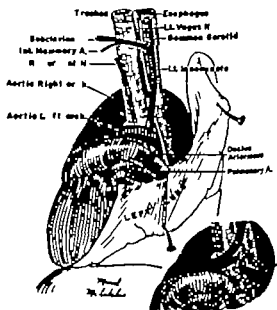


Figure 3. A drawing illustrating the anatomic findings in a patient with constriction of the esophagus and trachea produced by a double aortic arch. The insert shows the method of producing release of the constriction by ligation and division of the arterial vessel.

innominate artery arising from the aorta at the point where the arch becomes the descending portion and passing usually behind the esophagus, rarely gives rise to dysphagia severe enough in degree to make surgical interference necessary. Difficulty in swallowing caused by this anomaly has acquired the name dysphagia lusoria.

CONGENITAL HYPERTROPHIC STENOSIS OF THE PYLORUS

Congenital hypertrophic stenosis of the pylorus is one of the most common conditions requiring surgical treatment in early life, but there is still some difference of opinion as to its true etiologic background. Grossly there is marked hypertrophy of the circular smooth muscle fibers of the pylorus. The resultant mass is usually described as being of the shape and size of an olive; actually it is more spool shaped as its ends are not oval. The most striking characteristic of this mass is its firm, gristly almost cartilaginous consistency. The consistency is important in the diagnosis for there is no doubt in the mind of the surgeon when he palpates the tumor of true pyloric stenosis. If he thinks he feels a tumor the chances are great that he is wrong. He may be feeling the liver's edge, the pole of the kidney or an edge of a muscle.

While there is no racial predisposition, there is a striking sex preponderance, far more so than in any other surgical condition found in this age group. Males are affected more commonly than females in a constant ratio of 4:1. This is of considerable significance in the differential diagnosis. The condition is more likely to occur in a first born child than in subsequent siblings, but this is by no means as striking as the sex incidence. It may occur in more than one sibling, in our series there was one instance in which four siblings had this condition.

The symptoms are those of obstruction at the pylorus. The vomiting usually begins in the second or third week of life rarely before the tenth day. The vomiting at first is not striking and may easily be considered as little more than the regurgitation so frequently occurring after feeding an infant. As the condition progresses, the vomiting becomes more and more forceful and though often spoken of as "projectile," "explosive" seems to be a better term. The mother will often volunteer a characteristic description of the vomiting and her statement should never be disregarded. In the well-established case, the vomiting will occur shortly

after each feeding. Of prime importance is the fact that the vomitus does not contain bile. With the increasing obstruction and vomiting, the infant will show the results of starvation. He fails to gain and later rapidly loses weight. There is a marked loss of subcutaneous fat particularly noticeable about the orbits. There is loss of turgor in the skin which has a characteristic wrinkled appearance especially marked in the face neck and extremities. The infant is not "sick." He is extremely hungry and will nurse eagerly even after a bout of vomiting. The mother usually states that the child is constipated. This is not true: the stools are scanty and infrequent because little of the food intake reaches the bowel. One of the most characteristic features is the presence of vigorous waves of peristalsis in the upper abdomen. These waves are readily seen if the child is examined during a feeding and the more advanced the disease the greater the loss of subcutaneous fat and the more prominent are these waves passing from left to right. The abdomen is not distended, as the obstruction is high, though there is usually a fullness in the epigastrium. The diagnosis is definitely established on palpation of the characteristic tumor mass in the right upper quadrant. With increasing experience this tumor has been felt in over 95 per cent of our subjects. The firm, gristly tumor is most likely to be palpated just after the infant has vomited, at which time the abdominal wall relaxes. Palpation must be done slowly patiently and with gentleness. Little will be learned by hasty sudden palpation, especially with a cold hand.

In the average case, x ray confirmation is not needed. In the unusual subject, it is simple and easy to give a small amount of a thin mixture of barium by mouth and, if true pyloric stenosis is present, the pyloric canal will appear to be greatly narrowed and lengthened, with dilatation of the stomach associated with deep waves of peristalsis.

In summary then, this condition is manifested by vomiting which usually starts about the end of the second week of life and becomes increasingly frequent and more forceful. The vomitus does not contain bile. The infant shows progressive evidence of weight and fluid loss. The stools are scanty but otherwise normal. The infant is constantly hungry and this also helps to differentiate the vomiting and loss of weight from that caused by infections. The most significant finding, which with care and patience should be obtained in over 95 per cent of the

subjects is the hard, firm tumor in the pyloric area.

Intracranial injuries occurring during birth or a blood dyscrasia responsible for intracranial bleeding may cause vomiting in the newborn. In such infants, however the vomiting is not characteristic of that due to an obstruction nor does it usually have a specific relationship to the taking of food. There are likely to be other symptoms of intracranial damage such as convulsions or a bulging fontanel and in such instances a spinal or subdural tap is indicated. The vomiting is likely to appear sooner after birth than when it is due to pyloric stenosis as is also true of the vomiting caused by malformations of the esophagus. Congenital obstruction, however in the region of the duodenum may show many of the characteristics of pyloric stenosis but in this condition there is usually bile in the vomitus and this excludes the diagnosis of simple and true pyloric stenosis.

It is probable that there is an element of spasm in all cases of true pyloric stenosis, but the symptoms of pylorospasm alone are much less characteristic than are those of true pyloric stenosis. In pylorospasm the vomiting is as a rule not progressive, though it is often forceful and there are periods of remission. The child having pylorospasm often exhibits a generalized hypertonicity and his symptoms can often be entirely relieved by use of atropine and mild sedation. These drugs do not affect the symptoms in true pyloric stenosis.

Pyloric stenosis is in no sense a surgical emergency. It is always possible and desirable to take time to rule out the possibility of other organic bases for the vomiting with a careful evaluation of the feeding program as regards its suitability for the particular infant.

Twenty four to forty-eight hours may be well spent in getting the patient in good condition for operation. He needs, in addition to a 10 per cent solution of glucose for its caloric value, physiologic saline solution to make up the loss of chlorides caused by vomiting due to a high intestinal obstruction. Normal saline solution, 15 cc. per pound of body weight, and 10 per cent aqueous solution of glucose, 10 cc. per pound, are given twice daily. It is most desirable to administer the glucose intravenously but the fluid may be given subcutaneously if the sites of injection are changed frequently. For the subcutaneous injection, a 5 per cent aqueous solution of glucose is safer than a 10 per

cent solution and the addition of hyaluronidase is helpful. If the red blood count is much below 4 million per cu. mm., transfusion of blood is useful, as there is likely to be a low plasma protein level in such infants, 10 cc. of blood per pound of body weight at any one transfusion is the upper limit of safety. In the usual patient, however blood transfusion is not needed. The fluid requirements are well fulfilled by the parenteral administration of glucose and saline solutions. Preoperatively milk given by mouth is of little benefit and may indeed do harm. Since vitamin C is of considerable importance in promoting better healing of the wound, 25 mg. of ascorbic acid a day are routinely given.

The anesthetic of choice is ether administered by the open-mask method. This has been used in 98 per cent of our patients and there have been no deaths which could be attributed to this anesthetic. Atropine, 0.08 mg. is given preoperatively but morphine should not be used. If the staff anesthetist has not had sufficient experience with the open-mask method in the infant patient, local anesthesia, supplemented with the use of a brandy-and-sugar nipple, may be safely employed, although it prolongs the operation and does not give as satisfactory relaxation as ether. The usual precautions in preserving the infant's body heat by appropriate covering of the arms and legs and using a hot water bottle under the back are essential. A small urethral catheter introduced through the nose into the stomach aids greatly in deflating the stomach.

The right upper quadrant gridiron incision is superior to all others. It allows prompt and solid healing and, if properly placed, permits adequate exposure of the pylorus. The incision should be about 3 cm. long, extend outward from the lateral border of the right rectus muscle and run parallel to and just below the liver edge. The costal margin is a less reliable landmark. The Ramstedt incision of the pylorus is made on the less vascular anterosuperior surface of the pyloric tumor. There is danger of entering the bowel at the duodenal end where the hypertrophied pyloric muscle fibers end abruptly. Should such an accident occur the opening may be readily and securely closed with one or two fine silk sutures to invert the wound in the bowel. Under such circumstances, it is advisable to leave the gastric catheter in place and to provide constant suction for twenty four to forty-eight hours after the operation. Nothing should be given by

mouth. Meticulous care in closing the peritoneum and abdominal wall is necessary.

In the first two or three days following operation, it is neither necessary nor desirable to give the normal amount of nutriment. It is, however, important to maintain the fluid intake. The plan of giving 3 ounces of fluid per pound of body weight per twenty-four hours is a useful guide.

The actual content of the formula may vary greatly but the amounts given at any one time must be rather small at first. Although the schedule of feeding may be varied to meet the individual need, it is well to have some standard form of procedure so that the house staff and nurses may appreciate the usual routine. At the Children's Hospital the following formula and feeding schedule have been found most useful.

FEEDING FORMULA

Evaporated milk	6 parts
Karo (red label) 50 per cent	1 part
Water	8 parts

FEEDING SCHEDULE

Beginning 4 hours after operation.

1 oz. 5% glucose	every 2 hrs.	3 feedings
1 oz. formula	every 2 hrs.	till 8 P.M. day after op.
		6 feedings

Beginning second morning after operation.

2 oz. formula	every 3 hrs.	8 feedings
---------------	--------------	------------

Beginning third morning after operation.

3½ oz. formula	every 4 hrs.	6 feedings
----------------	--------------	------------

After the fourth or fifth day, fluid and caloric requirements can usually be met by oral feedings. Not infrequently there will be some postoperative vomiting. Should this occur it is usually necessary only to reduce the oral intake in amount or to build up the intake more slowly.

With increasing knowledge better anesthesia, adequate preoperative and postoperative care, the results of surgical treatment of pyloric stenosis have improved tremendously. In the decade following the introduction of the Ramstedt operation, the over-all mortality in this country was approximately 50 per cent. Since 1938 at our hospital, over 1500 infants have been treated, with a mortality of less than 1 per cent. An incomplete division of the pyloric muscle made necessary a secondary operation in four of these patients. There has been complete and lasting relief of the symptoms. We have patients who have been followed for as long as thirty-five years and as yet no late complications have been observed.

ATRESIA AND STENOSIS OF THE INTESTINE

Atresia of the intestine is a complete lack of intestinal continuity. Stenosis is a narrowing of the intestine. Congenital atresia and stenosis of the small intestine are the result of an arrest in the development of the embryo at an early age. Prior to the fifth week of fetal life the intestine has acquired a lumen lined with epithelium. Between the fifth and twelfth weeks the intestinal lumen becomes obliterated by epithelial confluence and is changed into a solid cord. By the twelfth week the solid cord has become vacuolized, the vacuoles have coalesced and the intestinal lumen has become re-established. Atresia is the result of an arrest in development during the solid stage (Fig. 4). The cause of such arrests in development is still a matter of surmise. Stenosis of the intestine represents an arrest of growth in a slightly later stage of development, namely the stage of vacuolization and is manifested by a septum across the bowel with a small perforation in it. There may be two blind ends, the proximal separated from the distal by a small, fibrous cord of varying length.

Atresia and stenosis may occur at any point in the small intestine but are most commonly found in the ileum next in the duodenum and next in the jejunum they may be multiple. Atresia and stenosis of the colon are rare lesions. Gross described only six colonic atresias in a series of 140 intestinal atresias observed in the Boston Children's Hospital, and only one stenosis in seventy-one patients with stenosis of the intestinal tract. In intestinal or colonic atresia, the bowel above the obstruction becomes dilated and that below remains collapsed. In the patient with atresia, the bowel below the obstruction appears like a small hard cord, although, on examination, it is found to contain all the normal elements, including a lumen. This lumen, however may be only large enough to admit a small probe. In patients having stenosis the bowel below the point of obstruction is not as small as in those with atresia.

In the newborn infant the symptoms of atresia and stenosis are pain, restlessness, vomiting and lack of passage of normal meconium. On examination, abdominal distention, visible peristalsis or coils of intestine and evidence of shock and dehydration are apparent. Exceptions to these usual findings may be noted if the obstruction is in the duodenum. In such patients the vomiting



Figure 4 The septum across the duodenum *a*, the result of the arrest of development at the solid step *b* duodenojejunostomy for relief of the duodenal obstruction caused by *a*.

may be so sufficiently effective that the stomach and duodenum are thoroughly emptied and consequently no abnormal distention or peristalsis is seen.

A roentgenogram taken without the administration of barium is of value in confirming the diagnosis (Fig. 5). The point of obstruction may be clearly outlined by the air in the dilated loop of intestine above it. The administration of barium to a patient with suspected atresia is not only unnecessary but is contraindicated, as the barium will block the small lumen of the intestine below the obstruction after an anastomosis has been performed and thus impede recovery. Another aid in confirming the diagnosis is the failure to find any keratinized cells in the stools.

When the diagnosis of congenital obstruction has been made, the patient is prepared for operation by parenteral administration of fluids in sufficient amounts to counteract dehydration, alkalosis or ketosis. In considering the operative measures to be employed, it is well to divide the various types into separate classifications, depending on the site of the obstruction. The obstructions in the duodenum are the first to be considered.

The intrinsic type of duodenal obstruction usually occurs in the third portion of this structure and it is often impossible, as well

as inadvisable, to attempt to determine whether the obstruction is due to stenosis or atresia. In either case the operation of choice is a retrocolic duodenojejunostomy. In a few subjects because of too great technical difficulties in performing such a duodenojejunostomy, a gastrojejunostomy may be performed. This gastric operation is not satisfactory physiologically because of the probable reflux of bile and duodenal contents, if the obstruction is beyond the ampulla of Vater as it often is. If obstruction exists immediately distal to the pylorus, gastroduodenostomy may be appropriate. If a membrane is present which obstructs the duodenum it may be difficult or impossible to determine the site and cause of obstruction without opening the proximal portion of the duodenum and inserting an exploring finger downward in the duodenum. At times such an opening subsequently may be utilized in the performance of duodenojejunostomy. If an obstructing membrane is found, this may be excised or by-passed with an intestinal anastomosis. In exceptional cases, concomitant obstruction of the common bile duct may be present, under these circumstances a simultaneous biliary intestinal anastomosis of an appropriate type is indicated.

For a patient who has stenosis or atresia lower down in the small bowel, a lateral

anastomosis should be performed. For this the special technique recommended by Ladd is of great value. As shown in Figure 6, a rubber catheter is inserted in the distal lumen. The caliber of the ileum beyond the site of the obstruction is very small. The lumen of the gut has never been distended with meconium. By gentle distention of the distal gut with saline solution introduced through the catheter the disproportion between the dilated proximal gut and the small undeveloped distal gut is lessened and the placing of the sutures becomes technically much easier.

In the after-care, great attention must be paid to maintaining the fluid requirements as well as the electrolyte balance and the serum protein levels of the patient.

Chemotherapy and antibiotic therapy in their various forms are an added safeguard and should be used in all these patients. It is important to emphasize however that they are by no means a substitute for the other important details of good surgical treatment, especially the establishment and maintenance of proper fluid and electrolyte balance. The use of chemotherapeutic agents within the peritoneal cavity should be abandoned. In these small patients there is danger of too sudden and too great a blood level of the agent employed and, instead of aiding in recovery it may greatly

impede it or even result in a fatal outcome. The drug should be administered parenterally by the continuous intravenous route which is best established before the actual operation is begun.

It is desirable that feedings be given by mouth as soon as possible, as this assists greatly in enlarging the lumen of the bowel distal to the site of the anastomosis. Saline enemas help to restore the large bowel to a more normal size and greatly assist in the restoration of bowel function.

With improved technique and particularly with improved preoperative and postoperative care, the prognosis is becoming more favorable. The nearer the obstructive lesion is to the pylorus, the better the chance for a successful operative result. In the Children's Hospital from 1940 through 1952, recoveries were 57 per cent. In 1952 there were nineteen cases of atresia or stenosis with thirteen recoveries (68 per cent).

There is one rather striking finding in these cases which demands further investigation this is the apparent association of mongolism with congenital obstruction in the duodenum. In reviewing our results it has been found that about 25 per cent of the long term survivors of the operative correction of duodenal atresia or stenosis are Mongolians. This figure is a disturbing one and has been confirmed by a report of similar cases from the Great Ormond Street Hospital for Sick Children in London. This hospital reports an incidence of nearly 30 per cent of mongolians in the subjects who survive.

Obviously the diagnosis of mongolism cannot be established with certainty in the



Figure 5. Roentgenogram of a patient with atresia of the duodenum which shows clearly the point of obstruction without the administration of barium.

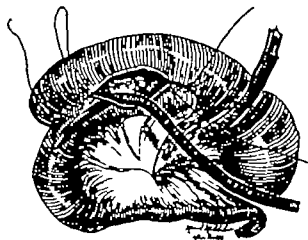


Figure 6. Method of using a soft rubber catheter to dilate, with saline solution, the narrow undeveloped distal portion of the ileum at the time of side-to-side anastomosis.

first forty-eight hours of life. Efforts must then be continued to salvage an increasing number of patients born with this anomaly. The great challenge, however is to find out why mongolism is apparently so frequently associated with this anomaly and also the etiologic basis of mongolism itself. Certainly if 25 to 30 per cent of the survivors of brilliant surgery done in the immediate neonatal period prove to be mongolians, the results can hardly be called brilliant for the patient or his family.

MALROTATION AND NONROTATION OF THE INTESTINE

Incomplete rotation, malrotation and non rotation are terms used to describe various abnormal abdominal conditions which are the result of arrest in fetal development during or after the return of the midgut from the umbilical cord to the abdominal cavity. For an understanding of these conditions and for their successful treatment it is necessary to review some of the embryologic events which take place between the fifth and tenth weeks of fetal life.

At approximately the fifth week of fetal life the abdominal viscera grow much more rapidly than does the abdominal cavity with the result that there is insufficient room within the cavity to accommodate the organs which are destined to be located there. Nature meets this situation by temporarily transferring the midgut, i.e., that portion of the intestine supplied by the superior mesenteric artery into the base of the umbilical cord. In other words a temporary and normal omphalocele occurs which lasts from the fifth to the twelfth week of fetal life. During this period the rate of growth of the abdominal cavity is accelerated so that by the tenth week it has acquired sufficient size to reaccommodate its organs.

The return of the midgut to the abdomen takes place in a particular way. The prearterial segment, or that portion of the intestine extending from the duodenum to the vitelline duct, returns first and passes from right to left behind the artery. The postarterial segment, or that portion of the midgut from the vitelline duct to the middle of the transverse colon, returns next and passes from left to right in front of the superior mesenteric artery.

The next stage of development consists in the descent of the cecum to the right lower quadrant and the fusion of the visceral and parietal peritoneum to give the mesentery of the small intestine its oblique attachment

to the posterior abdominal wall and the cecum and ascending colon their stabilizing attachment to the right side. An arrest in development may take place at almost any stage of this process (Figs. 7 and 8). Then the cecum may remain on the left side, under the ensiform or in the neighborhood of the gallbladder (Fig. 9). These faulty positions may not of themselves be important or cause disturbance of the normal function of the bowel. A faulty position is important only when it causes pressure on one part or another of the bowel and results in obstruction. When obstruction occurs, the condition becomes of importance and necessitates relief by surgical intervention.

In instances in which the development has been arrested before the parietal and visceral peritoneum were fused, the midgut remains attached to the posterior abdominal wall by only a very small area at the origin of the superior mesenteric artery. Under these circumstances, a volvulus of the midgut is particularly liable to develop. The twisting of the midgut on itself with the origin of the superior mesenteric artery as a pivot is most likely to take place in a clockwise direction and may go through an arc of 300 degrees or more. Of course, the more it twists, the more complete is the obstruction and the greater the interference with the blood supply of the bowel.

The symptoms of the conditions resulting from faulty rotation or volvulus of the midgut are those of intestinal obstruction, namely vomiting, pain, restlessness and evidence of shock and dehydration. These symptoms may appear soon after birth or in contrast to the symptoms of congenital atresia and stenosis, may not appear for months or even years. They may be intermittent in character and of variable severity. On examination, abdominal distention and visible peristalsis are noted. It is to be remembered, however that the obstruction takes place most commonly in the duodenum, so that the distention may be less marked than in other forms of obstruction lower down in the intestine. Distention, if present, is the result of the volvulus of the midgut. If the patient is not too acutely ill, a barium enema with roentgen ray examination may be used to confirm the diagnosis by showing the abnormal position of the cecum. In the still less severely ill patient, a barium meal and a series of roentgen ray examinations may yield useful information (Fig. 10).

The treatment of these conditions is sur-

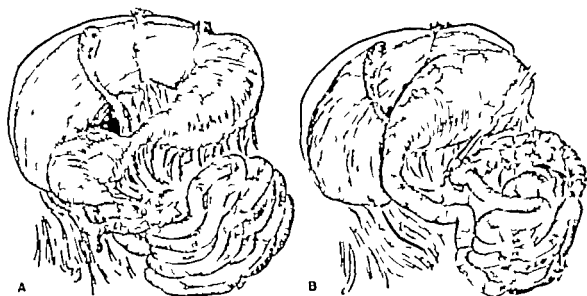


Figure 7 A, Drawing showing the obstruction and dilatation of the duodenum caused by the mesenteric attachment of an unrotated and undescended cecum. B After completion of the operation for malrotation and non-descending of the cecum with volvulus of the midgut. The duodenum is exposed throughout its entire length and the cecum and appendix now lie in the left upper quadrant.

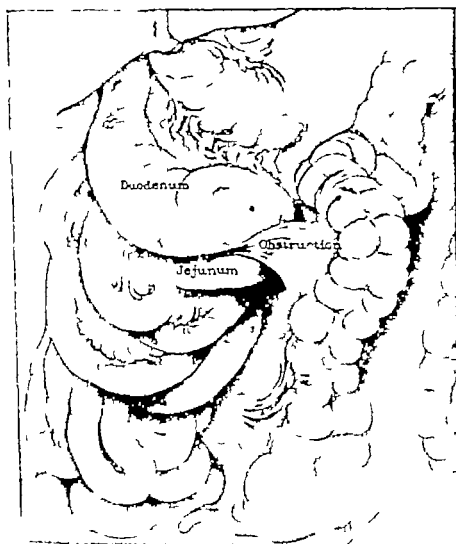


Figure 8. Malrotation of intestine with fibrous band obstructing terminal portion of duodenum.

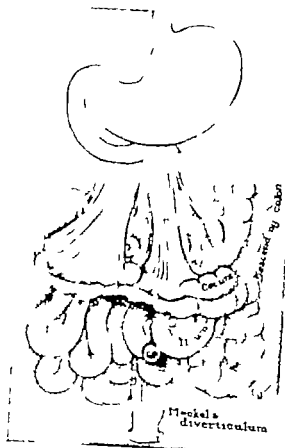


Figure 9 Malrotation of cecum with coincidental Meckel's diverticulum of ileum (Mayo, C W., and Remington, J H. *Am. J. Surg.*, vol. 72)

gical. The operation may be one of the most confusing in abdominal surgery and cannot be successfully carried out unless the embryologic development is kept in mind and the operation performed with a view to restoring normal physiologic function, regardless of normal anatomic relations.

Acidosis and dehydration are relieved preoperatively.

The method of surgical treatment to be described was first advocated by Ladd in 1933 and is the procedure of choice. In a patient without volvulus, the obstructed point is identified by the distended duodenum above and the collapsed bowel below it. The parietal peritoneum is incised, the cecum is transferred to the left upper quadrant and any constricting bands across the duodenum are severed so that the whole length of the duodenum is exposed and free. In a patient with associated volvulus of the midgut, the whole midgut must be delivered out of the wound so that the direction of the twist can be ascertained. The intestine is untwisted until no interference with circulation is apparent. The duodenum is then exposed in its entirety to insure free func-



Figure 10 Roentgenogram made after ingestion of small amount of barium in a five-year-old child. It reveals dilatation and obstruction of the duodenum caused by fibrous bands secondary to incomplete rotation of the colon.

tion and reduce the chances of recurrence. The cecum is usually placed in the left upper quadrant.

In dealing with these patients with midgut volvulus, it must be remembered that reduction of the volvulus is only a part of the procedure necessary for cure. The abnormally placed cecum must be well freed from the right and toward the midline so that the entire duodenum is exposed. Experience has shown that reduction of the volvulus alone will almost invariably result in a recurrence of the symptoms.

In our series, however, there have been two patients in whom a volvulus of the entire small bowel below the ligament of Treitz, without involvement of the ascending colon, had taken place and was made possible by an abnormally long mesentery. In such patients there has been no malrotation or nondescent of the cecum itself. In the treatment it is not only unnecessary but inadvisable to free the right colon and expose the duodenum. In these rare instances of volvulus of the whole small bowel, the duodenum is not obstructed, but the obstruction occurs below it, in contrast to midgut volvulus associated with malrotation and nondescent of the cecum.

Since it is usually unwise to remove the

appendix at the time of the original operation, especially in those patients having associated volvulus it must be kept in mind that appendicitis developing in these patients may produce quite unusual physical manifestations. In one child in our series who developed appendicitis some years following the operation for malrotation, the pain and tenderness were in the left epigastrium and it was from this region that the inflamed appendix was removed.

In the postoperative care, problems concerned with fluid balance must be uppermost in the surgeon's mind. Normal peristalsis ordinarily returns relatively soon after release of the obstruction and improvement may be striking. An indwelling intubation tube should be attached to a suction apparatus and checked frequently to be certain that the suction is working properly and that thick material within the intestine is not plugging the tube. Gentle irrigation of the tube may be required. When the surgeon is satisfied that peristalsis is normal and effective beyond the previously obstructed point, he may gradually withdraw the tube, since its prolonged use may result in esophagitis, difficulties in the eustachian tube and other unpleasant sequelae. Bowel movements should be established in a relatively normal fashion by the fourth postoperative day or sooner. Gentle enemas of saline solution may be of some help. Early feeding of properly selected foods is allowed when the surgeon is satisfied that flatus is being expelled properly.

In earlier years, the mortality rate after this operation was forbidding, but more recently a decidedly favorable change has been noted. Since sepsis usually does not play a particularly large part in this type of obstruction, as the intestine need not be opened at any time the deciding factors have been those of fluid balance and chemical replacement. The most distressing feature about this condition has been the relatively high rate of recurrence as there is no reliable method of attaching the intestine or taking steps at the time of operation to guarantee that the patient will not experience recurrence. Gangrene of the intestine has been a large factor in the mortality rate associated with this condition.

In the treatment of 106 patients with malrotation in whom Ladd's operation was used, there was an 83 per cent survival rate in the group having no other congenital anomaly. There was a 43 per cent survival rate

in those with malrotation associated with some other abnormality.

MECKEL'S DIVERTICULUM

The vitellointestinal or omphalomesenteric duct is the communication between the midgut and the yolk sac during the first few weeks of fetal life. Persistence of this duct, or parts of it, in postnatal life results in various anomalies, one of which was first described by Meckel in 1815 and has since been known as Meckel's diverticulum (Fig. 11). Meckel's diverticulum arises from the free, or antimesenteric, portion of the ileum, about 20 inches above the ileocecal valve. The persistence of this duct is estimated to occur in from 2 to 4 per cent of infants.

Though a Meckel's diverticulum may be present throughout life without causing symptoms, it is subject to a wide variety of pathologic changes and must always be considered a potential source of danger. It may remain attached to the umbilicus with an unobstructed opening or intestinal fistula, it may remain attached to the umbilicus, without an unobstructed opening, it may become detached from the umbilicus and hang loose in the abdominal cavity or its tip may become attached to some other point in the peritoneal cavity and cause intestinal obstruction. It may become the site of acute inflammation, or diverticulitis, in many instances it contains aberrant gastric mucosa and is prone to show ulceration with hemorrhage or perforation, and, in some instances it inverts itself into the lumen of the ileum and acts as the advancing point of an intussusception (Figs. 12 and 13).

The symptoms caused by Meckel's diverticulum are variable according to the type of developmental error and the pathologic conditions arising from it. The unobstructed opening at the umbilicus gives the inconvenience and annoyance of any fecal fistula, with the difficulty of taking care of the fecal discharge and the irritation or digestion of the surrounding skin. The diverticulum which remains attached to the umbilicus may give intermittent abdominal pain of rather mild character referred to the umbilicus or more severe pain associated with vomiting, abdominal distention and visible peristalsis—in short, the signs and symptoms of intestinal obstruction.

The diverticulum which is not attached to the umbilicus may become adherent to almost any point in the abdominal cavity across a loop of intestine and cause intes-

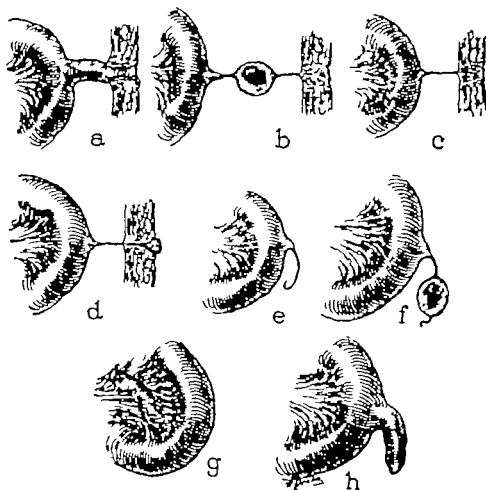


Figure 11 a, Persistent omphaloenteric duct with umbilical fistula. b, Incomplete closure of duct with midportion presenting a cyst. c, Closure of duct with persistent fibrous remnant. d, Closure of duct with umbilical cyst persisting. e, Same as c without attachment to umbilical region. f, Same as b without attachment of Meckel's diverticulum almost covered by mesentery. h, Typical appearance of Meckel's diverticulum (Dixon, C. F. and Steward, J. A. S. Clin. North America, vol. 12)

tinal obstruction. When a Meckel's diverticulum is the site of acute inflammation, the signs and symptoms are similar to those of acute appendicitis and the erroneous diagnosis of appendicitis is frequently made. The importance of hemorrhage from the bowel as a symptom of Meckel's diverticulum must be emphasized.

Massive hemorrhage from the gastric mucosa in a Meckel's diverticulum is far more common than was once thought and is particularly likely to occur in small infants. In ten of the last thirty-one cases at the Children's Hospital, massive hemorrhage from the bowel was the presenting symptom. One of these patients was six years old, one was one year old and the other eight were less than a year old. Bleeding in varying degrees occurs in over 60 per cent of the subjects. If the ulceration perforates, the signs and symptoms are those of peritonitis and are

frequently preceded by a history of blood in the stools. If a Meckel's diverticulum is inverted into the lumen of the intestine, causing intussusception, the symptoms and signs are, of course, those of intussusception.

The treatment of Meckel's diverticulum is surgical and is aimed at relieving the variety of conditions which this appendage causes. Patients who have a massive hemorrhage should have immediate transfusion and operation. If the diverticulum is inflamed, early operation is indicated to prevent free perforation just as in acute appendicitis. If intestinal obstruction is present, it must be relieved and when intussusception is found, that must be reduced. It is desirable to remove the diverticulum in all subjects except when there are conditions which contraindicate this procedure at the time of the first operation. Removal should be done through a transverse incision in the ileum at the base

of the diverticulum and the incision should be sewed up transversely so as not to obstruct the lumen of the ileum. It is important that the entire diverticulum be removed as a common location for ulceration is at its base. There is always a temptation to remove a diverticulum as one would an appendix by tying its base and inverting its stump with a purse-string suture. This procedure should be condemned because of the probability that intestinal obstruction will result

from the narrowed lumen and because some of the diverticulum may be left.

In certain conditions particularly those in which the Meckel's diverticulum is the cause of intussusception it may be inadvisable to remove the Meckel's diverticulum at the time of the original operation. Each case must be judged on its own merits, but if the diverticulum is not removed at the first operation it should be removed later when conditions are more favorable. If bowel resection is needed, and this is especially likely in intussusception, the Mikulicz procedure is the one of choice. We have found that this operation is far safer in the over all results than is primary resection followed by anastomosis.

Of the last 149 cases of Meckel's diverticulum at the Children's Hospital including all types 128 of the patients survived, the mortality rate being 14 per cent. Since 1940 there have been seventy-one of these patients with sixty six survivors, the mortality rate being 7 per cent.

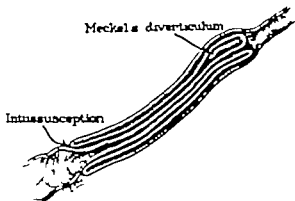


Figure 12. Meckel's diverticulum producing classic intussusception of small intestine (Mayo, C. W., Miller J. M., and Stalker L. K. *Surg. Gynec. & Obst.*, vol. 71)

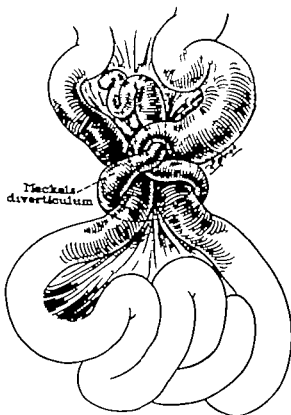


Figure 13. Greatly elongated Meckel's diverticulum with complete twist and formation of knot, obstructing ileum (Mayo, C. W., and Remington, J. H. *Am. J. Surg.*, vol. 72)

DUPLICATIONS OF THE ALIMENTARY TRACT

The term "duplications of the alimentary tract" is used in the hope of simplifying the nomenclature and because it more accurately describes the condition found than do other designations. Some of the terms employed to describe the same condition are mesenteric cyst, enteric cyst, enterogenous cyst, diverticulum, giant diverticulum, ileum duplex, jejunum duplex and unusual Meckel's diverticulum.

It is now well established that although these malformations vary greatly in location, size and shape and may or may not communicate with the lumen of the bowel itself they all present certain common characteristics.

After the solid stage in the development of the gastrointestinal tract, multiple vacuoles appear within the solid mass. These normally coalesce later so that the normal single hollow tube of the alimentary tract is formed. When duplications occur Bremer believes that they are the result of the continued development of some of these vacuoles outside of the bowel. The duplications may or may not communicate with the lumen of the bowel itself.

Even though the duplications vary greatly in size and shape, their walls are at some point closely attached to the alimentary tract itself and, in the vast majority their blood supply is connected with and is a continuous

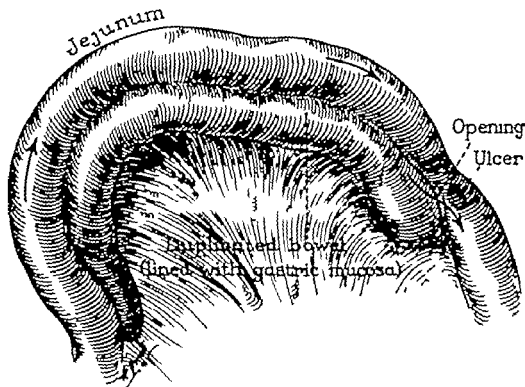


Figure 14 Duplication of small intestine (Johnston, J. B., Hallenbeck, G. A., Ochner, A., and DeShazo, J. W. *Arch. Surg.*, vol. 68)

part of the blood supply of the adjacent intestinal canal. Duplications of the alimentary tract may occur anywhere from the base of the tongue to the rectum, but one-half are in the ileum itself (Fig. 14). Their walls have a serous coat with a mucous lining and a well-developed coat of smooth muscle. These bowel-like walls are easily recognized and help to differentiate the mass from the true lymphatic or chylous cysts. Lymphatic cysts are seen in the mesentery of the bowel and do not have a common blood supply with the bowel itself. If the duplication does not communicate with the lumen of the bowel, the contained fluid is usually a clear mucoid material secreted by the membranous lining, if it does communicate with the bowel, the contents are characteristic of those of the particular segment of the bowel involved.

A section through the wall will show a serous layer, a muscular layer and a layer of mucous membrane, as in the alimentary tract. The mucous membrane may be like that of the colon, the stomach, the duodenum or the ileum. The contents of the cyst may be mucoid fluid, sometimes chocolate colored and sometimes almost colorless. In some instances the fluid is sterile but it may contain pus cells or colon bacilli. The chemical content of the fluid has been found simi-

lar to the succus entericus found in an isolated loop of bowel.

The symptoms vary according to the size and the situation of the malformation. When the cyst is in the chest, it causes coughing, cyanosis or dyspnea. If it is in the abdomen, there is pain and symptoms of acute or chronic intestinal obstruction may be evident. Hemorrhage from a peptic ulcer of the lining of the duplication may take place and may be sufficiently profuse to be fatal. Perforation with attending peritonitis has also occurred. A correct preoperative diagnosis is seldom made. It may be difficult to differentiate between this condition and Meckel's diverticulum, intussusception, an omental or mesenteric cyst and intestinal obstruction resulting from other causes.

In all our patients in whom duplication occurred in the abdomen, vomiting was a prominent symptom. Wild pain or abdominal discomfort was present in all and under nourishment or feeding difficulties were usually a part of the picture. Most of these patients had been under treatment because of "feeding problems."

The blood found in the stools may come from a peptic ulcer next to misplaced gastric mucosa in the lining of the duplication or from congestion of the intestine by pressure of the duplication. The tumor can usually be

felt and is likely to be rather more mobile than is a tumor of some other origin. Roentgen ray examination of the abdomen may be helpful but often is not. In one instance it showed free air in the peritoneal cavity denoting perforation. Once it showed pressure on the stomach and on the colon. It may demonstrate partial obstruction which, of course, can be diagnosed by other means.

From our experience, duplication of the alimentary tract should be suspected in a child suffering from symptoms of partial intestinal obstruction in whom a freely movable tumor can be felt. It should be suspected also when blood is found in the stools, if the symptoms are less acute than those of intussusception and if the tumor is larger than would be expected of a Meckel's diverticulum.

The treatment of this condition is, of course, surgical. Because of the danger of obstruction, perforation or hemorrhage the operation should not be unduly delayed. The operative technique is of importance and the operation usually involves resection of the loop of the bowel to which the duplication is attached.

It is a temptation for the surgeon who is confronted with his first patient having this anomaly to attempt to dissect the duplication away from the normal bowel. It is unwise to do this except in the rare patient in whom the duplication is detached from the normal bowel and has a mesentery of its own. Usually the blood supply of the duplication and the blood supply of the bowel to which it is attached are the same and, if one dissects the duplication, the blood supply of the bowel is destroyed. Another factor which makes it unwise or indeed impossible, to dissect the duplication from the normal bowel is that there is no line of cleavage between the two. The partition between the duplication and the normal bowel consists of two layers of mucous membrane with the muscle layers between. It is almost impossible to effect separation without perforating the lumen of one or both structures.

If operation is undertaken prior to the occurrence of infection, perforation or severe hemorrhage, the results have been increasingly good. Of the last sixty-eight duplications treated at the Children's Hospital thirty-five were in the duodenum, jejunum or ileum. The mortality from 1928 to 1940 was 27 per cent, but in the last twenty patients there have been no deaths.

OMENTAL CYSTS

Congenital cysts of the omentum are probably caused by obstruction of lymphatic channels. They are thin walled, usually rounded, may be small and embedded within the omentum itself or they may attain a huge size.

There is a history of a slowly enlarging abdomen usually without pain and on abdominal examination the cyst may be moved from side to side and there is evidence that it contains fluid. The treatment is simple and involves only excision. If the cyst is extremely large the entire omentum may have to be removed, but it is usually possible to dissect out the usual cyst and preserve a part or all, of the omentum itself.

MESENTERIC CYSTS

These may arise in any part of the mesentery or the mesocolon and, like omental cysts they may be enormous. They grow slowly and produce little in the way of symptoms unless when large they give moderate abdominal pain associated with vomiting. As they become large there may be some loss of appetite and failure to gain weight. Rarely the symptoms of acute intestinal obstruction occur particularly if the cyst is in the free border of the mesentery where by greater mobility it may compress or angulate the bowel. The cyst, if large, can usually be palpated as a soft, fairly well-defined fluctuant mass. As the usual location is in the jejunum or ileum, this mass, when so located, is more freely movable in a lateral than in a vertical direction as compared with an omental cyst.

Unlike duplications, these cysts can usually be safely dissected out from the mesentery itself and this is the preferred method of treatment. There is not a common blood supply to these cysts and the adjacent bowel and a good line of cleavage is usually found. The cysts can be dissected from the intestine or colon without impairing the blood supply to the bowel. After removal, there may be a large defect in the mesentery which should be closed with interrupted sutures. The results are very good.

ANNULAR PANCREAS

Constriction of the second portion of the duodenum may be caused by a maldevelopment of the pancreas. The degree of obstruction will vary with the extent of this anomaly. The head of the pancreas is in its usual position, but two branches or arms of pancreatic tissue enfold the duodenum at this

point. These arms are usually fused and completely encircle the duodenum. The symptoms are the same as those of congenital intrinsic stenosis in this area. This anomaly is not infrequently associated with other congenital defects and, if so the prognosis will depend to a large extent on the seriousness of the associated defects.

Annular pancreas is best treated by establishing a duodenojejunostomy. This method has been used successfully since 1942 and it appears to be the ideal form of surgical correction. When done properly it relieves the duodenal obstruction without interfering with the normal function of the stomach and the biliary tract and it does not include the dangers of cutting pancreatic tissue. Of the last ten patients so treated in our hospital, six are alive and well, four died, but death in three of these was caused primarily by other anomalies.

MECONIUM ILEUS

It is now recognized that intestinal obstruction in the newborn caused by impacted meconium is the result of a generalized disease. There is a marked disturbance and deficiency in the secretion of the pancreatic enzymes and also an abnormality of the mucus-secreting glands in both the alimentary and respiratory tracts. It is important to realize that in addition to the disturbance of pancreatic function which has been appropriately called fibrocystic disease of the pancreas and which results in these abnormal features in the meconium, there are also complications resulting from abnormal secretion of mucus elsewhere in the body which has been termed mucoviscidosis. This mucus is abnormally thick and viscid and often leads to severe pulmonary complications.

The intestinal symptoms caused by meconium ileus are similar to those of intestinal obstruction from other causes. The vomiting begins early and is of a progressive type. The vomitus is at first clear but later becomes dark, as abdominal distention progresses. In this condition, one of the important and characteristic physical findings is the presence of a firm mass or masses palpable within the bowel itself, these masses being the thick, inspissated meconium.

X ray findings are often characteristic. Obstruction will be demonstrated in the lower ileum, but the proximal distended loops will vary greatly in the degree of distention. This variation in size is seldom found to be striking in the patient with an ordinary in-

testinal obstruction. The x-ray picture of the impacted meconium itself is quite characteristic. The mottled or granular appearance is seldom noted in subjects with intestinal obstruction of other causes. Although the above characteristics are not always present, the evidence of intestinal obstruction is usually so clear that the indications for operative treatment are unmistakable.

There are occasional patients in whom the evidence of obstruction is not severe and it is sometimes possible in these subjects that the condition will resolve itself by the use of cleansing enemas and the giving of pancreatic enzymes by mouth. But the great majority require surgical treatment. After many and discouraging methods of surgical attack, the present treatment of choice is a Mikulicz resection with a double-barreled ileostomy. It is necessary to resect the greatly distended portion of the terminal ileum, for it is in this area that the meconium is greatest in amount and is thickest in character. The double-barreled ileostomy allows decompression of the intestine, permits flushing out of the terminal ileum and colon and the giving of pancreatic enzymes. By this method an increasing number have been relieved successfully of their obstruction and it is hoped that the results will be even better in the future. It is important, however, to remember that the intestinal obstruction is only part of the picture. The child has a severe and chronic nutritional disturbance resulting from the pancreatic insufficiency and has frequently severe pulmonary damage resulting from the generalized mucoviscidosis. The poor nutrition in these children is best compensated for by the addition of a fortified casein hydrolysate such as Nutramigen. This is added to the formula. The caloric value in the diet must be increased to about 50 per cent above that offered a normal infant of the same age. The vitamin intake should be high and it is desirable to use vitamins which are water miscible. It is seldom necessary to keep the ileostomy stoma open more than three weeks at which time the crushing clamps can be used on the septum and thus intestinal continuity is re-established. Later the small ileostomy fistula may be safely and simply repaired surgically. These patients, however, demand a long period of careful medical observation through the following months and years.

Of twenty two patients successfully operated upon in recent years, eight have succumbed to a severe respiratory distur-

or in pulmonary infection, or both within a few months after operation.

HINDCHAPRINGS DOBZOE

A recent view there have been changes of fundamental importance in the concept and treatment of this disease. The work of Swenson and others has clearly shown that the true pathological process of this disease is found in the intestinal portion of the retroperitoneum and not in the cecum and hyperextended part of the large bowel.

The present concept of the disease is supported by macroscopic examination of the bowel wall, a thin or a constriction in the distal ends of the intestinal portion in this case involved area in the retroperitoneum. There is close adherence with normal peritoneum in this area and this causes in effect an area of constriction. The membranes hyperplastic in dilatation above it is the result of Nature's effort to force the fecal stream through this area where there is no true peristaltic action. The present-day treatment then is aimed toward the removal of this abnormal and narrow area instead of procedures directly concerned with the dilated portion itself.

As Hirschsprung's disease is a true congenital lesion, symptoms characteristically appear shortly after birth or in early infancy. This helps in differentiating the disease from the acquired megacolon seen in older children. In the latter condition, although there may be a compensatory dilatation and hypertrophy of the large bowel, the symptoms of chronic constipation usually do not appear until after the second year of life.

Hirschsprung's disease seems to be far more common in boys than girls and the history is that of long-continued, progressive and constant constipation. With this there is marked and progressive enlargement of the abdomen. There is evidence of a very poor nutritional state and, in extreme cases, there may be interference with breathing and even circulatory embarrassment caused by the increasing upward displacement of the diaphragm. Vomiting and pain are seldom encountered, but it is well to remember that there may be attacks of "diarrhea." This paradoxical character occurs because only the fluid contents of the lumen can be passed around the solid impacted fecal masses in the lumen. On rectal examination there is absence of contraction or stool-passage and in this area there is material, even though be felt higher up. It is

important to note that mucus or this soft consistency appeared in Hirschsprung's congenital lesion. The clinical picture varies a great deal, but the usual case is progressive and as operation and treatment is now so effective, it is advisable for most patients before an hour's time has elapsed.

Very examination should be made with some caution. A thin section of the intestine is advisable. The diagnosis is often suggested by a plain film roentgen shows the markedly dilated area, much air and impacted fecal material. Examination following a cautious enema will show visualization of the barium in the "pouches" of the lower bowel in the oblique view and it is seen that the narrowed character of the lesion is usually found. Views taken only in the anteroposterior plane will not demonstrate this area because of the overlying distended bowel.

Excision of the abnormal area with an anastomosis of the pull-through type of operation has given extremely promising results. In seventy patients operated there were two deaths, but the surviving surviving subjects have been followed for periods of from one to five years. They have excellent bowel control, take a normal diet and have progressed normally in growth and development. In two patients there was transverse hypospadias of the clitoris. This may have been the result of the operative procedure itself. As there may be some associated structural defect in the bladder of these patients, it is important to evaluate the bladder function in all subjects before operation, particularly in regard to the presence or absence of residual urine.

MALFORMATIONS OF THE ANUS AND RECTUM

Malformations of the anus and rectum are varied in type and are frequent enough to be of considerable importance. The variation in type depends on when and where the arrest in development of the primitive gut occurred and is intimately associated with the development of the urogenital system. In the early embryo the anus is a cavity which includes both the intestinal tract and the urogenital tract. These two become separated by a transitory of mesoderm and this separation is normally completed at about the seventh week. In the further development, there is a division in this area into the urogenital membrane anteriorly and the anal membrane posteriorly. About the time of separation, there appears an external

point. These arms are usually fused and completely encircle the duodenum. The symptoms are the same as those of congenital intrinsic stenosis in this area. This anomaly is not infrequently associated with other congenital defects and, if so, the prognosis will depend to a large extent on the seriousness of the associated defects.

Annular pancreas is best treated by establishing a duodenojejunostomy. This method has been used successfully since 1942 and it appears to be the ideal form of surgical correction. When done properly it relieves the duodenal obstruction without interfering with the normal function of the stomach and the biliary tract and it does not include the dangers of cutting pancreatic tissue. Of the last ten patients so treated in our hospital, six are alive and well, four died, but death in three of these was caused primarily by other anomalies.

MECONIUM ILEUS

It is now recognized that intestinal obstruction in the newborn caused by impacted meconium is the result of a generalized disease. There is a marked disturbance and deficiency in the secretion of the pancreatic enzymes and also an abnormality of the mucus-secreting glands in both the alimentary and respiratory tracts. It is important to realize that in addition to the disturbance of pancreatic function which has been appropriately called fibrocystic disease of the pancreas and which results in these abnormal features in the meconium, there are also complications resulting from abnormal secretion of mucus elsewhere in the body which has been termed mucoviscidosis. This mucus is abnormally thick and viscid and often leads to severe pulmonary complications.

The intestinal symptoms caused by meconium ileus are similar to those of intestinal obstruction from other causes. The vomiting begins early and is of a progressive type. The vomitus is at first clear but later becomes dark, as abdominal distention progresses. In this condition, one of the important and characteristic physical findings is the presence of a firm mass or masses palpable within the bowel itself, these masses being the thick, inspissated meconium.

X-ray findings are often characteristic. Obstruction will be demonstrated in the lower ileum, but the proximal distended loops will vary greatly in the degree of distention. This variation in size is seldom found to be striking in the patient with an ordinary in-

testinal obstruction. The x ray picture of the impacted meconium itself is quite characteristic. The mottled or granular appearance is seldom noted in subjects with intestinal obstruction of other causes. Although the above characteristics are not always present, the evidence of intestinal obstruction is usually so clear that the indications for operative treatment are unmistakable.

There are occasional patients in whom the evidence of obstruction is not severe and it is sometimes possible in these subjects that the condition will resolve itself by the use of cleansing enemas and the giving of pancreatic enzymes by mouth. But the great majority require surgical treatment. After many and discouraging methods of surgical attack, the present treatment of choice is a Mikulicz resection with a double-barreled ileostomy. It is necessary to resect the greatly distended portion of the terminal ileum, for it is in this area that the meconium is greatest in amount and is thickest in character. The double barreled ileostomy allows decompression of the intestine, permits flushing out of the terminal ileum and colon and the giving of pancreatic enzymes. By this method an increasing number have been relieved successfully of their obstruction and it is hoped that the results will be even better in the future. It is important, however, to remember that the intestinal obstruction is only part of the picture. The child has a severe and chronic nutritional disturbance resulting from the pancreatic insufficiency and has frequently severe pulmonary damage resulting from the generalized mucoviscidosis. The poor nutrition in these children is best compensated for by the addition of a fortified casein hydrolysate such as Nutramigen. This is added to the formula. The caloric value in the diet must be increased to about 50 per cent above that offered a normal infant of the same age. The vitamin intake should be high and it is desirable to use vitamins which are water miscible. It is seldom necessary to keep the ileostomy stoma open more than three weeks at which time the crushing clamps can be used on the septum and thus intestinal continuity is re-established. Later the small ileostomy fistula may be safely and simply repaired surgically. These patients, however, demand a long period of careful medical observation through the following months and years.

Of twenty-two patients successfully operated upon in recent years, eight have succumbed to a severe nutritional disturbance.

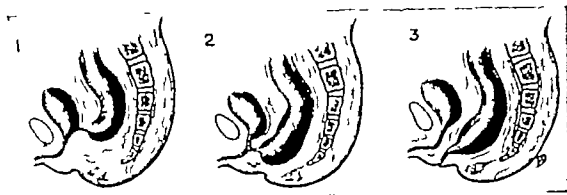


Figure 16. Types of fistulae encountered in male patients. 1 Rectovesical fistula. 2, Rectourethral communication. 3, Rectoperineal fistula (the opening being in front of the area where the anus should normally open) (Gross: *The Surgery of Infancy and Childhood*)



Figure 17. Types of fistulae encountered in female patients. 1 Rectovaginal fistula. 2, Recto-fossa navicularis fistula. 3, Rectoperineal fistula (Gross: *The Surgery of Infancy and Childhood*)

paired from below. If it is higher than that, it can usually not be repaired from below and, in such instances, sigmoidostomy can be done and the malformation may be repaired later when the child is one or two years of age. This procedure is probably the safer one if the facilities of the hospital or the experience of the surgeon is not ideal. But with the proper facilities and a surgeon who is familiar with this type of work, it is our opinion that such patients, if in good condition, should be treated immediately by the combined abdominoperineal approach.

As noted above, there is a very high incidence of associated fistula either with the skin or with the genitourinary tract. These complications are most likely to occur in association with the type 3 and type 4 anomalies and there is a much higher incidence of rectourethral and rectovesical fistulae in the male than in the female. Commonly the associated fistulae in the female are those to the perineum just anterior to the anus or to the vagina. The presence of these fistulae in the male is readily recognized in the usual case. If the fistula communicates with the perineum, it is seen that the meconium comes out in small quantities anterior to the anal pit. If it communicates with the bladder or urethra, meconium will

be noted in the urine. The most frequent error in early diagnosis in the female is that of a fistula which empties close to, or actually into the vagina. In such patients, meconium may appear to be coming normally from the rectum. The temperature, supposedly taken by rectum is actually taken by inserting the thermometer in the fistula.

These complicated anomalies were poorly handled in the past, but a recent review shows that a satisfactory result can be obtained in well over 90 per cent of the patients and that the anus will function normally. It has also been possible to cure satisfactorily in a single operation well over 90 per cent of the patients with associated high fistulae. However it is much better if ideal facilities are not available, to do a temporary sigmoidostomy. It is extremely difficult to do a combined abdominoperineal repair in a patient who has had a previous unsuccessful operation. It is also important to call attention to the fact that anomalies of the anus and rectum are found to have other associated anomalies in about 40 per cent of the patients. In many instances another anomaly was the cause of death, among these fatal conditions being congenital heart disease, atresia of the intestine and atresia of the esophagus.

opening for the urogenital sinus and posteriorly there is an in-pocketing at the anal pit, which continues until the proctodeum meets the rectum. When the anal membrane ruptures at about the eighth week, there is a continuous passage from the hindgut to the outside.

As will be seen in Figure 15, there are four main types of anomaly and the treatment of these four types will be briefly outlined later. It is important to remember that in all types the anal sphincter muscle develops from the regional mesenchyma and is therefore not dependent on the maldevelopment of the bowel itself. This is fortunate because the sphincter muscle is of great importance in the repair of these anomalies.

Unfortunately imperforate anus of the four types shown in Figure 15 is, in over 70 per cent of the cases, associated with a communication between the bowel and either the perineum or the genitourinary system. Figures 16 and 17 illustrate the common types of associated fistulae found in the male and in the female. The symptoms will vary greatly and will depend on the type of anomaly and, more important, on whether or not there is an associated fistula.

In patients without fistulae the symptoms will depend on whether there is merely stenosis or whether there is, in fact, an imperforate anus. Those with type 1 abnormality have symptoms which will vary with the degree of stenosis and in some this stenosis does not give very marked symptoms until weeks or months following birth, when the bowel contents become more solid. In types 2 and 3 abnormalities there is of course, complete obstruction and the symptoms are obvious. Every newborn child, however, should be carefully examined at birth to see whether the anus is perforate and, if not, early and effective treatment

can be started. It is only in type 4 abnormality in which there apparently is an anal opening, that the true condition is likely to be overlooked. In our series, for example, those with type 2 and type 3 abnormalities are seen within the first twelve hours of life, whereas those with type 4 may be brought to the hospital on the third or fourth day, sometimes even later.

In patients with type 1 abnormality, repeated dilations are usually enough to establish normal function. Dilatation is best done at first with graded metal dilators, later the little finger can be employed with great advantage. Dilatation can be done by the mother if she is properly instructed. It is of great importance to keep these patients under observation for at least two years.

In patients with type 2 abnormality the treatment is the same except that the membrane which closes the opening of the bowel must be incised. Incision alone is not enough and frequent observation with necessary dilations must be insisted on.

The treatment of patients having type 3 abnormality depends on the distance the blind pouch is from the anal skin. This distance can readily be determined by making an x ray examination of the abdomen and pelvis with the patient head-down and with a small metal marker on the anal pit. This will show how far the gas in the obstructed bowel is from the skin itself. It must be pointed out that in such an examination the gas within the bowel may not have distended the bowel to its most distal point until after eighteen or twenty-four hours following birth. After this time the x ray film will be a very accurate method of determining the position of the blind pouch. If the blind end is within 1.5 cm. of the anal pit, it can in most instances be successfully re-

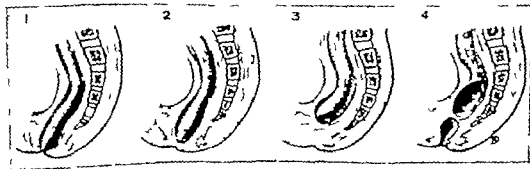


Figure 15. Types of anal and rectal abnormalities. Type 1 Stenosis at the anus. Type 2, Imperforate anus. Obstruction only by a persistent membrane. Type 3, Imperforate anus. Rectal pouch ending blindly some distance above anus. Type 4 Anus and anal pouch normal. Rectal pouch ends blindly in hollow of sacrum (Gross: *The Surgery of Infancy and Childhood*)

of sufficient of these vessels to carry a good supply of blood to the cut edge. Special mention should be made also of the relation of the esophagus to the thoracic duct because of the serious prognosis of chyloous hydrothorax which results from an unrecognized injury to that structure.

The venous system of the esophagus, with the exception of the lower portion empties by way of the hemiazygos and azygos veins into the superior vena cava. Blood from the lower third of the organ enters the portal system through branches of the left gastric veins and the branches of the vasa brevia of the splenic vein. This communication between the systemic and portal circuits has great clinical importance in the production of varicosities of the esophageal and upper gastric veins.

The lymphatic drainage from the cervical portion and to some extent the superior mediastinal segment of the esophagus is to the large groups of lymph nodes in both sides of the neck. The drainage from the remainder is principally downward. The main lymphatic channels empty into the superior mediastinal nodes, the nodes about the hila

of both lungs the lower mediastinal nodes including those in the pulmonary ligaments the subdiaphragmatic paracardial nodes and the group which lies in relation to the branches of the left gastric vessels along the lesser curvature of the stomach (Fig. 19)

Finally it is important for the surgeon to keep in mind that the esophagus because of its fragile structure and lack of serous covering, must be handled with much greater gentleness than the stomach or intestines. Its musculature the greater portion of whose fibers are longitudinal is delicate and does not hold sutures well. The mucosa is the toughest layer of the esophagus and much of the strength of an esophageal anastomosis depends upon the integrity of this layer.

The function of the esophagus consists almost entirely in conveying food and liquids from the pharynx to the stomach. A voluntary contraction of the pharyngeal musculature and the associated muscles of the tongue and floor of the mouth initiates deglutition and forces the material which is being swallowed downward into the esophagus. An associated nerve impulse brings about relaxation of the cricopharyngeus muscle. Once the ingested matter has entered the esophagus, two factors are brought to bear upon its further passage to the stomach. The effect of gravity is most important in

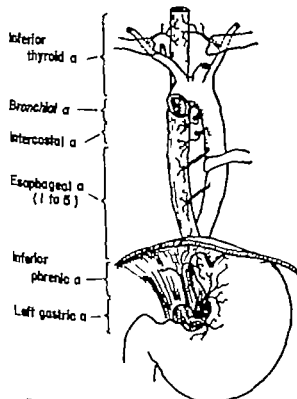


Figure 18. Diagram illustrating the blood supply of the esophagus. Note (1) that the areas of supply actually overlap, one with the other; (2) that the number of esophageal branches arising directly from the aorta is variable.

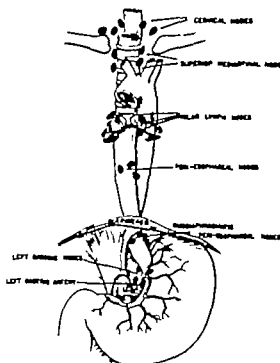


Figure 19. Diagram showing the six important groups of lymph nodes which drain the esophagus and adjacent structures.

READING REFERENCES

- Brenner J. L. Diverticula and Duplications of the Intestinal Tract. Arch. Path. 38:132, 1944.
 Bremer J. L. Congenital Anomalies of the Viscera. Cambridge, Mass., Harvard University Press, 1957

- Gross, R. Surgery of Infancy and Childhood. Philadelphia, W. B. Saunders Company 1953
 Swenson, O. Neuhauser E. B. D. and Pickett, L. K. New Concepts of Etiology, Diagnosis and Treatment of Hirschsprung's Disease. Pediatrics 4:201, 1948.

The Esophagus

By RICHARD H. SWEET, M.D.

RICHARD HARWOOD SWEET was educated at Columbia University and at Harvard Medical School where he now is on the faculty. Trained in general surgery, he has the ability to apply general surgical principles to the special physiologic and anatomic conditions found within the thoracic cavity. He has made many original contributions to esophageal surgery.

Progress in the surgical management of disorders affecting the esophagus is a modern development. Although several factors were involved in the delay, the most significant was the failure until recent years to overcome the difficulties presented by the necessity of operating through the open thorax. Of particular importance was the solution of the problems of maintaining control of the expansion of the lung and of securing an adequate degree of oxygenation of the blood during the operative procedure and the early recovery period. The resolution of these difficulties is the result of the utilization of knowledge of the physiology of the chest, which has made it possible to approach the esophagus with as much safety as any other portion of the alimentary tract.

Other difficulties which had to be overcome have yielded to the application of knowledge accumulated from many sources and applied first in other branches of surgery. The great progress which has been made in the field of nutrition has made it possible to overcome to some extent the effects of anemia and emaciation which result from the starvation produced by an obstructing lesion. As a result, many patients whose condition would formerly have been considered a contraindication to surgical treatment can now be operated upon successfully. Furthermore, the utilization of this knowledge has improved the management of the

postoperative period so as to minimize the incidence of complications.

Of great importance likewise in the progress of esophageal surgery is the almost complete elimination of the element of infection, which formerly was responsible for the majority of complications and postoperative deaths. This result has been brought about by the routine use of appropriate antibiotics in addition to the improvements in operative technique and the general management of the case as a whole.

For the surgeon, a thorough knowledge of the topographic relations of the esophagus is important. The esophagus in its course from the pharynx to the cardiac orifice of the stomach traverses three important regions of the body, the neck, the thorax and the abdomen. In its course it assumes an important relation to such structures as the trachea, the thyroid gland, the aorta, the great vessels as they ascend into the neck, the pericardium, the inferior pulmonary veins, the azygos and hemiazygos veins, the vagus nerves with their recurrent laryngeal branches, the thoracic duct, the diaphragm and the vertebral column.

Of vital importance to the surgeon likewise is realization of the fact that the arterial blood supply of the esophagus (Fig. 18) is strictly segmental in distribution, so that when a resection is performed, adequate provision can be made for the preservation

such as this holds enormous quantities of food and emits only small amounts at a time into the stomach. Patients sometimes discover that by straining, or with changes in position, and often by taking large draughts of liquid, they can force some of the food down. But pain resulting from spasm is a rare occurrence.

In the remaining one-third of patients the esophagus, though dilated, never assumes the gigantic proportions of the first type. Furthermore, in sharp contrast with the first type in which the muscle layer at the distal end is atrophic, the lower segment is not much narrowed and shows a striking degree of hypertrophy of the circular muscle fibers. Above this level the esophagus though much larger than normal, never reaches the enormous size of the first type. When observed under the fluoroscope instead of being atonic, it appears hyperactive but with erratic peristalsis and often antiperistaltic waves extending the entire length (Fig. 21). Patients with this form of the disease, at least in the early phases, experience cramp-like pain of esophageal reference usually initiated by eating.

In both types when the disease is of long standing, and particularly in elderly patients, repeated aspiration of stagnant esophageal contents into the trachea and bronchi may lead to pulmonary complications such as atelectasis, pneumonitis and fibrosis. Although many of the patients are able to maintain a satisfactory state of nutrition, there are those who become emaciated and require urgent treatment to relieve them.

Patients with each type of the disease have been observed over long periods with out changes in the character of the condition and there is no evidence that one is actually the precursor of the other.

As with any lesion which causes a partial narrowing of the esophagus, the degree of obstruction is frequently increased in response to emotional tensions experienced by the patient but there is no evidence that this disease is basically psychosomatic in origin. In fact the etiology is unknown. There is always found, however, a diminution or an absence of the ganglion cells of Auerbach's plexus involving the entire length of the esophagus.

This alteration in the intrinsic nervous mechanism of the esophagus is in some way responsible for the disturbed neuromuscular



Figure 20. Achalasia of the esophagus, type 1 comprising about two-thirds of the cases. Enormous dilatation, atrophic lower segment; absence of spasm or pain; no peristaltic activity.



Figure 21. Achalasia of the esophagus, type 2 comprising about one-third of the cases. Moderate dilatation, hypertrophy of circular muscle layer of the lower segment, spasm and pain experienced, peristalsis active but abnormal.

the case of liquids but for the passage of solids, active waves of peristalsis play an important role. The entry of food into the stomach may be delayed temporarily by a sphincter-like activity of the circular musculature of the lower esophageal segment. There is no true sphincter muscle of the cardia.

DIAGNOSTIC METHODS

The most frequent symptom which points to the esophagus as a source of difficulty is some disturbance of deglutition, often so slight at first that the patient may give it little consideration. Other symptoms such as eructation of material swallowed, pain, loss of weight and weakness, are late manifestations of lesions which cause obstruction to the passage of food with or without ulceration and infection. Pain arising from esophageal lesions consists of two types. The first is the result of esophagospasm at or just above a point of obstruction and is referred in a segmental manner to the substernal region, to the back and sometimes to the neck. It is the type of sensation which everyone has experienced from swallowing too large a bolus of food, with temporary lodgment at some point in the esophagus. A reasonably accurate prediction of the location of an esophageal lesion can be made from the level beneath the sternum or in the back to which the pain is referred. The second type of pain is continuous and deep and is felt within the middle of the chest or in the back. This is due to invasion of the periesophageal mediastinal tissues by tumor or to the extension of infection with the production of periesophagitis or mediastinitis.

Röntgenography Every patient who complains of dysphagia, no matter how insignificant it may seem, should be examined with a contrast medium. Examination of the esophagus while barium is being swallowed frequently leads to enough suspicion to make further investigation by esophagoscopy imperative, if it does not prove the presence of esophageal disease at once. It is important, however, that the physician who refers the patient to the roentgenologist should make it clear that a disorder of the esophagus is suspected. Otherwise attention is likely to be concentrated on the stomach and duodenum after a cursory inspection of the descent of the barium through the esophagus.

Esophagoscopy By means of direct visualization of the interior of the esophagus, a definite diagnosis can often be made when

esophageal disease is suspected. This possibility is further enhanced by the fact that bits of tissue from suspicious-looking areas can be removed for histologic examination. An esophagoscopic examination should be advised in every patient in whom the clinical history points to an esophageal lesion, especially if the roentgenographic findings are inconclusive or negative. If this precaution is not observed, the opportunity to establish an early diagnosis, particularly of carcinoma, may often be missed. The performance of esophagoscopy and the use of the esophagoscope for local treatment or as a means of directing the passage of bougies for dilation of a stricture require training, skill and experience.

MEGASOPHAGUS (ACHALASIA)

Idiopathic dilatation and hypertrophy of the esophagus, the most frequent disorder of this organ after carcinoma, is commonly and erroneously spoken of as cardiospasm because of the false concept that the condition results from muscular spasm at the cardia. The condition however is in fact a disease of the esophagus and has nothing to do with the gastric cardia. Furthermore, it is not primarily due to muscle spasm nor is spasm an outstanding characteristic in every instance. It may occur in individuals of any age. It has been observed in infants, but it may not become obvious until old age. In the majority of patients however it becomes troublesome in early adult life.

Two types can be recognized by the differences in their clinical manifestations, the roentgen ray findings and the anatomic characteristics as demonstrated at the operating table. In the more common variety comprising approximately two-thirds of the cases the esophagus except at the lower end is enormously dilated and hypertrophied. In the distal few centimeters, however there is a striking narrowing with a diameter often no greater than that of the little finger. In this narrow segment the muscular layer is thin and atrophic, in sharp contrast with the hypertrophy of this layer above.

The roentgen ray examination in patients with dilatation of this type portrays the narrowed lower end of the esophagus and the large dimensions of the major portion of the organ which will often hold a quart or more of liquid (Fig. 20). In addition, when observed under the fluoroscope, the esophagus is found to be atonic with almost complete absence of peristaltic activity. An esophagus

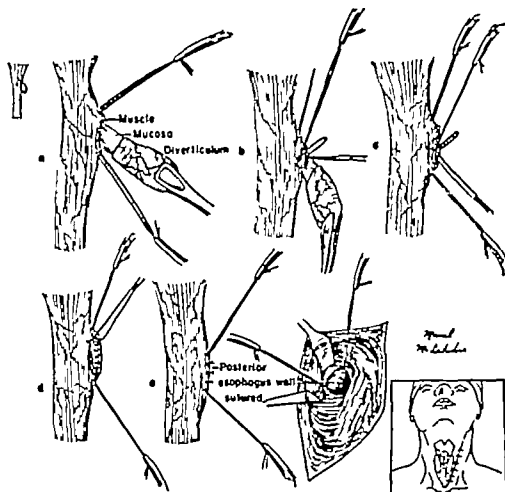


Figure 23. A diagram of the steps in excision of a diverticulum of the pharyngoesophageal junction and closure of the resulting defect. a, Incision through the muscular layer completed. The mucosa is not yet cut; stay sutures placed at the upper and lower ends of the muscle incision. Note the retraction of the muscularis. b, Incision of the mucosa is begun. The first mucosal suture is tied, the second suture is in place but not yet tied. c, The diverticulum is removed; mucosal suture is continued. d, Closure of the mucosal layer is completed. The first suture in the muscle layer is in place but not yet tied. e, Closure of the muscle layer is completed. In situ view of the field of operation before closure of the wound.

operation may still be of value for an occasional patient.

Pulsion or true diverticula developing in the lower esophagus are of rare occurrence. They emerge through a weak point in the muscular coats just proximal to the sphincter-like lower mediastinal segment of the esophagus. Thus they lie several centimeters above the level of the diaphragm, a fact which gives rise to the frequently used designation "epiphrenic diverticulum." Symptoms consist chiefly of dysphagia, regurgitation of previously ingested food, substernal or back pain and sometimes loss of weight and serious interference with nutrition. Their removal is easily and safely accomplished by a one-stage transthoracic operation. The actual details of technique in the handling of the diverticulum itself and the closure of the opening which remains in the esophagus after the removal of the sac are identical

with the technique applied in the treatment of those of the pharyngoesophageal type in the neck.

False Diverticula. Diverticula of the so-called traction variety (Fig. 24) are produced by the pull upon the esophageal wall of a cicatrizing adjacent inflammatory process usually tuberculous lymphadenitis in the subcarinal region of the mediastinum. They often consist merely in a distortion or sharp angulation of the side of the esophageal wall which becomes adherent to the nearby inflamed lymph nodes. In the typical case, they produce no symptoms and are discovered during routine roentgen ray examination of the esophagus. No treatment is necessary except in the rare patient in whom a calcified lymph node may ultimately ulcerate into both the esophagus and the trachea or a bronchus with the development of a fistulous tract between the two organs. In such

control of the lower segment which is characteristic of this disease. It was Hurst who proposed that megaeosophagus results from a lack of the normal coordination between the mouth of the esophagus and the lower segment, leading to a failure of the latter to open at the proper moment after deglutition is initiated. For this phenomenon he proposed the term *achalasia*.

In a large proportion of the patients a favorable response results from dilation of the lower esophagus with the hydrostatic dilator or a mercury bougie. In many instances a single treatment is followed by striking improvement or actual disappearance of the symptoms often for long periods. In others, the response is either incomplete or of limited duration and resort must be had to surgical intervention to bring about further relief. The procedure to be employed depends upon the anatomic type of the lesion involved. When an obvious hypertrophy of the distal circular muscle fibers exists the dysphagia can be relieved by making a longitudinal incision completely through the thickened muscle layer down to but not into the mucosa (esophagomyotomy). In the group of patients who have a thin walled, atrophic segment at the lower end, esophagoplasty consisting of a lengthwise incision through the esophageal wall in the constricted area followed by a circumferential closure of the opening accomplishes the same result.

Permanent relief of dysphagia can be brought about by surgery but it is not possible to restore the esophagus to a normal condition. Although the degree of distention of the esophagus decreases, often to a striking extent, after the operation, the histologic changes characteristic of the underlying pathologic process remain unchanged.

DIVERTICULA

Diverticula of the esophagus are usually spoken of as being of the pulsion or traction type, according to their supposed etiologic background. Only the former however is a true diverticulum. The other is a false or pseudo diverticulum, resulting from the traction of an adherent adjacent inflammatory process.

True Diverticula. Pulsion diverticulum of the esophagus which consists of a herniation or bulging of mucosa and submucosa through a weakened portion of the muscular coat, occurs either at the pharyngoesophageal junction or in the lower segment of the

thoracic portion of the esophagus just above the diaphragm. Pharyngoesophageal diverticula (Fig. 22) are much the more frequent. The diverticulum arises from the posterior pharyngeal wall just above the cricopharyngeus muscle and, as it enlarges, extends downward behind the esophagus often veering slightly to the left as it descends. It may assume large proportions over a period of years. The symptoms are characterized by varying degrees of dysphagia, regurgitation of food (often that consumed days previously) the occurrence of peculiar and often embarrassing noises in the neck and throat during deglutition and, ultimately in the very large ones, serious interference with nutrition because of the difficulty in swallowing as a result of compression of the esophagus by the overdistended pouch. Elderly patients sometimes aspirate material into the trachea. Cough from tracheal irritation is a frequent symptom.

The ideal surgical treatment is excision of the sac. Under the prophylactic protection of penicillin and streptomycin, and with careful surgical technique, this can be accomplished in one stage with a minimum of inconvenience to the patient (Fig. 23). Under unusual circumstances of poor general condition, or for other reasons, the two-stage



Figure 22. A roentgenogram made after ingestion of barium, showing a large pulsion diverticulum of the pharyngoesophageal junction.

INFLAMMATORY LESIONS

Chemical Burns with Cicatricial Stenosis.

The ingestion of caustic liquids in the form of strong acids or alkalis causes severe chemical burning of the esophagus, often with total destruction of the mucosal layer. This results ultimately in the formation of a stricture as the acute inflammatory reaction subsides and is replaced by cicatrization. The extent of such an injury depends upon the amount of liquid swallowed, its concentration and the rapidity of its passage through the esophagus. Victims vary from those with one or more short areas of involvement, usually at the lower end, to those with complete destruction and cicatricial obliteration of the entire esophagus from the pharynx to the cardia (Fig. 25).

The immediate treatment consists in administering the appropriate antidotal solution and combating the pain, shock and dehydration. During the acute phase of the injury the administration of Meticorten may diminish the degree of swelling and the severity of the inflammatory changes. The usual precautions required for the employment of this drug must be observed.

Gastrostomy may be required in order to maintain nutrition. If the damage is not too severe, ultimate obstruction may be avoided.

tion ensues, is enhanced by the edema and esophagitis which soon develop. If prompt treatment is not instituted secondary periesophagitis, mediastinitis and empyema may develop. Early in the management of such a case, it is important to determine from the clinical history or the use of roentgenography the size, shape and density of the foreign body. The roentgenogram will establish the location if the object is radiopaque. A negative finding, however, is not conclusive and must be supplemented by esophagoscopic examination in the hope not only of making the diagnosis but also of removing the object. The majority of foreign bodies can be removed by means of instrumentation through the esophagoscope. In some patients, however, this is best accomplished by pushing the object into the stomach whence it will probably pass through the remainder of the gastrointestinal tract without difficulty. Esophagotomy is rarely indicated but should be applied without hesitation if difficulties are encountered with the use of other methods.

If the impacted object is known to be a piece of meat, it can usually be dislodged promptly by the administration of a 5 per cent solution of papain which softens and liquefies the bolus so that it will pass on into the stomach.



Figure 25 Roentgenogram showing an extensive chemical burn of the esophagus with involvement of almost the entire length of the organ in a young girl, seventeen years of age, who had swallowed a solution of lye with suicidal intent (posteroanterior and lateral views)

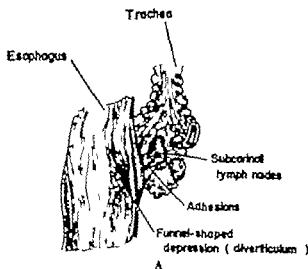


Figure 24 A, Drawing of a traction diverticulum showing funnel-shaped distortion of the wall of the esophagus produced by the pull of adhesions to adjacent inflamed subcarinal lymph nodes (Terracol, J. and Sweet, R. H. Diseases of the Esophagus). B, A roentgenogram made after ingestion of barium, showing a traction (false) diverticulum of the esophagus in the midthoracic segment.

a patient, a direct exposure of the area through a thoracotomy incision is necessary to make it possible to close the fistula tract.

TRAUMA

Injury to the esophagus by external violence is rare and is almost always associated with serious damage to other viscera. Penetrating wounds produced by the passage of missiles or sharp instruments on the other

hand, may involve the esophagus without much damage to adjacent structures. In such patients immediate direct exposure of the injured area may make it possible to deal with the laceration in a satisfactory manner and prevent the prolonged disability which results from fistula formation, with the attendant infection of the neck or mediastinum.

In nonmilitary practice the most frequent source of injury to the esophagus is the result of perforation either with the esophagoscope or more often, as a result of attempts to pass a bougie. The most frequent site of injury by the esophagoscope is the cervical area. The location of perforations by instrumentation with a bougie is usually just proximal to or actually through the area of constriction which it is intended to dilate. In either case an early operation to suture the damaged area should be considered. The majority of small injuries produced by the esophagoscope however heal spontaneously and if the element of infection is minimized by prompt and adequate administration of antibiotics periesophageal abscesses and severe mediastinitis rarely develop. If such an abscess should occur however drainage through the neck or by posterior mediastinotomy is necessary.

Spontaneous rupture of the esophagus sometimes occurs as a complication of violent attacks of vomiting resulting from sudden compression of an overdistended stomach due to external violence such as a fall from a height. The rupture always develops in the lower mediastinal segment and is attended by the sudden development of excruciating pain in the lower portion of the chest with radiation to the back. If the condition is recognized promptly an immediate operation to close the defect is curative. If untreated, however a mediastinal abscess develops which may perforate into one or both pleural cavities. Surgical drainage then becomes necessary and a prolonged illness ensues usually complicated by the presence of an esophagopleurocutaneous fistula which may require an additional operation for closure. The mortality rate of this condition is high.

Impaction of a foreign body in the esophagus may result in perforation, either because of ulceration through an area of pressure necrosis or by laceration produced by a sharp edge or point. Manipulative attempts to remove the object are also a frequent source of injury. Impaction of a foreign body may also cause obstruction which, if infec-

with the Plummer Vinson syndrome are of a precancerous nature. In fact, carcinoma of the upper end of the esophagus occurs much more frequently among women than among men, whereas in the lower esophagus the reverse is true.

The involvement of the esophagus by specific infections, such as tuberculosis, syphilis and actinomycosis, is a rare occurrence in the present day. No distinctive subjective symptoms are observed. The diagnosis can rarely be established without the aid of histologic studies of biopsy or autopsy specimens. The treatment should be directed toward the control of the generalized infection by whatever means may be indicated, including the use of penicillin and streptomycin.

Esophagitis and stricture formation sometimes severe enough to require surgical intervention, is an occasional complication of scleroderma.

VARICES OF THE ESOPHAGUS

Esophageal varices are the most frequent cause of massive bleeding from the esophagus. They develop in patients with portal hypertension, either from thrombosis of the portal or splenic veins or from cirrhosis of the liver. The diagnosis suggested by the occurrence of copious vomiting of bright blood can usually be confirmed by roentgen ray visualization after the ingestion of barium. This condition has been the subject of much study in an attempt to elaborate a satisfactory method of treatment. Injection of sclerosing solutions directly into the swollen submucosal veins, which can be effected through the esophagoscope, has been tried with indifferent success. Certain surgical operations, such as splenectomy, ligation of the splenic artery and interruption of the periesophageal veins, have proved disappointing. A great advance in the management of the condition has been made in recent years by producing a shunt of the portal blood into the systemic venous system, either by anastomosis between the splenic vein (after splenectomy) and the left renal vein or by direct anastomosis between the portal vein and the vena cava (Eck fistula). The success of this procedure arises from the reduction of the pressure within the dilated veins, which in turn diminishes or abolishes their tendency to bleed.

BENIGN TUMORS

Benign tumors of the esophagus occur infrequently but when they become large,



Figure 27 A roentgenogram made after ingestion of barium, showing the defect produced by a leiomyoma of the esophagus.

they may cause dysphagia severe enough to require surgical intervention. Solid tumors of a benign character are encountered somewhat more frequently than cysts. The diagnosis is usually strongly suggested by the x ray examination made after ingestion of barium (Fig. 27). In some patients the use of the esophagoscope may be necessary for confirmation. The tumors are usually leiomyomas or fibromas. Lipomas are seen occasionally and cysts, often of a congenital origin, may be observed. Some of the solid tumors hang from a pedicle and can be removed through the esophagoscope by using a snare. Others grow within the wall of the esophagus and their removal requires a direct surgical approach to the region involved.

MALIGNANT TUMORS

Sarcoma of the esophagus is a rare tumor. In the cervical or upper thoracic segments, where striated muscle fibers exist, a rhabdomyosarcoma may occasionally be found. Leiomyosarcomas may develop at any level. A lymphosarcoma of the esophagus may be seen on rare occasions. The symptoms produced by these lesions are not characteristic and the x ray appearance may not be helpful. A positive diagnosis can be established only by biopsy or examination of the lesion

by bougienage repeated at intervals as required. In order to facilitate the passage of the bougie the patient should be made to swallow a silk thread as early as possible. In spite of the relative success of treatment by bougienage, many patients finally develop such a tight stricture that either the treatment fails of its accomplishment or perforations result. When it becomes apparent that dilation is either too dangerous or too unrewarding, the patient can be cured permanently by reconstructive surgery to establish a new passageway from the pharynx to the stomach. This may be accomplished by partial esophagectomy with primary intrathoracic esophagogastric anastomosis or in some instances, by substitution of a segment of jejunum or of the right half of the colon as a by-passing procedure without the removal of the esophagus. Either of these techniques is superior to the use of skin tube substitution because of the tendency of the latter to form strictures and because of the time required to complete them.

Nonspecific Esophagitis and Ulceration. Localized areas of inflammation not due to any recognizable mechanical or chemical trauma occur actually more frequently than was formerly supposed. The etiologic factor cannot always be determined. Esophagitis may exist as a complication of acute pharyngitis. It has been known also to result from agranulocytosis following the ingestion of sulfonamide drugs (Fig. 26). It may be found at any level in the esophagus but it is most frequently encountered in the lower end, where it is usually associated with the presence of a short esophagus and thoracic segment of stomach. It is unusual for esophagitis to occur as a complication of an esophageal hiatus hernia of the acquired type.

Ulceration, sometimes considered of the peptic variety is a frequent occurrence in the esophagitis which develops near the cardia. These ulcerations may develop in anomalous gastric mucous membrane which extends upward from the cardia into the esophagus, but they frequently involve the mucosa of the esophagus itself as well. The symptoms are pain, often of a dull gnawing variety referred to the substernal or epigastric area, mild to severe degrees of dysphagia, the sensation described as "heart burn" and sometimes hematemesis. Treatment is dietary and medicinal at first. If the dysphagia becomes a problem, one or more dilations may be required. Some cases, however result ultimately in the formation of a tight stricture caused by the circumferential contraction of the scar tissue which de-

velops in the ulcer bed as healing of the ulcerated area progresses. Attempts to overcome this difficulty by the passage of bougies if not disastrous because of perforation, are ineffectual because of the prompt re-formation and contraction of scar tissue. Under these circumstances a resection becomes necessary. An occasional indication for resection also is the occurrence of severe bleeding.

An important form of chronic esophagitis seen almost exclusively in women beyond middle age is a part of the clinical condition known as the *Plummer-Vinson syndrome*. Patients who exhibit this picture experience dysphagia especially for solid foods, which become lodged at the mouth of the esophagus, to which the stenotic process is confined. Ease of fatigue which accompanies the condition is due to a sideropenic (iron deficiency) anemia of long duration. The mucosa of the mouth, pharynx and tongue may be pale, smooth and glistening. The latter may show fissures and superficial erosions with a tendency to leukoplakia. The skin of the vulva and anus may present a similar appearance. There is usually achlorhydria.

There is strong clinical evidence to suggest that the mucosal changes in the retro-cricoid segment of the esophagus in patients



Figure 26. A roentgenogram made after ingestion of barium in a patient with agranulocytic esophagitis with resulting constrictive atresia of the esophagus. The agranulocytosis was caused by ingestion of a sulfonamide.

so pitiable that the possible benefits of surgical treatment should be offered to all if there is a reasonable prospect of removing the growth and re-establishing the continuity of the alimentary canal by anastomosis. The operation is planned to bring about a cure in the relatively few favorable cases but, of equal importance it should provide the maximum relief of discomfort in the larger group of patients whose disease is beyond the hope of cure. X-ray treatment is of limited value as a method of palliation and has produced few long survivals in patients with proved carcinoma of the esophagus.

The technical problem in the surgical management of carcinoma of the esophagus varies somewhat from one level to another. It is now possible to resect the growth and re-establish continuity so that the patient can eat in a normal manner in every case in which the growth is resectable.

In the cervical segment of the esophagus the growth can be removed and a flap of skin turned in from the neck so as to make a skin-lined tubular substitute for the excised portion. The operation is performed in two stages. By this means, complete restoration of function can be brought about, but it is applicable only in early favorable cases because of the fact that not a large enough number of regional lymph nodes can be excised.

The older Torek procedure which permitted only relatively local resection of the growth and made no provision for excision of many of the regional lymph nodes, resulted in a very small number of long survivals. Furthermore, because it left the patient with a proximal esophagostomy in the neck and a gastrostomy for feeding purposes, it provided a most inadequate degree of palliation even if the surgeon succeeded ultimately in constructing a substitute esophagus, either by a skin plastic procedure or by the interposition of a segment of the jejunum or the colon. This operation is therefore no longer used.

As a result of recent advances in surgery of the esophagus, radical resection of the diseased segment and wide excision of many of the regional lymph nodes are possible, especially in the lower region of the thorax. At the same time a restoration of the continuity of the alimentary canal can be brought about by esophagogastric anastomosis. The exact details of the technique must be altered from case to case, depending on the location of the growth. The op-

eration is performed through a left thoracotomy incision and involves opening the abdomen by incising the diaphragm in order to gain access to the stomach, which must be pulled up to meet the proximal segment of the esophagus for the performance of the anastomosis after the growth has been removed. Depending upon the location of the tumor and the level at which the esophagus must be severed, the anastomosis is made either just below or just above the aortic arch. In the rare patient in whom the tumor involves the superior mediastinal segment of the esophagus an additional cervical incision must be made and the stomach brought up through the chest and into the neck, because of the necessity for cutting across the esophagus in the cervical segment (Figs. 29 and 30). An alternative technique, the use of which is now being explored in this group of cases, is the substitution of a long segment of the colon (Fig. 31).

Carcinoma of the lower esophagus and cardiac end of the stomach is treated in exactly the same manner as that arising at a higher level in the thorax, with the exception that more of the stomach must be resected with the excised segment of esophagus. The esophagogastric anastomosis in these patients is made at a relatively low level in the chest.

So far as late results are concerned, there is already enough knowledge available from the experiences of patients whose operations

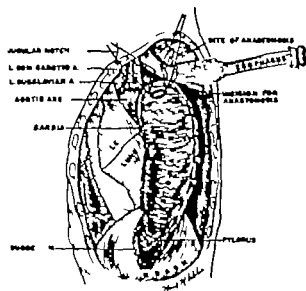


Figure 29 A diagram showing a step in resection of the esophagus for carcinoma of the mid-thoracic portion, with high intrathoracic (supra-aortic arch) anastomosis.

after its removal. In the case of lymphomatous tumors, a favorable temporary response may result from x ray treatment.

Carcinoma of the esophagus comprises the overwhelming majority of malignant neoplasms of that organ. In fact, the most frequent lesion of any kind in the esophagus is carcinoma. It may develop at any level within the esophagus but the most frequent site is in the middle half. In the lower end, a certain amount of confusion results from the fact that approximately four out of five growths which invade the cardia arise in the stomach, are adenocarcinomatous and are not actually esophageal in origin, although the lower segment of the esophagus is usually invaded. Carcinoma arising primarily in the esophagus itself is typically epidermoid histologically but the degree of malignancy of these tumors is of a high order. In over 70 per cent of the patients in whom resection of the growth can be performed, metastases to the regional lymph nodes are already present. Extension of the growth in the wall of the esophagus is often wide and involvement of adjacent organs by direct invasion frequently occurs relatively early in the progression of the disease. All hope of cure therefore rests on the establishment of an early diagnosis and the prompt resort to surgical extirpation.

The onset of the disease is insidious and its development is well advanced before the patient is aware of any abnormality. The first symptom is dysphagia, often intermittent, depending upon the physical properties of the food ingested. As the condition advances, the encroachment upon the lumen of the esophagus increases and in a relatively short time the patient finds that only soft foods will pass down and finally nothing but liquids. In the late phase of the disease, pain, which is of two types, may be experienced. The first is that which results from local distention or muscle contraction just above the level of the growth and is the result of esophagospasm. It is referred in a segmental manner to the substernal region, with radiation to the back and sometimes into the neck. It may be high or low depending upon the level of the tumor in a given subject. The second type of pain is a steady boring discomfort felt deep within the chest and in the back. This is the result of the direct invasion of surrounding tissues by tumor growth or the development of periesophagitis and mediastinitis from an ulcerating infected lesion.



Figure 28 A roentgenogram made after ingestion of barium, showing an annular filling defect, characteristic of carcinoma of the esophagus.

In the majority of patients a roentgenogram made after the ingestion of barium will demonstrate the characteristic irregular filling defect, usually annular in extent, involving the esophagus at whatever level the tumor may be located (Fig. 28). If the growth happens to be an early one, the x-ray examination may not prove to be conclusive. In any such patient, if the history suggests the probability of an esophageal lesion, it is mandatory to perform esophagoscopy. By means of the endoscopic examination, a diagnosis can usually be made with certainty by obtaining tissue for biopsy. It should be pointed out, however that unless the grasp of the forceps is deep it may happen that only surrounding inflammatory tissue may be obtained for histologic study. In such an instance the procedure should be repeated or resort made to surgical exploration.

Recent experience has shown that cytologic studies of material obtained from the esophageal lumen by washing with saline solution result in a high percentage of positive results. In many instances the employment of this method eliminates the need for esophagoscopy to establish the diagnosis. A negative result, however must not be assumed to indicate the absence of this disease.

The ultimate fate of patients with carcinoma of the esophagus is so hopeless and their plight during the last days of their lives

- Health and Disease. Springfield, Ill., Charles C Thomas, 1958.
- Dornbort, A. C., Pierce J. W., and Whimster L. W.: Esophageal Lesions in Scleroderma. *Lancet* 1 698 1954
- Garlock, J. H.: The Re-establishment of Esophago-gastric Continuity Following Resection of the Esophagus for Carcinoma of the Middle Third. *Surg. Gynec. & Obst.* 78:23, 1914
- Garlock, J. H., and Klein, S. H.: The Surgical Treatment of Carcinoma of the Esophagus and Cardia (An Analysis of 457 Cases) *Ann. Surg.* 129:19 1951
- Heller, E.: Extramucosa Cardioplastik beim chronischen Cardiospasmus mit Dilatation des Oesophagus. *Mitt. u. Grenzgeb. d. Med. u. Chir.* 27:141 1913
- Hunt, A. F., and Rake, C. W.: Achalasia of the Cardia (So-Called Cardiospasm) *Quart. J. Med.* 23, 491, 1930.
- Libson, R. R., and Ellis, D. S.: Emergency and Definitive Treatment of Bleeding Esophageal Varices. *J.A.M.A.* 160:1017 1958.
- Lortet-Jacob, J. L.: *Chirurgie de L'Oesophage* Paris, Editions Medicales Flammarion, 1951
- Meyers, R. T., and Bradshaw H. H.: Benign Intramural Tumors and Cysts of the Esophagus. *J. Thoracic Surg.* 10-101 1949
- Nardi, G. L.: Surgical Treatment of Lye Strictures of the Esophagus by Mediastinal Colon Transplant without Resection. *New England J. Med.* 256:777 1957
- Raven, R. W.: The Surgical Treatment of Carcinoma of the Hypopharynx. *Brit. J. Surg.* 42:8, 1951
- Ray E. S., and Morgan, D. L.: Cortisone Therapy of Lye Burns of the Esophagus. *J. Pediat.* 49:304 1956.
- Sanson, P. C.: Injuries and Wounds of the Esophagus (A Classification) *California Med.* 60:303, 1954.
- Sosa, M. L., and Arnold, L. M.: Esophagoscopy in the Diagnosis and Treatment of Esophageal Diseases. *Am. J. Surg.* 93:183, 1957
- Sweet, R. H.: Subtotal Esophagectomy with High Intrathoracic Esophago-gastric Anastomosis in the Treatment of Extensive Cicatricial Obliteration of the Esophagus. *Surg. Gynec. & Obst.* 83:117 1916.
- Sweet, R. H.: Idiopathic Dilatation of the Esophagus. *S. Clin. North America* 27:1128 1917
- Sweet, R. H.: Carcinoma of the Superior Mediastinal Segment of the Esophagus. *Surgery* 21:929 1918
- Sweet, R. H.: The Technique of Transthoracic Resection of the Stomach and Esophagus. *Nelson's Loose Leaf Surgery* New York, Thomas Nelson & Sons, 1918.
- Sweet, R. H.: The Treatment of Carcinoma of the Esophagus and Cardiac End of the Stomach by Surgical Excision. *Surgery* 23:932, 1918.
- Sweet, R. H.: Pulsion Diverticulum of the Pharyngo-esophageal Junction. Technique of the One Stage Operation. *Ann. Surg.* 125:41 1919
- Sweet, R. H.: *Thoracic Surgery* 2nd ed. Philadelphia, W. B. Saunders Company 1951.
- Sweet, R. H.: Excision of Diverticulum of the Pharyngo-Esophageal Junction and Lower Esophagus by Means of the One Stage Procedure. *Ann. Surg.* 143:433, 1956.
- Sweet, R. H.: Surgical Treatment of Achalasia of the Esophagus. *New England J. Med.* 251:87 1956.
- Sweet, R. H.: End Results in Cancer of the Esophagus. *Proceedings of the Third National Cancer Conference American Cancer Society Philadelphia, J. B. Lippincott Company* 1957
- Sweet, R. H., Robbins, L. L., Cephart, T., and Wilkins, E. W., Jr.: The Surgical Treatment of Peptic Ulceration and Stricture of the Lower Esophagus. *Ann. Surg.* 130:258 1954.
- Sweet, R. H., Souther, L., and Valenzuela, C. T.: Muscle Wall Tumors of the Esophagus. *J. Thoracic Surg.* 27:13, 1954
- Terracol, J., and Sweet, R. H.: *Diseases of the Esophagus*. Philadelphia, W. B. Saunders Company 1958.
- Wilkins, E. W., Jr.: Current Considerations of Esophageal Physiology Normal and Abnormal. *New England J. Med.* 257:24, 1957
- Woolley H.: The Surgical Treatment of Carcinoma of the Pharynx and Upper Esophagus. *Surg. Gynec. & Obst.* 75:489 1942.



Figure 30 A roentgenogram made postoperatively after ingestion of barium, showing high intra-thoracic esophagogastric anastomosis with the fundus of the stomach in the apex of the chest.

were performed five or more years before the observations were made to demonstrate that there is hope for cure in an appreciable number of patients with carcinoma of the esophagus.

In an experience of over eighteen years, it has been demonstrated that, given favorable cases with no evidence of lymph node metastases, at least 34 per cent of the patients who have had a resection of the lower end of the esophagus for carcinoma and 23 per cent of those operated upon for a carcinoma of the midthoracic segment have survived five or more years after operation.

There is reason to believe, therefore, that the long term results following resection and primary anastomosis in patients with carcinoma of the esophagus are as good as those for resection of any other portion of the alimentary canal, with the possible exception of the rectum.

As with carcinoma elsewhere, a large number of the patients who have had resection performed will ultimately succumb to the effects of recurrent or metastatic disease. In this group the operation of resection and intra-thoracic esophagogastric anastomosis is the best method of palliation available at the present time, because in the majority of these patients a recurrence of the growth does not develop at the site of anas-

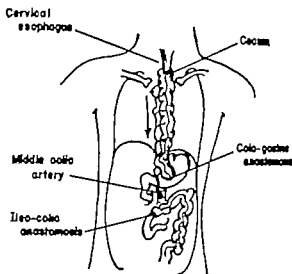


Figure 31 Diagram illustrating the employment of the right colon to supplant the esophagus. Arrow shows the direction of peristalsis in the transplanted bowel.

tomosis. The result is that the patient continues to eat in a normal fashion as long as he survives.

With those patients whose growth cannot be removed, the interposition of a segment of the colon between the cervical esophagus or the pharynx and the stomach, followed by intensive irradiation of the tumor appears to be a satisfactory means of amelioration. Further experience with this method is required before final evaluation of its efficacy from the standpoint of survival can be made.

READING REFERENCES

- Allison, P. R. Peptic Ulcer of the Esophagus. *Thorax* 3:20, 1948.
- Allison, P. R., and Johnstone, A. S.: The Esophagus Lined with Gastric Mucous Membrane. *Thorax* 8, 87, 1953.
- Barrett, N. R. Chronic Ulcer of the Esophagus and Esophagitis. *Brit. J. Surg.* 36:175, 1950.
- Benedict, E. B. Endoscopy. Baltimore, Williams & Wilkins Company, 1951.
- Benedict, E. B., and Gillespie, J. E. O. Esophageal Stenosis Caused by Peptic Esophagitis or Ulceration. *New England J. Med.* 250:642, 1954.
- Benedict, E. B., and Nardi, G. L.: The Esophagus; Medical and Surgical Management. Boston, Little, Brown & Company, 1958.
- Benedict, E. B., and Sweet, R. H.: Benign Stricture of the Esophagus, Hiatus Hernia, Esophageal Ulcer and Duodenal Ulcer. *Gastroenterology* 11: 618, 1948.
- Bingham, J. A. W., and Logan, J. S.: The Nature and Treatment of Plummer-Vinson Dysphagia. *Brit. M. J.* 2:650, 1953.
- Burford, T. H., Webb, W. R., and Ackerman, L.: Caustic Burns of the Esophagus and Their Surgical Management, A Clinico-experimental Correlation. *Ann. Surg.* 138:453, 1953.
- Code, C. F.: An Atlas of Esophageal Motility in

that a greater predisposition to gastric ulcer exists in these Andean natives. In the high altitudes, vascular and hematologic factors as well as chronic occupational stress, may play a most important role in the etiology of gastric ulcer.

The stomal ulcer is generally rated as occurring in 18 to 34 per cent of patients following gastroenterostomy, while following subtotal resection it has a lower incidence usually rated as around 8 per cent. The incidence of perforation in cases of peptic ulcer is estimated as from 3 to 8 per cent and of recurrent acute perforations as 1 to 1.5 per cent. The incidence of hemorrhage is much higher, varying according to different observers between 5 and 25 per cent. The risk of later hemorrhages is greater in patients who have had more than one hemorrhage. The belief that a greater incidence of ulcer activity occurs in the spring and fall seasons with an apparent high incidence of hemorrhage from October through January has become a tradition not easily proved by reported statistics. It is probable that many gastric ulcers are undiagnosed during life because of the bizarre symptoms which they present.

Anatomy, knowledge of the anatomy, blood supply and innervation of the stomach and duodenum is fundamental to an understanding of the basic features of the disease. Such an understanding is also important in

evaluating the various methods which have been employed to treat peptic ulcer.

The stomach lies between the cardiac and pyloric sphincters and includes the fundus, the corpus and the antrum. The lower part of the antrum is loosely termed the pyloric or prepyloric region. The arteries to the stomach are plentiful. This excellent blood supply has the advantage that healing is prompt after operation but at the same time the disadvantage that bleeding is difficult to control. Bentley and Barlow demonstrated that while in the rest of the stomach the mucosal arteries come from a rich submucosal plexus along the lesser curvature they rise directly from the long, slender branches of the right and left gastric arteries which pierce the muscularis directly. This apparently more precarious arrangement of the vascular supply may explain the predisposition of the *magenstrasse* to develop gastric ulcer. The presence of arterial-venous anastomosis in the stomach was demonstrated by Barlow, Bentley and Walter in 1931 and was confirmed by Sherman and Verumann in 1954 (Fig. 32). The veins and the lymphatics tend to follow the arteries.

The nerves include the vagi, a left anterior and a right posterior which stimulate gastric secretion and the sympathetic fibers which probably inhibit gastric secretion.

The most important cells in the stomach are the parietal cells which are especially

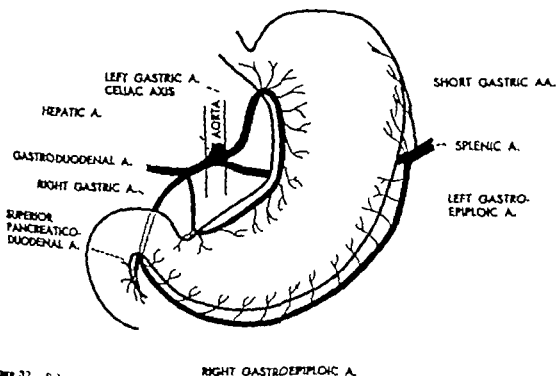


Figure 32. Schematic diagram of the arterial blood supply of the stomach (Barborik, C. J., and Tester E. C., Jr. *Peptic Ulcer—Diagnosis and Treatment*. Boston, Little, Brown & Company)

The Stomach

Peptic Ulcer and Benign Gastric Lesions

By CLIFFORD J BARBORKA M D

CLIFFORD JOSEPH BARBORKA attended Simpson College in Iowa, graduated from the University of Chicago and received his degree in medicine from Rush Medical College. His interest in gastroenterology was stimulated during his internship and developed during his years as a staff physician at the Mayo Clinic. Doctor Barborka, Director of the Gastrointestinal Clinic at Northwestern University Medical School, is a leader in his specialty and is well qualified by his wide experience and flexibility in methods of therapy to present the physician's views upon peptic ulcer.

PEPTIC ULCER

Peptic ulcer is a localized loss of the mucous membrane with secondary involvement of the muscularis. The identity of peptic ulcer is recognized in four areas of the upper gastrointestinal tract: (1) in the lower esophagus where it occurs rarely and is ascribed to regurgitation of the acid contents of the stomach, (2) in any part of the stomach itself where it must be differentiated from an ulcerating carcinoma, (3) in the first or second portion of the duodenum, and (4) in the jejunum at the site of an operative anastomosis.

Incidence. The incidence of ulcer in the United States is difficult to determine with any degree of precision since there is a marked discrepancy in statistics from various sources and for various reasons. In autopsy studies, the results of which often vary greatly from studies in living groups, it is thought that scars of previous ulcers, particularly in the duodenum, may have been overlooked or may not be present at all when an ulcer has completely healed. In studies of living individuals age and sex variations distort the true perspective in ulcer incidence. However statistics from all these sources indicate that between 8 and 10 per cent of our population will be afflicted with this disease within their lifetimes. Of patients seeking relief for diges-

tive symptoms, about 15 to 20 per cent are found to have an ulcer. This means that a most important productive age group of the population is struck by this disease and the economic repercussions are extremely important.

The annual mortality from peptic ulcer is 10,000 in the United States. Persons of any age may be affected, although it is most common in those in the younger and middle years. Males are affected four times as often as females. The incidence of duodenal ulcer is much higher than that of ulcer in other locations. Its incidence, as compared to that of gastric ulcer, is estimated as three to ten or more times as great. There is an interesting exception to this estimation. Carrido, Klinge and Luis Pena, observing patients in the Peruvian Andes, made a most provocative report on gastroduodenal ulcer in respect to high altitudes. The general incidence of gastroduodenal ulcer in their group of 17,500 insured laborers, chiefly miners, born and living in the Peruvian Andes at an altitude of 10,000 to 16,000 feet above sea level, is 0.4 per cent. The relation of gastric to duodenal ulcer is astounding, the proportion being 20.6 to 1 per cent. The greatest incidence of gastric ulcer occurs in persons between twenty-one and thirty years of age. Particularly astonishing is that hemorrhage occurred in 66 per cent of cases. It appears

nonreciprocal permeability which allows only water with hydrogen and chloride ions to pass through it into the lumen of the gland. Bicarbonate is returned to the blood.

Davenport has shown that carbonic anhydrase an enzyme which catalyzes the reaction of carbon dioxide and water to form carbonic acid and its ionic breakdown is present in high concentration in the parietal cells. The carbonic anhydrase serves primarily to maintain ionic and isotonic equilibrium within the parietal cells. It participates in the reverse chloride shift wherein chloride is secreted in exchange for bicarbonate. It appears that carbonic anhydrase furnishes one source of hydrogen ions for hydrochloric acid. This is supported by the observation that the administration of carbonic anhydrase inhibitors such as acetazolamide may markedly inhibit the production of hydrochloric acid. It has been postulated by Conway and others that the hydrogen is concentrated by a series of steps employing a so-called redox cycle (Fig. 34).

Pure parietal secretion contains per liter 166 mEq of chloride, 159 of hydrogen ion and 7 of potassium ion. This solution is iso-

osmotic with blood. Gastric juice does not reach this concentration because nonparietal secretion neutralizes and dilutes the parietal secretion. High concentration of acids has been observed up to 110 clinical units (a clinical unit being equal to 1 mEq per liter) and under strong stimulation the acid concentration of gastric juice may reach as high as 150 mEq per liter.

Pepsin Pepsinogen a protein, is formed by the chief cells and is converted to pepsin in an acid medium (below pH 6). The optimum pH for the proteolytic activity of pepsin is about 2. Stimulation of the vagus nerve produces an increase in secretion of gastric juice which contains mucus and is high in peptic activity. Stimulation with histamine on the other hand, yields a highly acid secretion which is low in peptic activity and practically mucus free.

Mucus Gastric mucus is secreted by the surface epithelial cells, by the chief cells of the neck and fundic glands, by the pyloric gland cells and by the cardiac gland cells. There are two components of mucus in the fundus and body of the stomach. The first is a layer of viscous mucus which covers

REDOX CONCEPT OF HYDROCHLORIC ACID SECRETION (CONWAY)

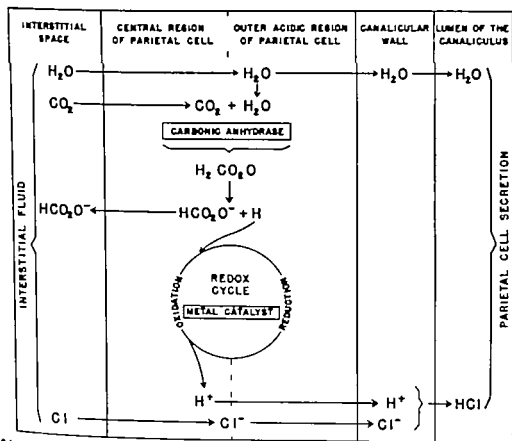


Fig. 34. Schematic diagram of the redox concept of gastric secretion, according to Conway (Texter E. C., Jr., and Barborika, C. J. Gastroenterology vol. 28)

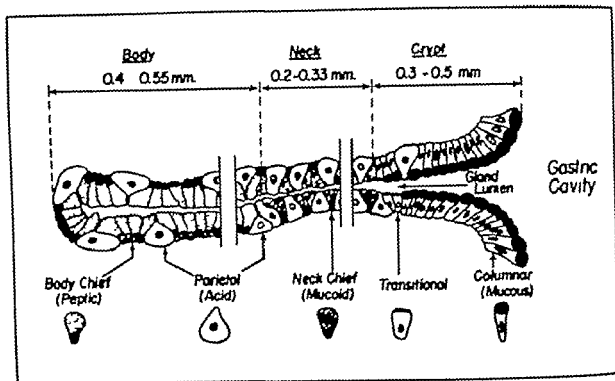


Figure 33 Cross section of a gastric gland (Barborka, C. J. et al. *Quart. Bull. Northwestern Univ. Med. School*, vol. 28)

plentiful in the corpus. There are essentially no parietal cells in the antrum. The parietal cells secrete hydrochloric acid. The chief cells secrete pepsin. In the proximal duodenum there is a special layer of cells the so-called Brunner's glands which secrete mucin that may protect the duodenum against peptic digestion.

Physiology Knowledge of the basic physiologic processes of gastric secretion and motility is as important as (if not more important than) knowledge of the anatomic features. Peptic ulcer results from an alteration in normal physiology. An understanding of the normal physiology is essential to an understanding of the abnormal. The physiology of the stomach and the duodenum is of great importance in studying and understanding the different affections of this region, not only peptic ulcer but the others as well.

Gastric secretion An imbalance between the eroding or destructive factors in gastric secretion and the defense factors is considered important in the development of an ulcer. This balance may be disturbed in several ways. There can be an increase in the quantity or duration of the secretion of the hydrochloric acid-pepsin combination; there can be a decrease in the secretion of the buffering fluids which neutralize and inactivate the hydrochloric acid-pepsin com-

bination and, lastly, there can be a decrease in the resistance of the local tissues to the action of the erosive hydrochloric acid-pepsin. In order to understand the factors underlying this imbalance it is essential to know what the normal mechanism of gastric secretion is.

Two portions of the stomach are concerned with gastric secretion. The proximal two-thirds of the stomach contains millions of tubular glands whose function is to produce the active digestive elements of gastric juice namely pepsin and hydrochloric acid. Pepsin, the digestive enzyme is formed by the chief cells which are located in the body of the gland, while hydrochloric acid is formed by the parietal cells. The surface epithelial cells secrete mucus. The distal third of the stomach contains the pyloric glands, whose chief function is the formation of mucus (Fig. 33).

COMPONENTS OF GASTRIC SECRETION
Hydrochloric acid The complete details of the mechanism of secretion of hydrochloric acid are not entirely clear. It is known that the main constituents—water, carbon dioxide and the chlorides—are derived from the blood. It is probable that water furnishes the source of the hydrogen of hydrochloric acid. Hollander has proposed the "membrane hydrolysis" theory, which postulates that the canalicular wall has a selective

partially digested protein act as gastric juice secretagogues when they are within the bowel. This hormone is not believed to be enterogastrone, an inhibitory hormone.

4. Hypothalamic-pituitary-adrenal phase
During the past five or six years there has been a growing body of evidence suggesting that gastric secretory activity may be influenced by endocrine as well as neurogenic factors. Recent studies suggest that stress may be mediated to the stomach not only by the vagus nerve but by a purely hormonal mechanism transmitted through the hypothalamic-pituitary-adrenal pathway independent of the vagus nerve or gastric antrum. Thus, the gastric and peptic glands may be integrated into the general endocrine system.

The hypothalamus has been described by Cannon as the seat of primitive human emotions. An intact hypothalamus is essential for the transmission of emotions to the pituitary. Leonardo da Vinci many centuries ago located the soul in the vicinity of the hypothalamus. A number of systemic stress factors, such as muscular exertion, fatigue and temperature changes as well as chronic emotional stress, that is, rage, fear, anxiety and frustration, may induce the hypothalamus to secrete a humoral substance which then stimulates the pituitary gland to secrete corticotropin (adrenocorticotrophic hormone) as part of the general adaptation syndrome. Corticotropin, in turn, activates the adrenal cortex to release a number of steroid hormones including cortisone and cortisone-like compounds.

Gastrointestinal ulceration with hemorrhage or perforation is an integral part of the alarm reaction. Repeated administration of corticotropin stimulates certain forms of stress. Both the corticotropin and the stress may produce an increase in the output of adrenal corticoids which can be measured in the blood and urine.

The adrenal cortex may have a dual role in the gastric responses to stress according to the "permissive" concept and the "conditioned" theory of hormonal activity. The adrenal may function as an agent which permits a gastric response to occur without whose presence the response could not occur. The second role casts the adrenal in a more direct relationship between the levels of adrenal cortical hormone attained and the extent of the gastric responses. Excess adrenal hormone may sensitize the stomach to respond more readily or stress itself may condition the stomach to respond to adrenal

hormone. It would therefore appear that the adrenal steroids may sensitize the stomach to ulcerogenic influences or to other factors yet to be determined, or that the stress itself may cause the stomach to respond abnormally to the adrenal hormone.

Present studies suggest that there are two routes by which stress may be relayed from the hypothalamus to the stomach: (1) a neurogenic pathway involving the cerebral cortex, the anterior hypothalamus and the vagus nerve and (2) a hormonal mechanism mediated through the posterior hypothalamus, the pituitary gland and the adrenal cortex.

a. Neurogenic pathway The neurogenic pathway is by way of the anterior hypothalamus to the vagal centers and the vagus nerve. Direct electrical stimulation of the anterior hypothalamus in animals produces a prompt but unsustained increase in gastric secretion of hydrochloric acid in thirty to sixty minutes which is abolished by vagotomy. Insulin hyperglycemia also induces prompt gastric hypersecretion in animals and man attributed to anterior hypothalamic and vagal stimulation.

b. Hormonal pathway A second hormonal phase of gastric secretion mediated through the adrenal cortex to the stomach by way of the hypothalamus and pituitary gland was postulated in 1950 by the observations in man of an increase in gastric hydrochloric acid and pepsin secretion during prolonged corticotrophic hormone stimulation unaffected by vagotomy but requiring an intact adrenal gland. It was suggested that chronic emotional and physical stress might be transmitted to the stomach by a hormonal pathway through the hypothalamic-pituitary-adrenal mechanism independently of the vagus nerve and gastric antrum and that acute or chronic peptic ulcer might be induced in man through this purely hormonal mechanism.

Porter and his associates have shown in monkeys that electrical stimulation of the posterior hypothalamus produced a delayed increase in gastric acidity after two and one-half to three hours, which was abolished by adrenalectomy but not by vagotomy or low cervical cord section. Insulin hypoglycemia also induced a delayed gastric response mediated through the posterior hypothalamus and pituitary-adrenal axis which was abolished by adrenalectomy but not by vagotomy.

A similar delayed gastric acid response to insulin hypoglycemia has been demonstrated in man by Shay and Sun, who observed that

the inner wall of the gastric cavity. The second is a layer of tall columnar cells immediately beneath this sheet of mucus together with low columnar and cuboidal cells which line the crypts of the gastric glands. Several components have been isolated from gastric mucus including two types of mucopolysaccharides. Gastric mucosa exists in two physical forms, dissolved mucus and visible mucus. Its function is to coat the mucosa of the stomach, protecting it from erosion. It also inhibits pepsin and neutralizes hydrochloric acid.

STIMULATION OF GASTRIC ACID SECRETION
Gastric acid secretion is stimulated by the following factors:

1 *Cephalic (nervous or psychic) phase by action of the vagi*. This usually acts early in the course of a meal occurring even at the sight, taste, smell or thought of food. Hypoglycemia sufficient to reduce the blood sugar level to below 50 mg. per 100 cc. or to produce definite symptoms of hypoglycemia acts upon the vagal centers producing an increased secretion of gastric acid. This action is the basis of the so-called insulin or Hollander test for vagal function. If the test shows no increase in free hydrochloric acid secretion after administration of sufficient insulin to fulfill the requirements listed above, vagotomy has been complete.

Vagotomy eliminates the secretory response of the stomach to sham feeding as shown by Pavlov in 1910. In 1947 Harkins showed that sectioning of the vagus nerves to the stomach in the Shay rat (when the pylorus was ligated in the fasting rat, an average of twenty two gastric ulcers per rat developed within twenty four hours) prevents the formation of gastric ulcers. Harkins and Hooker in 1947 demonstrated that sectioning of the vagus nerves to the stomach in the Mann-Williamson dog markedly lowers the incidence of marginal ulcer formation. In 1950 Dragstedt and his colleagues demonstrated that sectioning of the vagi to an isolated total stomach pouch or to a Pavlov pouch causes a marked reduction in acid secretion from that pouch. In 1952 Storer and his colleagues showed that the action of the vagi is not a simple one. They demonstrated that cutting the vagi to the main stomach causes an increase in the acid output of a Heidenhain pouch, possibly because of secondary stimulation of the hormonal phase. In 1953 Savage and his associates revealed that sectioning of the vagus nerves to a stomach pouch decreases the incidence of stomal peptic ulceration in the

connecting jejunal loop. In 1953 Movius and French showed that stimulation of the anterior hypothalamus produces a prompt rise in the acid secretion of the stomach of monkeys. This response is believed to travel over the vagi, is blocked by vagotomy but not by adrenalectomy and is related to the acute stress ulcer.

2 *Antral (gastric or hormonal) phase*. This is by stimulation of the antrum to produce gastrin which travels by the blood stream acting as a hormone to stimulate the parietal cells of the corpus and the fundus to secrete, in turn, more gastric acid. There is some evidence that the cephalic phase exerts its full effect in the presence of an intact antrum and vice versa, indicating some interdependence of the two stimulating mechanisms. The hormone, gastrin, is an internal secretion of the antral mucous membrane which contains no parietal cells and acts on the rest of the stomach which secretes no gastrin.

The following points demonstrate the importance of the antrum:

In 1906 Edkins of England showed that antral extract injected intravenously caused an increase in acid secretion. The hard-learned clinical fact, that if the excluded antrum is left in place after a partial gastric resection marginal ulcer is more apt to occur was demonstrated by physiologists and surgeons in the following manners.

In 1950 Dragstedt and his colleagues demonstrated the rather fundamental observation that when the antrum is in contact with intestinal contents it stimulates the secretion of hydrochloric acid from Pavlov or total stomach pouches in dogs. When the antrum is isolated, excised or transplanted subcutaneously it does not act in this stimulatory manner.

In 1952 Oberhelman and his group placed foods and other chemicals in the isolated antrum of dogs and found that it stimulated acid production of Heidenhain pouches. In 1954 Dragstedt and his coworkers showed that physical stimuli, including distention, to the antrum attached to the colon of dogs caused an increase in the acid secretion of Heidenhain pouches.

3 *Intestinal phase*. A hormone similar to but less powerful than gastrin may be secreted by the intestinal mucous membrane when in contact with food. The secretagogues which produce gastric secretion when they are in the stomach also stimulate gastric secretion when they are in the intestine. Substances such as soaps, fatty acid and

ments of the empty stomach give rise to the sensation termed "hunger pangs." These hunger pangs are peristaltic waves which start in the upper stomach region and pass down through the duodenum.

The motor activities of the stomach during digestion can be divided into two types: gastric peristalsis and changes in muscle tone. Gastric peristalsis consists of regular periodic contractions which segment off the distal end of the stomach and carry the portion of the gastric contents through the pyloric sphincter and into the small bowel. In addition to these gross movements, smaller changes, called "tone" changes, have been observed within the stomach. These consist of a gradual shortening of the muscle fibers of the stomach as the volume of gastric contents decreases.

The functions of the stomach during digestion include the reception of the meal, mixing of the contents of the stomach and assuring passage of the gastric chyme into the small bowel. This is a highly coordinated function which has been carefully studied by Quigley and his coworkers. He has observed that the distal portion of the stomach, the pylorus, and the first portion of the duodenum behave as a coordinated segment and he has termed this the "antral pump." It is his opinion that the pyloric sphincter's chief function is to prevent regurgitation of the duodenal contents into the stomach.

Several mechanisms operate to prevent any great increase in intragastric pressure when food enters the stomach. Initially there is a relaxation of the walls of the stomach and a concomitant inhibition of motor activity. The major portion of the food is held in the reservoir or upper portion of the stomach, and at periodic intervals portions of the mass are moved down into the antral pump region. The major stimulus for evacuation of the stomach is the size of the meal. Therefore, a large meal leaves the stomach more rapidly than a small meal and for this reason frequent small feedings are advised for patients who have peptic ulcer. The rate of gastric emptying is also regulated by factors acting within the duodenum. It can be observed fluoroscopically that in some patients an initial bolus is passed into the duodenum and then there is no further gastric emptying. An inhibitory reaction takes place within the stomach, suppressing peristaltic activity. The exact nature of this duodenal inhibitory regulation is not understood. In other patients, once emptying is

initiated it is continued until the stomach is emptied of its contents.

Etiology. Peptic ulcers, which represent a localized loss of the mucosal lining tissue in the areas of the gastrointestinal tract bathed by gastric juice, may be the result of multiple etiologic factors. As we all know the underlying cause of ulcer is still unknown. The two associative causative factors—hydrochloric acid secretion and changes in motility—are as yet the only two targets for practical treatment although tissue resistance and circulatory changes are also recognized as intermediate factors between the constitutional factor and the culminating production of ulcers. The studies of stress ulcers have been of great interest. The postulate that acid secretion is augmented by increasing circulating adrenal cortical hormones has been widely discussed. The fact that similar ulcers appear in the adrenalectomized animal suggests the possibility that ulcer occurs in the shock phase of the alarm reaction and that its mediation is by some unknown mechanism. Of interest is the fact that increased acid production resulting from corticoid therapy is not blocked by vagotomy, suggesting that the cerebral vagal and the pituitary-adrenal pathways bear two distinct influences on the production of ulcer. With limitations in our knowledge of the exact underlying factor and of its influence on secretion of hydrochloric acid, motility and tissue resistance, we are forced at present to work backward from the suppression of acid and decrease in motility as effective therapeutic measures instead of forward to such measures from a clearly outlined etiology.

Eroding factors. Erosion takes place both by chemical irritation and as the result of mechanical factors. Hydrochloric acid is necessary for the production and maintenance of a peptic ulcer. Pepsin is likewise important in the development of an ulcer. Gastric secretion tends to be higher than normal in duodenal ulcer patients, but slightly lower than normal in gastric ulcer patients.

Inadequate neutralization of the hydrochloric acid formed by the parietal cells probably plays a role in the development of an ulcer. Normally the surface or mucous cells of the stomach secrete alkaline mucus which tends to neutralize the acid produced by the stomach. It has been suggested that this mucous barrier is inadequate in ulcer patients.

the late gastric response to the stress effect of insulin hypoglycemia was abolished by total adrenalectomy.

5. Pancreatic phase. The pancreas plays a double role in the etiology of peptic ulcer. The first depends upon the fact that exclusion of the external secretion of the organ increases the tendency to ulceration of the duodenum. This observation is the basis of production of the Mann-Williamson experimental ulcer in dogs. The reason for the importance of the internal secretions of the pancreas in gastric secretion is much more complex. Basic studies on the role of the pancreas indicate that its external secretion may prevent marginal ulcers and its internal secretions may be a factor in (1) the acute ulcer (Curling's ulcer stress ulcer posterior hypothalamus-stimulated ulcer) or (2) the acute ulcer (Cushing's ulcer anterior hypothalamus stimulated ulcer) and possibly (3) the intractable ulcer (ulcer diathesis Strom-Zollinger Ellison ulcer).

INHIBITION OF GASTRIC ACID SECRETION. Gastric acid secretion is inhibited by the following factors.

1. Viscous factor. The action of the sympathetic fibers either may affect the stomach directly or hold the vagal stimuli in check. This was shown by Cushing in 1932.

2. Antral acid inhibition. It is believed that when excess acid strikes the antrum, it acts as a signal to the parietal cells to slow down in the production of acid. It is of interest that one organ, namely the antrum, can be active in both stimulating and inhibiting the production of acid secretion.

3. Duodenal acid inhibition. This is a postulated mechanism similar to antral inhibition except that the excess acid acts upon the duodenum rather than on the antrum to cause the production of acid. Enterogastrone, a possible inhibitory hormone produced by the duodenum, may be the factor in duodenal acid inhibition.

4. Enterogastrone. This material prepared from the first six to eight feet of fresh hog intestine is a colorless product easily soluble in water. Twenty five mg. of it will suppress histamine-induced gastric secretion in the total pouch dog. In 1948, Greenwood and his associates reported that only 23 per cent of eight Mann-Williamson dogs injected with purified enterogastrone daily for one year developed ulcer whereas 100 per cent of ten such dogs given hog muscle extract injections as controls developed ulcer. Preliminary clinical trial by these authors is suggestive of benefit, but the hormone is not

commonly accepted as being of clinical benefit at present.

Gastric secretion as well as gastrointestinal motility may be inhibited by nervous impulses mediated by way of the sympathetic nerves. Inhibition of secretion of hydrochloric acid is also observed after administration of fats or sugars when they are present in sufficient concentration. This is a result of the action of fats and sugars upon the intestinal mucosa, with the production of the hormone enterogastrone. This natural inhibitory mechanism is made use of in treating ulcer patients. Liberal amounts of fat, usually in the form of cream, are included in such diets. This fat stimulates the release of enterogastrone which inhibits hydrochloric acid secretion and also delays gastric emptying.

REGULATION OF GASTRIC ACIDITY. The formation of gastric acid is regulated by the factors discussed above both those which enhance secretion and those which inhibit it. The acidity of the gastric contents is also related to the rate of removal of parietal secretion by emptying of the stomach, the ingestion of food or antacids, the amount of nonparietal or mucous secretion and the amount of secretion extrinsic to the stomach. The last factor includes swallowed saliva, regurgitated intestinal contents and alkaline bile. The amount of acid secretion at the time of a meal depends on both mechanical and chemical factors concerned with that meal and also with the enthusiasm with which the person ingests the meal. It is thought that all patients with free acid form parietal secretion at about the same concentration (approximately 159 clinical units). However during the course of a meal the level of acidity seldom reaches higher than 50 clinical units, because of the addition of nonparietal secretion and the neutralizing capacity of the ingested food.

Duodenal secretion. The duodenum is supplied with Brunner's glands which resemble histologically the pyloric glands of the distal stomach. The secretion from these glands is a thin, alkaline mucous one which is capable of neutralizing a significant amount of gastric hydrochloric acid. The stimulus for its production is the presence of acid in the duodenum. The vagus nerves stimulate activity of the Brunner's glands while the sympathetic nerves have an opposite effect.

Gastrointestinal motor activity. Motor activity can be observed both when the stomach is empty and when it is full. The move-

Certain personality factors and traits are found in association with peptic ulcer. The ulcer patient is frequently a tense anxious driving, active individual.

Pathogenesis. The available evidence indicates that peptic ulcers of the stomach or duodenum have their origin in the acute ulcer. The acute ulcer or an erosion goes on to form a chronic ulcer unless the balance between the eroding and the defense mechanisms is restored. The study of experimental ulcers has demonstrated that two factors are common to all of these ulcers. These consist of engorgement of the mucosa and necrosis.

Alterations in the secretory and motor functions of the stomach are equally important. Hypersecretion is present with most duodenal ulcers. Wolf and Wolff have observed that hypersecretion is accompanied by hypermotility and engorgement of the mucosa. All of these act to render the mucosa less resistant to trauma and subsequent ulceration.

Pathology. Acute ulcers. Acute ulcers are superficially located and are frequently multiple. They often follow infections, intracranial disease, neurologic trauma or forms of stress. Most of these ulcers heal, but a few become clinically manifest by the development of bleeding or perforation. Some may ultimately form chronic ulcers.

Chronic ulcers. Chronic ulcers account for the majority of peptic ulcers which cause symptoms. They may be located in the distal esophagus, in the stomach or duodenum in the small bowel following gastroenterostomy or in a Meckel's diverticulum when heterotopic gastric mucosa is present (Fig. 35). Esophageal ulceration is rare as a primary entity but is frequently found in association with hiatus hernia. Esophageal ulcers account for 4 per cent of peptic ulcer in autopsy subjects.

Gastric ulcers occur with greatest frequency on the lesser curvature side of the stomach, 85 per cent of gastric ulcers being in this region. Sixty per cent of gastric ulcers are within 6 cm. proximal to the pyloroduodenal junction. The pyloric channel is the site of 12 per cent of gastric ulcers. The cardia is less frequently involved and the greater curvature only on a rare occasion.

Ninety five per cent of duodenal ulcers are within the first 3 cm. of the duodenum. Ulceration can occur in the postbulbar area of the duodenum. Either the anterior or posterior wall of the duodenum may be in-

involved. Duodenal ulcers on the posterior wall are difficult to demonstrate radiologically. Ulcers of the anterior wall are more likely to perforate than those on the posterior wall. Posteriorly located ulcers are more likely to form a walled-off perforation.

The gross appearance of a gastric ulcer is striking. A chronic gastric ulcer usually has a deep crater with sharp edges and a clean base. The base of the ulcer is whitish gray and consists of a thickened subserosal coat. If the ulcer perforates to involve adjacent tissues, the base of the ulcer may be formed by these organs (Fig. 36).

Duodenal ulcers are less easily recognized grossly. A small, white puckered scar on the subserosal coat may provide the only external evidence of the disease. If the ulcer is located on the posterior wall it may be impossible to diagnose it by palpation. A duodenostomy with inspection of the duodenum may be necessary to learn whether an ulcer is located in this area.

The microscopic features of all peptic ulcers are similar. There is a loss of the mucosa down to the muscularis mucosa. The lining of a typical ulcer crater has four zones: purulent exudate, a thin zone of fibrinoid mucosa, granulation tissue, and an outer dense layer of scar tissue.

Symptoms and Signs. Pain. Pain is the outstanding symptom of peptic ulcer. The pain is frequently sufficiently characteristic to indicate the diagnosis almost without further examination. Ulcer pain has four

CLASSIFICATION OF PEPTIC ULCER

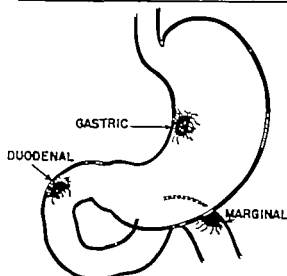


Figure 35 Diagram showing the most frequent locations of peptic ulcer

Mechanical factors are also important in the development of an ulcer. Ulcers tend to occur where there is a maximum degree of trauma, in the stomach on the lesser curvature and also in the duodenal cap where the acid chyme contents are ejected from the stomach.

Local tissue resistance and defense factors.

Local tissue resistance has an important role in the development and location of an ulcer. If only general factors were operative then the entire lining of the stomach would be digested away by the gastric contents. However, peptic ulcer is a localized disease and it is probable that in an area where an ulcer develops there is some breakdown of the local defense factors. This may be the result of ischemia inasmuch as the neutralizing effect of alkaline blood is an important defense factor. The enzyme systems of the stomach will also contribute to the defense of the mucosa. Urease which is concentrated in the surface cells of the stomach, catalyzes the breakdown of urea to form ammonia and carbamate, both of which aid in neutralizing parietal secretion.

Systemic and constitutional factors. Hereditary influences, hormonal factors and neurogenic and personality factors all have some bearing on the development of an ulcer. Peptic ulcer is not a hereditary condition, but it does tend to run in families.

The development of ulcers is influenced by local gastrointestinal hormones and by hormones arising in more distant parts of the body. Gastrin, a hormone similar to histamine, produced by the antrum, stimulates secretion of hydrochloric acid. There is no evidence that antral hyperfunction has an important role in the development of peptic ulcer. Enterogastrone produced by the duodenum, inhibits gastric secretion and gastric emptying. Enterogastrone and similar substances have not been demonstrated to be effective in man in the prevention of ulcer.

Peptic ulceration can occur during prolonged administration of corticotropin or cortisone. This observation has suggested that adrenal hyperfunction may have some part in the etiologic background of peptic ulcer. ACTH markedly increases uropepsin production and also stimulates the production of hydrochloric acid. It has been suggested that stress may induce the hypothalamus to secrete some agents which stimulate the production of the ACTH. Stimulation of the anterior hypothalamus is accompanied by a rise in gastric acidity which can be abolished by a bilateral vagotomy while

stimulation of the posterior hypothalamus produces an increase in gastric acidity which is eliminated by adrenalectomy.

A pancreatic islet cell adenoma was described in 1953 which appeared to induce gastric hypersecretion and peptic ulceration that resisted every therapeutic measure short of total gastrectomy. This ulcerogenic adenoma of the pancreas islets is composed of non-beta cells and does not secrete insulin.

Study of the clinical cases with ulcerogenic tumors of the pancreas has resulted in the definition of a diagnostic triad. (1) a fulminating ulcer diathesis persists despite the most intense medical therapy, radiation or radical operative procedure short of total gastrectomy. The usually definitive surgical measures, including vagotomy below and above the diaphragm, and the most radical subtotal gastrectomy have uniformly failed to prevent recurrent ulceration so long as gastric mucosa remains under the influence of this islet adenoma, (2) marked gastric hypersecretion is characteristic with twelve-hour overnight resting volumes of 2 to 3 liters in the absence of any pyloric obstruction. Acid is uniformly present and often its concentration as well as its volume is very high. Twelve hour acid production of more than 300 mEq has been observed as compared to the normal of 18. Such large volumes of secretion promptly recur post-operatively so long as gastric mucosa is retained in the presence of functioning tumor. (3) when a non-insulin-secreting, non-beta cell islet pancreatic adenoma is demonstrated at operation, the clinical triad is complete.

The nature of the influence which the ulcerogenic adenomas exert upon gastric mucosa strongly suggests a hormone. This is so far unidentified but must be powerful and probably acts directly upon the gastric secretory cells. That the pancreas plays a role in gastric secretory function has long been suspected but never proved. If such an influence could be identified, it would have important implications not only for the rare ulcerogenic tumor patient, but also for the great number of patients afflicted with benign peptic ulcer and all its complications.

The sex hormones appear to be related to the development of peptic ulcer. Ulcer is far more common in men than in women. Ulcer is rarely associated with pregnancy.

Acute ulceration of the upper gastrointestinal tract can occur after intracranial injury or disease. These ulcers resemble the acute ulcers following massive infection or in association with burns or other forms of stress.

approximately 15 per cent of patients. It is more common in gastric than in duodenal ulcer. Perforation without previous ulcer distress can occur. This is more frequent with acute ulcers.

Symptoms as related to the location of the ulcer. Esophageal ulcers present substernal pain and dysphagia. Pain in esophageal ulcer may occur on ingestion of food followed by a period of relief and then by a period of pain. Dysphagia is also common in patients with esophageal ulcer. It is most likely to occur on swallowing solids and is related to the inflammation and spasm in the region of the ulcer. The hiatus hernia, which is usually present, contributes to the dysphagia.

The symptoms of gastric ulcer tend to be less characteristic and more bizarre than those of duodenal ulcer. If an ulcer is located high in the stomach, it may be completely asymptomatic. When the ulcer is flat or shallow the pain is poorly localized by the patient. Ulcers located in the pyloric channel present bizarre symptoms: nausea and vomiting may be the primary complaints.

The symptoms of duodenal ulcer are rather characteristic. They consist of burning, aching or gnawing sensation located in the epigastrium, usually just to the right of the midline. The pain can usually be localized by covering the site of it with one finger. The pain is usually steady, lasting from fifteen minutes to an hour and it appears from one to four hours after meals. It is characteristically absent before breakfast.

Ulceration involving the stoma after the performance of a gastroenterostomy differs from the symptoms presented by an uncomplicated ulcer. The pain of marginal or stomal ulcer is located down and to the left from the site of the original ulcer pain. Nausea and vomiting are frequent because of edematous obstruction to the stoma. Loss of weight is common and diarrhea may occur. If either of these is severe, it may suggest the development of a fistula between the stomach, small bowel and colon. Gastroenterocolic fistula fortunately is a rare complication of marginal ulcer occurring in only about 1 per cent of the patients. In addition to the weight loss and diarrhea, edema due to hyperproteinemia is a common occurrence.

Diagnosis. Diagnosis in peptic ulcer depends primarily on obtaining a typical history of recurrent ulcer distress. Physical examination is much less important. Labora-

tory studies, including gastric analysis and stool examination may be helpful. Esophagoscopy and gastroscopy have a definite although limited value. The major diagnostic contribution is that of the radiologist who is called upon to confirm the clinical impression of ulcer. In addition the radiologist can detect ulcers which are asymptomatic and be of value in determining the incidence of complications and the results following therapy.

Physical examination. Physical examination in patients with uncomplicated ulcer may demonstrate no abnormality. Sometimes a patient can point to the area where ulcer pain has occurred. Occasionally there is tenderness on deep palpation over that area. The chief value of physical examination is in excluding other conditions accompanied by physical findings such as an enlarged liver or abdominal masses which might suggest some diagnosis other than peptic ulcer.

Laboratory examination. Gastric analysis is of most value when the physician is confronted with the problem of distinguishing a benign gastric ulcer from ulcerating gastric carcinoma. The usual findings on gastric analysis are hyperacidity with duodenal ulcer and somewhat lower acidity than normal with gastric ulcer. Patients with gastric carcinoma frequently have anacidity. The levels of gastric acidity are of little or no value in differentiating gastric ulcer from duodenal ulcer in the individual patient. Stool examination is also useful, particularly in the differential diagnosis of gastric ulcer and gastric carcinoma. Although occult blood may occur in the stool in patients with benign peptic ulcer the finding of persistent blood suggests that an ulcerating carcinoma is present. The newest technique in diagnosis is the study of the exfoliated mucosal cells from the lining of the stomach. These may be obtained by lavage, by abrasion or by use of the mucolytic agents. With the latter two methods the accuracy of distinguishing carcinoma is in the neighborhood of 85 per cent.

Endoscopic examination. Esophagoscopy is of value in differentiating esophageal ulcer from other lesions of the distal esophagus. A hiatus hernia with secondary ulceration of the esophagus is the usual finding, as primary ulcer of the esophagus is rare. Esophagoscopy is of value in the diagnosis and treatment of stricture formation which follows long standing inflammation of the esophagogastric junction. Some so-called

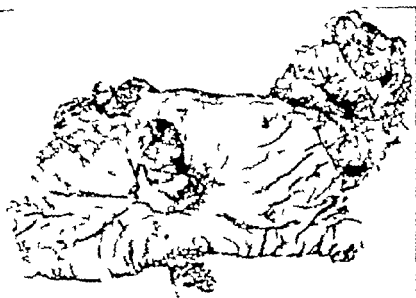


Figure 38 Photograph of gross specimen showing a chronic gastric ulcer which perforated into the pancreas (Barboiska, C. J. and Texter E. C., Jr. *Peptic Ulcer—Diagnosis and Treatment*. Boston, Little, Brown & Company)

typical characteristics the nature and in intensity the radiation and location, the rhythm and the periodicity

Ulcer pain is usually described as a gnawing, aching or burning sensation. It may resemble hunger contractions. It is located in the upper abdomen near the midline and is usually confined to a relatively small area. Pain of duodenal ulcer is better localized by the patient than that arising from gastric ulcer. Pain arising from a marginal ulcer is shifted downward and to the left from the site of the previous ulcer pain. If walled-off perforation occurs radiation of the pain to the back or other adjacent areas is common.

The rhythmic appearance and disappearance of pain in relation to the state of fullness or emptiness of the stomach is characteristic. The pain usually comes on following a meal from thirty minutes to two hours. It gradually becomes more severe for half an hour or an hour and, if not relieved by medication, the pain may pass off spontaneously. As the ulcer becomes more severe pain may occur at night. This classically wakes the patient from sleep from one to four hours after retiring, most usually about 1 A.M. Ingestion of food or alkalis at this time will relieve the pain.

Periodicity of the pain also is typical. The pain may be present for days or weeks at a time and then the patient may be symptom free for long intervals. Recurrences are most frequent in the spring or fall months. Recurrences occur in the months in which

greatest temperature change occurs. As the ulcer becomes more severe, the periods of remission between acute attacks become shorter.

Three theories have been proposed to explain the mechanism of ulcer pain. One of these holds that the pain is the direct result of chemical irritation of nerve fibers by the gastric acid another that ulcer pain is the result of motor disturbances of the stomach; and the third, that ulcer pain is the result of a combination of both these factors and, perhaps in association with other factors. The present evidence seems to indicate that, although acid is necessary for the production of an ulcer and ulcer pain, it is probable that some alteration in gastroduodenal motility is necessary for pain to develop. Engorgement of the mucosa of the stomach or duodenum may lower the pain threshold.

Other symptoms. Although pain is the paramount symptom of peptic ulcer other symptoms can occur. Nausea and vomiting are not common symptoms of the usual peptic ulcer but may result if the ulcer is located in the pyloric channel area or if there is obstruction to the stomach with resultant gastric retention. Constipation may be a presenting complaint, but it is probable that the constipation is more closely related to the type of diet which the ulcer patient takes rather than to the disease per se. Bleeding manifested either by hematemesis or melena may be the initial symptom of peptic ulcer. This occurs as an initial complaint in

ulcer patients. These complications include perforation walled-off perforation hemorrhage and obstruction. The majority of bleeding episodes respond readily to medical management and some of the patients with pyloric obstruction respond to medical management. The remainder of the ulcer complications require surgery. This is necessary in approximately 10 to 15 per cent of patients with recurrent peptic ulcer.

Perforation Perforation is a rare complication affecting about 2 per cent of ulcer patients. It may occur in subjects of any age but is more common in those in the middle years of life. The onset of perforation is marked by the development of sudden excruciating abdominal pain which may be followed by collapse. The pain may radiate to both shoulders owing to irritation of the undersurface of the diaphragm by free air or gastroduodenal contents. The immediate effect of perforation is the production of chemical peritonitis, but, with the passage of time, the peritoneum becomes infected and the prognosis more serious.

Boardlike rigidity of the abdomen with exquisite tenderness is present on examination. The patient lies still with knees flexed to minimize the pain. The temperature and blood pressure in the early period may be subnormal, but, if bacterial peritonitis supervenes, the temperature may become elevated.

Patients who present similar although less acute symptoms are thought to have subacute perforation. Here the perforation is rapidly sealed over by adhesions with consequent clinical improvement. Roentgenographic studies show pneumoperitoneum in about two-thirds of patients with acute perforation. When possible, the films should be exposed with the patient sitting up or lying on the left side.

Although the differential diagnosis of a perforated ulcer is seldom difficult, it may be simulated by other conditions. Biliary renal or small bowel colic presents pain but is not associated with boardlike rigidity. Coronary thrombosis may be differentiated by the radiation of pain to the left shoulder.

The condition which is most difficult to differentiate is pancreatitis. Here, even though severe pain is present, boardlike rigidity is uncommon and shock is less likely to occur. Perforation of the other abdominal hollow viscera can also simulate perforated ulcer.

Walled-off perforation. Walled-off perforation results when the process of perforation

involves ulcers located on the posterior wall of either the stomach or duodenum. Instead of perforating into the free peritoneal cavity these ulcers penetrate to involve the adjacent structures most frequently the pancreas and liver. Approximately 5 per cent of ulcer patients develop this complication.

The development of walled-off perforation is marked by either a change in the usual pain pattern the development of increasingly severe ulcer pain or the radiation of pain to the back. The physical findings do not differ markedly from those present in uncomplicated peptic ulcer. Moderate elevations of the serum amylase may be observed.

The roentgenographic demonstration of a pouch or pocket of air projecting from the stomach is suggestive of a walled-off perforated gastric ulcer. The x ray signs of a walled-off perforated duodenal ulcer do not differ significantly from those ordinarily present.

The change in clinical features suggests the development of a walled-off perforated ulcer. However a change in pattern may occur if gastric obstruction occurs or if the patient develops an ulcer in the pyloric channel. Walled-off perforation also must be differentiated from pancreatic disease, biliary tract disease carcinoma of the colon and disease of the spine.

Hemorrhage Hemorrhage is the most frequent complication of peptic ulcer. Most ulcer patients have periodic minor bleeding episodes and 20 to 25 per cent have manifest gastrointestinal hemorrhage. The presenting symptom may be hematemesis or melena. Hematemesis the vomiting of material resembling coffee grounds, is most likely to occur in patients with gastric ulcers or if the hemorrhage is a very large one. Melena is recognized by the passage of frequent bulky black tarry sticky stools.

The clinical manifestations depend upon the amount of blood lost and the rate of bleeding. Pallor is common and, if more than 30 per cent of circulating blood volume is lost, irreversible hemorrhagic shock develops. Endoscopy is rarely carried out during the acute bleeding episode. Roentgenographic studies are also deferred until the acute bleeding is stopped, in most instances. However if the bleeding is brisk and surgery is considered, roentgenographic study may be carried out without additional risk to the patient.

Hemorrhage from peptic ulcer accounts for 85 per cent of instances of massive gas-

esophageal ulcers may actually be ulcers of the herniated supradiaphragmatic portion of the stomach.

The diagnostic value of gastroscopy is limited to ulcers located in the stomach and even here it may be impossible to visualize an ulcer which is located in the gastroscopically "blind" area. Gastroscopy is also of value in aiding differentiation between apparently benign gastric ulcers ulcerating malignant lesions and infiltrative lesions of the wall such as gastritis and lymphoma.

Despite its limitations the diagnostic examination of a patient with a gastric ulcer is not complete unless gastroscopy is carried out. Recently it has been possible to take a biopsy specimen from the stomach and thus has further increased the value of this procedure.

Röntgenography The roentgenographic signs associated with ulceration depend somewhat on the location of the ulcer. In esophageal ulcer one may find a fleck of barium adhering to the esophageal wall. The majority of esophageal ulcers are associated with some other disorder most frequently hiatus hernia. The roentgenographic diagnosis of esophageal ulcer is difficult.

The primary sign of gastric ulcer is the demonstration of a niche or bud projecting from the stomach. The niche is usually rounded and has a comparatively smooth outline. Rarely the contour may be irregular even though the lesion is benign. Size which was once considered to be a valuable criterion in distinguishing between benign and malignant ulcers is of little value. Indirect signs of ulceration may be present, including localized spasm opposite the ulcer alterations of the rugal folds about the ulcer and localized tenderness in the region of the ulcer. Motor disturbances may also be observed and, if the ulcer is near the pyloric channel, gastric retention can result. If the ulcer is situated higher in the stomach, hyperperistalsis may be observed.

The roentgenographic signs of ulceration in the upper portion of the stomach above the incisura angularis are more easily delineated than those below the incisura angularis. Gastritis of the antrum is frequently present with ulcers below the angularis. Direct evidence of a crater is difficult to obtain and frequently the diagnosis must be based on indirect or secondary signs of ulceration.

The direct sign of a duodenal ulcer is the demonstration of a deformity in the outline of the duodenum. The deformity may as-

sume a variety of patterns, including niche formation, retraction in the region of the niche a defect in the curvature opposite the niche an eccentrically placed pyloric lumen and pseudodiverticular dilatation. The most commonly observed deformity is a pinching in of the sides of the duodenum which ultimately form the clover leaf pattern. A crater may be demonstrated at the middle of the clover leaf with folds radiating into it. Indirect signs of duodenal ulcer include irritability of the duodenum, active gastric peristalsis and reflex gastric spasm. Localized tenderness at the site of the duodenal ulcer itself is of little value in the diagnosis of a duodenal ulcer.

The x ray study is of particular value in following the progress of healing of gastric ulcers but is of little value in following the progress of duodenal ulcer. Once a deformity has developed in the duodenum, it is likely to become persistent regardless of whether the ulcer is healed or not.

There are definite limitations to the value of the roentgenologist in the diagnosis of peptic ulcer. Alterations in the stomach or duodenum may be present from extrinsic causes and not as the result of primary ulceration. The roentgenologist may acknowledge that the interpretation of the roentgenographic findings depends partly on the patient's clinical history and not entirely on what is demonstrated on the films.

Differential Diagnosis. Even though the clinical picture of ulcer is fairly characteristic, on occasion it must be differentiated from a variety of conditions. Among these are hiatus hernia, esophagitis, atrophic gastritis, hypertrophic gastritis carcinoma of the stomach, gallbladder disease and conditions involving the pancreas and intestines.

The most important conditions to be differentiated are those of nonulcer dyspepsia and carcinoma of the stomach. Nonulcer dyspepsia, or functional dyspepsia, denotes symptoms suggesting ulcer in patients in whom no definitive evidence of ulceration can be established. Patients with gastritis may present similar symptoms. Both these groups of patients differ from ulcer patients in that they fail to obtain complete symptomatic relief although they are improved by being placed on the dietary and medical management. Ulcerating carcinomas may simulate a benign gastric ulcer and render a differential diagnosis difficult.

Complications. Complications which require specific attention and hospitalization develop in approximately 30 per cent of

the stomach are necessary to distinguish benign gastric ulcer from ulcerating carcinoma. The gastric acidity in gastric ulcer is usually lower than normal but not as low as the anacidity frequently present with carcinoma. Persistent failure to demonstrate acid after histamine stimulation in the presence of a gastric crater strongly suggests that the lesion is malignant.

The persistence of occult blood in the stool is unusual in patients with benign gastric ulcer and again suggests that the patient has a malignant process.

The study of exfoliated cytologic material from the stomach is of great value in distinguishing ulcerating carcinoma from a benign gastric ulcer. The material which is obtained by saline lavage after the use of an abrasive balloon, or following instillation of mucolytic agents, is stained according to the Papanicolaou technique. The malignant cells differ in their cytologic characteristics, having larger more deeply staining nuclei and other evidences of malignant change.

If the gastric lesion is in a gastroscopically visible area, gastroscopy can be of immense value. The benign gastric ulcer presents a punched-out appearance with smooth edges and a clean-looking base, whereas the malignant ulcer may have heaped-up edges, irregularities of the margin and evidence of bleeding.

Three-quarters of all gastric craters occur along the lesser curvature of the stomach.

The prepyloric area is also a common site for benign ulcers and benign ulcers also occur in the cardiac portion of the stomach and in the pylorus. Certain criteria are useful in distinguishing malignant from benign ulcer. The malignant ulcer frequently presents a meniscus effect with the crater being superimposed on the top of a filling defect projecting into the stomach. Alterations in the pliability are frequent in malignant ulcer. Size is not a very useful criterion in distinguishing benign from malignant lesions. Occasionally one sees extremely large benign ulcers (Fig. 37).

The response to treatment is useful in distinguishing between benign and malignant ulcers. Even though malignant ulcers may show some improvement and decrease in size, they do not go on to complete healing such as occurs with most benign ulcers. Perforated walled-off ulcers may not heal despite good medical management.

Treatment. General considerations. The aims of treatment are threefold; to relieve symptoms, to heal the ulcer and to prevent recurrences. Relief of symptoms is achieved rather easily but complete healing of the ulcer requires more prolonged and intensive treatment. Approximately forty days are necessary for the healing of an ulcer. The prevention of recurrences is a still more difficult problem.

Good ulcer management is based on rest and sedation, diet, the use of antacids and



FIGURE 37 A, X-ray appearance of large benign gastric ulcer. B, X-ray appearance of same ulcer two weeks later showing healing (Barborka, C. J. et al., Quart. Bull., Northwestern Univ. M. School, vol. 28)

trointestinal bleeding. It must be differentiated from bleeding as a result of esophageal varices, carcinoma or blood dyscrasias. The bromsulphalein test is valuable in distinguishing bleeding from esophageal varices from bleeding from peptic ulcer. Bromsulphalein retention is likely in cirrhotic patients but rarely is persistent in patients with bleeding peptic ulcer. Bleeding can also occur from gastritis or hiatus hernia.

Obstruction. Transient obstruction of the distal portion of the stomach is a fairly common complication of ulcer. Edema, spasm and stenosis of the pyloric segment may all contribute to the gastric retention.

The initial clinical features of obstruction are not characteristic and the diagnosis may be made only after roentgenographic examination or aspiration of the stomach. As the obstruction becomes more severe, changes in the character of ulcer distress may be observed. The pain has less relation to meals. Breakfast is the best tolerated meal of the day and the patient complains of increasing fullness as the day goes on. Vomiting may occur in the late evening or early hours of the morning. The vomitus usually contains food remaining from the previous meal. Weight loss and dehydration are late signs of obstruction. Visible peristalsis which passes from the left to the right is characteristic of obstruction at the pylorus.

The barium meal is valuable in determining whether obstruction is present. Pyloric obstruction is present if there is significant gastric retention at six hours, or any retention at twenty four hours. Retention of more than 100 ml. of gastric secretion in the fasting stomach, or more than 750 ml. during the twelve-hour overnight period of continuous suction, is usually indicative of gastric retention and obstruction.

Gastric retention due to an obstructing ulcer can be simulated by obstructing carcinoma of the distal stomach and hypertrophic pyloric stenosis. Pyloric channel ulcers are likely to cause delay in gastric emptying.

Gastric Ulcer and the Ulcer-Cancer Problem. *Differences between gastric and duodenal ulcers.* Gastric ulcer is clinically less frequent than duodenal ulcer although autopsy statistics indicate that the frequency of the two lesions is identical. Gastric ulcers tend to come on at a later age than do duodenal ulcers. The etiologic factors also differ. The acidity in gastric ulcer patients is lower than in normal individuals and consider-

ably lower than the elevated values seen in duodenal ulcer patients. The pathogenesis of gastric ulcer seems to be related more to decreased mucosal resistance of the stomach than to overactivity of the aggressive acid-pepsin interaction. This observation is also supported by the fact that gastric ulcers occur in the area of the stomach subject to most trauma and supplied least well with blood.

Relation to gastric carcinoma. Because of the not infrequent association of gastric ulcer and gastric carcinoma, it has been suggested that gastric ulcers may undergo malignant degeneration. There is no proof of this concept and it is now believed that malignant ulcers were malignant from the beginning and not the result of degeneration of a previously benign lesion. The problem is one of differentiating the ulcerating malignant lesion from a benign gastric ulcer. The reported incidence of malignant ulcer in patients having apparently benign lesions varies between 8 and 20 per cent. Smith, Boles and Jordan, reporting on 1000 patients with the clinical diagnosis of benign gastric ulcer noted that 8.8 per cent of these patients ultimately were found to have ulcerating malignant lesions. The incidence of malignancy was 16 per cent in patients who developed a recurrent ulcer. The accuracy of diagnosis is improved by the more frequent use of exfoliative cytologic examination. If gastric ulcer patients are studied carefully including exfoliative cytologic study with the specific aim of excluding patients who have malignant ulceration, the error in diagnosis should be very small—perhaps as low as 1 per cent.

Diagnosis of gastric ulcer. The diagnosis of gastric ulcer is based upon the clinical features presented by the patient, roentgenographic study of the lesion, gastroscopic findings and a study of exfoliated cells from the stomach.

The history presented by the gastric ulcer patient resembles that of the duodenal ulcer patient except that it is less characteristic. If the ulcer is located high in the stomach, it may be asymptomatic. Ulcers located in the distal end of the stomach are frequently not recognized as such since the major complaint of these patients is nausea and vomiting.

The physical examination is of little value. Localized tenderness may be present if the ulcer is located in an area which is accessible to palpation.

Gastric analysis, stool examination for occult blood and study of exfoliated cells of

relationship with sympathetic understanding of the patient's problems is essential to good management. Mental and emotional conflicts have an important bearing on recurrences. Occasionally a change in environment or occupation may be necessary. If the patient can be convinced that he will be a more productive individual if he remains healthy by taking annual vacations and long week ends and by finding other outlets for his energy much can be accomplished.

Medical treatment of uncomplicated ulcer. For the patient with only occasional mild distress, only moderate restrictions are necessary. These consist of the use of an ambulatory diet with interval antacids and sedation. For patients with moderately severe distress which is not responding to simple management the addition of the anticholinergic drugs to the above-described program frequently produces symptomatic relief. However if symptomatic relief is not achieved promptly the patient should be hospitalized where he can be treated under optimum conditions.

Medical treatment of complicated ulcer.
PERFORATION. Closure of perforation as soon as possible after the diagnosis is established is indicated for perforated ulcer. On occasion, gastric resection is deemed advisable if the patient has had previous perforations or if the closure of the perforation presents technical difficulties. The mortality rate of perforated ulcer is below 5 per cent. It rises with increasing time after perforation.

The expectant-suction method of treatment of perforated ulcer should be limited strictly to patients having had perforation many hours prior to the institution of treatment or to those in whom surgical closure cannot be undertaken. Antibiotics are administered concurrently. Increasing rigidity and tenderness are indications for operative intervention.

WALLED-OFF PERFORATION. Intensive treatment in the hospital is indicated for patients suspected of having a walled-off perforated gastric or duodenal ulcer. Overnight gastric suction and the ulcer I diet during the day time may be supplemented by anticholinergic drugs. Many of these patients are refractory to the most rigid medical program and surgical treatment becomes necessary. The major indication for surgery is intractability although some of these patients also have had previous bleeding or obstruction.

HEMORRHAGE. The bleeding patient should be hospitalized and checked for the presence of shock. The blood pressure and pulse

should be recorded at hourly intervals and the patient's blood typed and crossmatched in the event transfusion should be necessary. The hematocrit reading or hemoglobin estimation should be made on admission and periodically thereafter to follow the status of the hemorrhage. Elevation of the blood urea nitrogen will also result if significant bleeding exists. Another index of the degree of bleeding is the number of stools passed daily. The blood in the stools acts as an irritant and more frequent stools occur while the patient continues to bleed.

If there are signs of shock an infusion should be started while whole blood is being obtained. If the blood volume decreases in spite of administration of blood it indicates that hemorrhage is proceeding at a rate faster than the blood is replaced and surgical therapy must be considered.

Dietary treatment, either of the ulcer I type or the Meulengracht type of diet, may be started as soon as the patient feels able to eat.

Although the majority of bleeding patients respond to medical management surgery may be necessary for patients who are bleeding more rapidly than blood can be replaced. The best results in treating bleeding ulcer patients are obtained when the medical and surgical departments act as a team. The mortality rate is lower if surgery is carried out in the interim phase rather than when the patient is acutely bleeding.

OBSTRUCTION. The medical treatment of pyloric obstruction consists of aspiration of the stomach in conjunction with giving a liquid diet and the administration of antacids and antispasmodics. Electrolyte disturbances are less prone to occur if intermittent aspiration, such as overnight suction is used rather than continuous aspiration. The tube may be removed during the day and the patient fed a liquid diet of milk and cream at hourly intervals and given antacids according to the ulcer I program. The amount of aspirate obtained during each twelve-hour period serves as an index of the progress of treatment. Roentgenographic study which can be repeated at intervals of five to seven days, also can be utilized to determine whether progress is being made with treatment.

Surgical treatment is necessary for patients who do not respond to medical management. Correction of nutritional problems and electrolyte disturbances must be carried out prior to the surgical treatment. The operations most frequently performed are gas

anticholinergic agents restrictions of coffee alcohol and tobacco and the use of psychotherapy

Rest and sedation. Rest is essential to good ulcer management. It is desirable that treatment be carried out in the hospital where the patient is separated from environmental stress. Hospitalization in addition to providing rest also has other advantages. It enables the physician to teach the patient the basic principles of ulcer treatment which are essential to maintaining a satisfactory program. Sedation obtained by means of phenobarbital 30 mg. three or four times a day is usually prescribed.

Diet. Dietary treatment is based on avoiding foods which stimulate the secretion of hydrochloric acid, the administration of foods which neutralize gastric acid, and elimination of irritating foods from the diet. The dietary principles of most ulcer programs are similar. They represent modifications of the Sippy type diet which are used in combination with antacid drugs. In most hospitals ulcer diets are divided into ulcer I, ulcer II, and ulcer III diets. The ulcer I diet consists of 3 ounces of a mixture of half milk and half cream given every hour during the day. One of the nonabsorbable antacids is administered every hour on the half hour. This diet is usually continued for one or possibly two weeks. It is deficient in vitamins and minerals, particularly the vitamin B and C groups. The second-stage ulcer management, ulcer II, permits the addition of bland foods such as cheese, eggs and cereal, so that the patient now is taking a morning, midday and evening meal with interval feedings of milk and cream and antacids being continued. The second-stage diet is usually continued for about one or two weeks, at which time the patient is shifted to the ambulatory-type program, ulcer III. This diet includes puréed vegetables, baked potato, baked apple and certain meats. Raw fruits and vegetables, highly seasoned foods and extremely hot or cold liquids are excluded.

Antacids. Antacids are substances which lower the acidity of the gastric contents. The most commonly employed antacids are calcium carbonate and the aluminum and magnesium compounds. Calcium carbonate is given in 2- to 4-gm. doses. It has a tendency to produce constipation and magnesium carbonate or magnesium oxide must be given from time to time. The combination of calcium carbonate with magnesium oxide and milk has good neutralizing capacity.

Aluminum hydroxide, which is frequently combined with magnesium trisilicate to combat the constipating properties of aluminum hydroxide, is the most commonly used. Neutralization of gastric acidity by aluminum hydroxide is not great, but this compound does have demulcent properties as does magnesium trisilicate. The usual dose is 15 to 30 cc. The most effective tablet antacid is dihydroxy aluminum aminoacetate, which is taken in 1-gm. doses.

The frequency of antacid administration varies with the severity of the ulcer and the type of management. With first-stage management, antacids are given at hourly intervals from 7:30 A.M. until 9:30 P.M. When second-stage management is used, the antacids are continued at hourly intervals between meals but omitted at mealtimes. When used with ambulatory program, antacids may be given one-half hour before meals and an hour and a half after meals or at any other time the patient has ulcer distress.

Anticholinergic agents. These agents can be divided into two groups: the tertiary amines, such as atropine and the quaternary amines such as methantheline. These agents decrease gastric secretion and inhibit gastrointestinal motor activity. It is recommended that they be employed as adjunct therapy along with the diet, antacids and sedation. They are not necessary for all patients. Symptomatic relief following their use is usually gratifying, but the eventual course of the patient with peptic ulcer is not altered by their long-term administration. They are administered in dosage of from one to two tablets four times a day prior to meals and bedtimes. The anticholinergic agents are contraindicated in the presence of hemorrhage, pyloric stenosis and suspected perforation and in patients with prostatic hypertrophy or glaucoma.

Restriction of coffee alcohol and tobacco. Most physicians feel that better ulcer management is achieved with the curtailment or elimination of coffee, alcohol, caffeine-containing beverages and smoking.

There are many excellent studies on the relationship of smoking to peptic ulcer. It appears unlikely however on careful analysis of the varied statistics that smoking is an important and direct cause of the disease. On present evidence, it seems more reasonable to suggest as a working hypothesis that the effect of smoking is to interfere with the healing of the ulcer and to help maintain its chronicity.

Psychotherapy. A good physician-patient

quate medical management should be considered for surgery. Many of these patients have walled-off ulcers which respond very poorly to medical treatment.

SUSPICION OF CARCINOMA Patients with gastric ulcers which do not heal within a brief time with medical management in the hospital, or who present clinical criteria suggestive of malignant disease are candidates for surgery. The operation of choice is gastric resection with removal of the ulcer.

COMPLICATIONS SECONDARY TO PREVIOUS SURGERY Surgical treatment is necessary for many patients with marginal or recurrent ulcer which followed previous gastroenterostomy or gastric resection. It also is occasionally necessary to operate in order to revise previous surgical results in an attempt to eliminate severe postsurgical disability.

CHOICE OF OPERATION The most commonly employed operation is gastric resection either of the Billroth I or II type. The commonly employed forms of this are shown in Figure 38. Section of the vagus nerves in conjunction with a drainage operation, either gastroenterostomy gastric resection or pyloroplasty is also employed. Wangensteen has suggested the use of tubular resection of the stomach in an effort to maintain gastroduodenal continuity and minimize postsurgical complications.

COMMON TYPES OF GASTRIC RESECTION

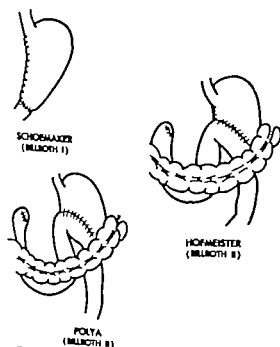


Figure 38. Diagrammatic representation of commonly employed types of gastric resection (Barbarik, C., and Dexter, E. C., Jr. *Peptic Ulcer—Diagnosis and Treatment*. Boston, Little, Brown & Company)

Results of Surgery Esophageal ulcer Massive hemorrhage perforation intractability to medical management and stricture formation may all require surgery. The procedures employed include correction of hiatus hernia partial resection of the esophagus subtotal gastrectomy and vagotomy and gastroenterostomy. The results of surgery for esophageal ulcer are fairly satisfactory.

Gastric ulcer Surgical treatment in gastric ulcer is very satisfactory. Marginal ulcer is rare. The mortality rate is approximately 5 per cent.

Duodenal ulcer After an adequate resection of two-thirds to three-quarters of the stomach, approximately 85 per cent of patients will have satisfactory results. Marginal ulcers can be expected to develop in 4 to 8 per cent of patients and an additional number have postoperative complications. The average mortality rate is between 3 and 5 per cent.

On the basis of a shorter follow-up period, the results of treatment following vagotomy and gastroenterostomy appear to be about as satisfactory with 85 per cent of patients obtaining a good result. Recurrences take place in approximately 5 per cent of patients and postoperative complications, mainly diarrhea, occur in an additional 7 per cent. The operative mortality is about 1 per cent. The operations of partial gastrectomy and vagotomy have also been combined with encouraging initial results.

Marginal ulcer The surgical treatment depends upon the type of surgery previously employed. In the presence of a gastroenterostomy the gastroenterostomy should be taken down and adequate resection of either the Billroth I or II type should be performed. Vagotomy may be performed in addition to gastric resection. If the marginal ulcer followed an adequate resection, the ulcerated area should be resected and a vagotomy performed. The results of treatment after these procedures are satisfactory.

Postsurgical Management. Careful post surgical management is necessary to prevent the development of complications. Dietary treatment should be continued for a variable time after surgery. If the diet is liberalized too rapidly after operation, symptoms may develop which are related to the altered size of the stomach and its lack of storage capacity.

Marginal ulcers may develop after either gastric resection or vagotomy and gastroenterostomy. Approximately 1 per cent of pa-

tric resection with or without vagotomy or gastroenterostomy and vagotomy.

Philosophy of Ulcer Management. Medical treatment should be the basic treatment for gastric ulcer with surgery being reserved for patients whose ulcers do not heal or who present evidence of malignant disease. Many surgeons, however, advocate surgery for all gastric ulcer patients.

The arguments presented in favor of immediate operation for all gastric ulcer patients are as follows: There has been little improvement in the survival rate of patients with carcinoma of the stomach owing primarily to failure to make a diagnosis and obtain surgical cure before metastasis developed. Radical resection in all patients with gastric ulcer before the diagnosis of cancer becomes obvious may salvage an appreciable number of patients, even though many so treated cannot be proved to have had a malignant ulcer. One cannot make a distinction between benign and malignant ulcer with sufficient accuracy to permit conservative treatment and continued observation. The results of gastric resection for gastric ulcer are uniformly good. To delay surgery until the results of medical treatment become evident is hazardous as far as the optimum time for resection is concerned.

A cure can also be made against immediate surgical intervention. Even though there are no diagnostic criteria absolutely indicative of benignancy, the diagnosis can be made with a high degree of accuracy. Complete remission of all signs and symptoms with the disappearance of the ulcer as demonstrated by x-ray findings and gastroscopy, the presence of free acid and the disappearance of occult blood in the stool, all suggest that the patient has a benign lesion. Patients without evidence of malignancy should be given a trial of four to six weeks of strict medical management in the hospital. This period is not sufficiently long to alter the five year cure rate for carcinoma which is low to begin with. If the patient's ulcer does not heal within this time, surgery is then recommended.

If, however, the patient has persistent achlorhydria with histamine administration, a positive Papanicolaou smear, evidence of roentgenographic changes suggestive of a malignant lesion or an ulcer on the greater curvature or in the prepyloric region, immediate operation should be advised. The diagnostic error of mistaking an ulcerating malignant lesion for a benign lesion should be at least as low as the usual surgical mor-

tality, namely 3 to 5 per cent, and, in the experience of some utilizing the recent diagnostic aids, the error in diagnosis may be as low as 1 per cent.

Patients with gastric lesions must be hospitalized and maintained in the hospital either until complete healing takes place or until a decision can be made as to whether surgery should be employed. Careful study in the hospital, including frequent roentgenographic examinations, gastroscopy, exfoliative cytologic study of the material of the stomach, stool examinations for occult blood and gastric analysis are essential to a complete diagnostic work up. The time allowable for medical treatment of a gastric ulcer varies somewhat, but usually a decision can be made within four to six weeks.

If healing of a gastric ulcer does not take place promptly with intensive medical management, surgical intervention is indicated. The operation of choice is gastric resection, including resection of the ulcer. Vagotomy may be carried out concurrently. Both the modifications of the Billroth I and Billroth II operations have been employed.

Surgical treatment. Surgical treatment is necessary for between 10 and 15 per cent of patients with chronic or recurrent peptic ulcers. Indications for surgical treatment are perforation, obstruction, hemorrhage, failure of medical management or intractability, suspicion of carcinoma and complications secondary to previous surgery.

PERFORATION. Surgical closure of a perforated ulcer is indicated as soon as the diagnosis is established. Simple closure is usually employed, although on rare occasions more definitive gastric surgery may be necessary.

OBSTRUCTION. Surgical treatment is necessary if the obstruction is persistent and does not respond to medical treatment. The operations employed are gastric resection with or without vagotomy and vagotomy with gastroenterostomy.

HEMORRHAGE. As the majority of patients with hemorrhage respond to medical management, surgery is necessary only for those patients whose bleeding is prolonged or recurrent. Surgery should be considered if the patient has had more than one massive bleeding episode. Continuous hemorrhage in patients over fifty five years of age usually requires surgical treatment. The operations of choice are subtotal gastric resection or partial resection with vagotomy.

INTRACTABILITY. Patients who have continued distress and disability despite ado-

treatment. They compared the results of medical and surgical treatment in 498 patients with chronic gastric ulcer. One half were treated medically, the remaining being treated surgically. Satisfactory results were obtained in 80 per cent of the surgically treated patients, moderately successful results in 10 per cent and poor results in the remaining 10 per cent. Three-quarters of the patients in the medically treated series developed a recurrence during the period of study. On the basis of Swynnerton and Tanner's study, earlier surgery might be advised for the gastric ulcer patient since a recurrent ulcer constitutes a strong indication for immediate surgery.

The inciting causes for recurrence are physical and mental fatigue, emotional disturbances, dietary indiscretions and infections. Specific measures have been utilized in an attempt to prevent recurrences. These include the use of enterogastrene and related substances, the use of radiation and the long-term administration of anticholinergic drugs. There is some suggestion that radiation might be of some value, but neither enterogastrene nor the anticholinergic drugs have been effective in preventing recurrences.

It is apparent that the major problem in peptic ulcer is the management of recurrences. The education of the patient in the nature and course of the disease is of great value. The resolution of occupational problems and the demonstration of the relationship between ulcer distress and the patient's personality are both valuable in preventing recurrences. The maintenance of dietary restrictions, the avoidance of food and drugs which stimulate gastric secretion and the prompt treatment of infections would all appear to be equally important. It is also thought that the prompt treatment of a recurrence is of value in minimizing the length of the recurrence and preventing the subsequent development of complications.

However, approximately 90 per cent of ulcer patients can be managed satisfactorily medically. Of the remaining 10 per cent who require surgery, approximately 90 per cent of these achieve satisfactory results. It can be seen, therefore, that with proper medical and surgical cooperation, satisfactory results can be achieved in the vast majority of patients with chronic peptic ulcer.

BENIGN GASTRIC LESIONS

Foreign Bodies in the Stomach. Almost any small foreign body may be accidentally

ingested and lodge in the stomach. Two types of objects may be found within the stomach. The first of these—individual objects—commonly includes coins, marbles, pins, needles and other small objects. The second class—the bezoars—consists of a conglomeration of indigestible swallowed material. The most common of these is a trichobezoar or hairball which consists of matted swallowed hair. Another common type is a phytobezoar. Phytobezoars are the result of accumulation of vegetable fibers and most commonly follow the ingestion of persimmon seeds.

Most foreign bodies in the stomach are the result of accidental ingestion. However, there are circumstances in which foreign bodies are ingested deliberately. This is particularly true in insane or psychopathic people who may swallow any variety of small object. There is also that small group of people who earn their living as side show freaks by swallowing broken glass, razor blades and the like.

Symptoms. Foreign bodies in the stomach seldom cause symptoms unless complications arise. Complications include obstruction, ulceration, perforation and hemorrhage. Gastric ulcers are not uncommonly seen in association with bezoars. If the object becomes large enough, obstructive symptoms may result.

Diagnosis. Radiopaque objects in the stomach can be demonstrated by x-ray examination. Some nonradiopaque objects, such as bezoars, can be seen because of the characteristic shadow which is present on the x-ray film. Gastroscopy can also be utilized to visualize foreign bodies within the stomach.

Treatment. Foreign bodies can be handled conservatively in most instances, as most of them will pass spontaneously. The addition of mineral oil and bulky well-cooked foods into the diet may be of some aid in the passage of unpointed objects. If the object constitutes a menace to the patient because of the possibility of perforation, laparotomy can be performed and the object removed. Operation is also mandatory if the foreign body is producing obstruction or hemorrhage.

Gastritis. Gastritis is an inflammation of the gastric wall due to a variety of causes. The early descriptions of gastritis were based upon the gastroscopic appearance of the stomach, whereas in more recent years the descriptions have been supplemented by pathological material from the stomach.

tients developing a marginal ulcer may develop a gastroenterocolic fistula. The treatment for gastroenterocolic fistula is division of the fistula, closing the opening into the colon. Resection of the stomal ulcer is then carried out in a manner similar to that employed in patients with marginal ulcers.

The symptoms which follow gastrectomy may be classified as follows: the dumping syndrome; symptoms due to alterations in carbohydrate absorption; postgastrectomy anemia; postoperative weight change and miscellaneous syndromes, including the development of vitamin deficiencies and post-operative gastritis.

The most serious of these is the dumping syndrome. The dumping syndrome may be defined as the occurrence of sweating, warmth, flushing, nausea, palpitation and faintness with or without diarrhea, which develops shortly after ingesting a meal. It is relieved by lying down. The incidence of the dumping syndrome varies from about 5 to 12 per cent after adequate gastric resection. It is thought to be due to rapid, uncontrolled emptying of large amounts of unprepared food into the jejunum. Meals with concentrated carbohydrate are the most likely to provoke an attack. Machella has simulated this syndrome by instilling protein hydrolysate or magnesium sulfate into the jejunum. Increased motor activity and distention of the jejunum have both been implicated as causative factors. The dumping syndrome is also influenced by the size and type of the gastroanastomosis. It is less frequent when the Hofmeister modification of the Billroth II or the Schoemaker modification of the Billroth I operation is used. The symptoms of the syndrome are similar to those produced by anxiety and it appears that the syndrome is most likely to occur in obviously neurotic individuals.

The avoidance of foods with high carbohydrate content, the avoidance of drinking fluids during meals and either eating while lying on the left side or lying down shortly after eating, have all been recommended for treatment. In patients with severe symptoms the Billroth II type of anastomosis is sometimes converted into a Billroth I, utilizing the interposition of a small jejunal loop.

Rapid absorption of glucose from the small bowel may be followed by hypoglycemia two or three hours after eating. The hypoglycemic attack is manifested by hunger, weakness, sweating and tremor, physical exercise usually being the precipitating factor. These symptoms are seldom disabling, but the patient's condition can be improved

by the administration of a high protein diet which minimizes the fluctuations in blood sugar concentration.

Postgastrectomy anemia may result from poor absorption of iron. It can be corrected by the administration of iron even in the presence of achlorhydria. Postgastrectomy patients may have difficulty in gaining weight in the postoperative period. This is more likely to occur with the Billroth II than with the Billroth I operation. Zollinger has suggested that more conservative procedures be employed in patients who are underweight prior to surgery either a 60 per cent Billroth I resection combined with vagotomy or vagotomy and a posterior gastroenterostomy.

Vitamin deficiencies are not uncommon after gastric resection.

Patients who have a high gastrectomy may develop postoperative gastritis as a result of the reflux of alkaline juices into the gastric pouch. The alkaline juices may have to be diverted away from the stomach.

The major symptom following vagotomy combined with an adequate drainage operation is the development of diarrhea. The cause is not known but it is thought that it is probably due to altered motor function of the stomach and upper small bowel. It occurs in about 7 per cent of patients and it can be managed by nonspecific measures used for the treatment of diarrhea.

Course and Prognosis. The clinical course of peptic ulcer differs from that of many other conditions in that it is characterized by long periods of complete or nearly complete freedom from symptoms alternating with shorter periodic recurrences of ulcer distress. Even though the long-term results of treatment are considered favorable by most physicians, the recurrence of ulcer symptoms at some time may be anticipated in the majority of patients who have a chronic peptic ulcer. Between 10 and 36 per cent will have a recurrence within the first six months after a course of medical treatment, while the incidence of recurrences rises at the end of the fifth year to between 46 and 93 per cent.

The results of treatment in gastric ulcer are thought to be somewhat more favorable than in duodenal ulcer. Smith, Boles and Jordan reporting on their experience with 1000 patients with apparently benign gastric ulcer indicated that of 397 patients treated medically 73 per cent had good results, 14 per cent had fair results and 11 per cent had poor results.

However Swynnerton and Tanner were less optimistic about the results of medical

Gastritis often follows operations for gastric or duodenal ulcer. This postoperative gastritis appears to be the result of the reflux of the alkaline intestinal contents into the stomach. It usually subsides without treatment. Postoperative gastritis is not different from other forms of gastritis. It may be acute or chronic and there may not be erosions or superficial ulcerations. Biopsy of the gastric remnant helps very much in determining the degree of gastritis. Sometimes the gastroscope and the biopsy forceps may be introduced through the stoma into the jejunum and a diagnosis of normal jejunal mucosa or jejunitis may thus be established.

Gastric Atrophy Partial or complete gastric atrophy tends to occur with advancing age and, with it, hypoaclidity or anacidity. Gastric atrophy may exist as an independent entity but is also commonly seen in pernicious anemia. The diagnosis of gastric atrophy can be made by gastroscopy when the mucosa appears pale thin and with numerous easily visible blood vessels. Gastroscopic biopsy and gastric atrophy may be hazardous. If the mucosa is exceedingly thin, biopsy is not recommended. However if the diagnosis of gastric atrophy is doubtful on gross gastroscopic inspection gastroscopic biopsy may be performed, this must be done with great care. In this way a firm pathologic diagnosis may be established.

Specific Inflammations of the Stomach.
Gastric syphilis. Syphilis may involve the stomach by the development of a gumma. Depending upon the frequency and appearance, a gumma may stimulate gastric ulcer or carcinoma. Roentgenographic changes have also been described in association with tabetic crises.

The symptoms may suggest those of either peptic ulcer or carcinoma.

Gastric syphilis responds to antisyphilitic therapy.

Tuberculosis of the stomach. Tuberculosis may involve the stomach to produce an ulcerating lesion. This may stimulate benign ulcer carcinoma or syphilis. It is a rare condition. The treatment is surgical with resection of the involved portion of the stomach.

Other inflammatory conditions Hodgkin's disease actinomycosis and other fungus infections may involve the gastric mucosa. They are extremely rare.

Hypertrophic Stenosis of the Pylorus. Hypertrophic stenosis of the pylorus is an obstructive narrowing of the pylorus due to hypertrophy of the pyloric muscle. It is most

commonly seen in infants but is occasionally seen in adults.

Infantile type Pyloric stenosis is recognized in infants usually when they are about two to three weeks of age. It is more common in males than in females. At operation the pylorus is seen to be enlarged and of cartilaginous consistency.

The symptoms consist of projectile vomiting accompanied by weight loss. Occasionally the enlarged stomach can be seen and rarely a tumor representing the pylorus can be palpated.

Conservative treatment should be tried using antispasmodic-sedative mixtures along with thick cereal feedings. If the patient does not respond to this treatment, the performance of a Ramstedt-Fredet pyloroplasty is indicated. This consists of a longitudinal incision through the pyloric muscle down to the mucosa. The results of treatment are gratifying.

Adult type Hypertrophy of the pylorus is occasionally seen in adults. It is also termed "acquired pyloric stenosis" because of its frequent association with gastric diseases such as gastric ulcer or gastritis. Ulcers located within the pylorus are most prone to produce this syndrome.

The primary symptom is vomiting which may or may not be accompanied by pain. Roentgenologically the stomach may appear dilated and shows marked delay in gastric evacuation.

Treatment depends upon the underlying condition. If a gastric ulcer is present, the medical management for gastric ulcer should be followed. If an ulcer is in the region of the pylorus, medical management may not be satisfactory and gastric resection or gastroenterostomy and vagotomy may be necessary.

Prolapse of the Gastric Mucosa Prolapse of the gastric mucosa consists of the prolapse of the mucosa of the distal stomach through the pylorus into the duodenal bulb. There is considerable difference of opinion as to whether this condition produces symptoms and what treatment should be recommended for it. It appears to be more commonly associated with hypertrophic gastritis, particularly of the antral area. Many workers feel that it is a normal physiologic variant, being seen as often in patients without symptoms as in those with symptoms. Conservative treatment is indicated.

Prolapse of the gastric mucosa is ordinarily a medical problem and in only the severe

Many classifications of gastritis have been suggested. From the point of view of the gastroscopist only three groups are important, namely acute gastritis, chronic gastritis and gastric atrophy. Since the advent of gastroscopic biopsy it has become obvious that the pathologist can differentiate only these three. The terms "superficial gastritis" and "hypertrophic" gastritis which have been used so long in gastroscopic interpretation, cannot be definitely correlated with the pathologic findings.

We believe the term "hypertrophic gastritis" is a misnomer and should be abandoned, since the verrucous appearance of the gastric mucosa, which upon gastroscopy appears to be chronically hypertrophic is associated with a normal histologic picture in most cases. This finding indicates that the verrucous nature of the mucosa is probably a normal variant.

Acute gastritis. SIMPLE EXOGENOUS GASTRITIS. Acute gastritis is a common condition usually of brief duration. It develops as a result of ingested foods or other irritants. Gastroscopically it is characterized by the presence of reddening, edema, superficial erosions and adherent slimy white mucus. This type of appearance has been considered diagnostic of "superficial" gastritis which corresponds fairly well to the acute gastritis described by the pathologist. The only way to make a positive diagnosis of acute gastritis is by biopsy. The specimen may be taken at the time of gastroscopy or by means of the blind gastric suction biopsy tube.

The symptoms most commonly associated consist of anorexia, epigastric distress and, occasionally pain. Vomiting may occur. Diarrhea may be present because of associated enteritis.

Treatment consists of brief abstinence from food, followed by a bland, liquid diet. Subsequently soft foods may be added. The course of the condition is generally self-limited.

CORROSIVE GASTRITIS. This form of gastritis most commonly occurs after the ingestion of acids, lyes or other corrosive substances. Corrosion results with inflammation and sloughing of the mucous membrane.

The symptoms of corrosive gastritis depend in part upon the corrosive agent. Epigastric burning and cramplike pain are usually present. Corrosion may occur in the mouth and esophagus with resultant dysphagia.

The initial treatment should be for the

shock which accompanies the gastritis. The stomach may be lavaged cautiously with water or specific antidotes. Treatment is not very satisfactory and stricture in the esophagus or pyloric stenosis may develop.

ACUTE INFECTIOUS GASTRITIS. This form of gastritis develops secondary to the influence of toxins or bacteria within the blood stream. It may be found in association with measles, scarlet fever, pneumonia or other infectious diseases.

It is manifested by nausea, anorexia and, occasionally vomiting.

Food should be withheld until the patient can eat a bland diet. The course of the illness is self-limited.

PHLEGMONOUS GASTRITIS. Phlegmonous gastritis is characterized by inflammation of the gastric mucosa caused by streptococci, staphylococci or colon bacilli. Abscesses may be found within the gastric walls.

The symptoms are those of an acute infectious abdominal disease. Chills, abdominal pain and fever may all be observed.

Antibiotics should be used. If a localized abscess is present, it may have to be drained.

Chronic gastritis. This is a fairly common disease, the diagnosis of which has been made very loosely clinically, radiologically and even gastroscopically. A clinical diagnosis of gastritis has frequently been used as a sort of diagnostic wastebasket and, taken by itself has little to substantiate it. A radiologic diagnosis of chronic gastritis based on thick folds and increased secretion of mucus is often of definite significance but should be confirmed by gastroscopy with biopsy. When the radiologist reports thick folds and suggests a diagnosis of hypertrophic gastritis, the question may arise as to the presence of lymphoma or even diffuse infiltrating carcinoma. In such instances, gastroscopic biopsy is exceedingly important.

Changes in the stomach may be found in association with other conditions. Inflammatory change may occur in association with primary gastric or duodenal disease and may render the differential diagnosis difficult. Gastritis may be associated with gastric carcinoma. It has been indicated that gastritis may be a precursor of carcinoma.

Gastritis may accompany either gastric or duodenal ulcer. It is more common with ulcers located in the stomach. Antral gastritis is extremely common, with ulcers located below the incisura angularis. Sometimes, on gastroscopic examination, one can visualize gastritis in the stomach in patients with a duodenal ulcer.

The Surgery of Peptic Ulcer

By JOEL W BAKER M D

JOEL W BAKER is a Virginian who received his education at The University as he and his fellow statesmen would say. He received his later surgical training in Washington where he is now chief of the surgical staff at the Virginia Mason Hospital and Chairman of The Mason Clinic in Seattle. He is an outstanding exponent of the teaching principle that surgery is the art of applying knowledge in the basic sciences to the patient.

INTRODUCTION

The high incidence of peptic ulcer in the general populace may explain the voluminous literature which has been devoted to considerations of the etiology and treatment of this disease. The multiple surgical procedures that have been sponsored for the control of ulcer attest the failure of present methods to supply an ideal solution. There is more recorded experimental and clinical research on the problem of peptic ulcer than on any other surgical subject.

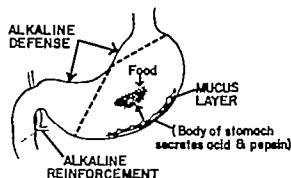
An attempt has been made to review the principal considerations which mold the judgment of the surgeon as he is confronted at the bedside by the varied problems of this disease. While an effort is made to review only the more established experimental work and but a few of the more pertinent clinical reports, nevertheless if this policy were followed too rigidly the challenge conveyed by some of the more recent and unconfirmed studies would then be lost and the fact might be overlooked that the final chapter in this problem has yet to be written. The surgeon cannot encounter this disease with complacency. The most popular procedures admittedly leave something to be desired both from the standpoint of untoward effects and sustained control of the ulcer diathesis. Nevertheless for the surgeon individually to substitute procedures less established and less familiar in his hands before they can be carefully evaluated in qualified centers, might indeed point to immature surgical judgment. Profound concepts must be based upon a thorough knowledge of the known factors in etiology, the supporting evidence from the experimental laboratory and, last

but not least, a conscientious follow up of one's own experience.

Be not the first by whom the new are tried,
Nor yet the last to lay the old aside—Alexander Pope

PHYSIOLOGIC CONSIDERATIONS

A concept of the normal gastric physiology is a prerequisite to a study of the etiology of peptic ulcer. The stomach's primary function is the acid peptic digestion of food and for this purpose the fundus and body (Fig. 39) are lined with parietal and chief cells secreting hydrochloric acid and pepsin.



LOCAL DEFENSE MECHANISM

1. Alkaline secretion neutralizes acid
2. Mucus layer protects surface
3. Food buffers the acid

Figure 39 The body and fundus of the stomach are lined by parietal and chief cells secreting hydrochloric acid and pepsin. A narrow rim of cardiac glands above and a 3-inch sleeve of antral pyloric glands below this acid-secreting area secrete a highly alkaline neutralizing and mucinous fluid, presumably as a protection to the very vulnerable esophagus on the one side and to the intestine on the other. This alkaline protection is reinforced in the second portion of the duodenum by the alkaline biliary and pancreatic secretions.

intractable cases and those with complications is surgery indicated. Medical treatment includes a bland diet and particularly antispasmodics. If obstruction, hematemesis severe pain or complications exist operation may be indicated.

READING REFERENCES

- Barborka, C. J. and Teater E. C., Jr. *Peptic Ulcers—Diagnosis and Treatment*. Boston, Little Brown & Company 1935.
- Barborka, C. J. and others. *Modern Concepts of Peptic Ulcer*. Quart. Bull. Northwestern Univ. M. School 28:223, 1934.
- Bartels, E. D., and Ekorn, H. Prolapse of the Gastric Mucosa through the Pylorus. *Physiological or Abnormal?* *Gastroenterology* 20:100, 1932.
- Browne, D. C., Mitchell, R. E., Jr. McHardy, G. and Welch, C. E. Evaluation of Surgical Intervention in Gastric Ulcer. *J.A.M.A.* 153:807, 1954.
- Cushing, H. *Peptic Ulcers and the Intestine*. Surg. Gynec. & Obst. 5:1, 1932.
- Dragstedt, L. R., Oberhelman, H. A. Jr. Evans, S. O., and Higler, S. P. Antrum Hyperfunction and Gastric Ulcer. *Ann. Surg.* 140:396, 1954.
- Dragstedt, L. R., and others. *Quantitative Studies on the Mechanism of Gastric Secretion in Health and Disease*. *Ann. Surg.* 132:628, 1950.
- Edkins, J. S. *The Chemical Mechanism of Gastric Secretion*. *J. Physiol.* 34:133, 1906.
- Garrido Klinge and Luis Pena. *The Gastrocholedal Ulcer in High Altitudes (Peruvian Andes)*. *World Congress of Gastroenterology*. Baltimore, Williams & Wilkins Company 1959 vol. 1, pp. 68-78.
- Greengard, H., Atkinson, A. J. Crossman, M. I., and Ivy, A. C. The Effectiveness of Parenterally Administered "Entero-gastrone" in the Prophylaxis of Recurrences of Experimental and Clinical Peptic Ulcer with a Summary of 59 Cases. *Gastroenterology* 7:625, 1946.
- Harkins, H. N. and Hooker, D. H. Vagotomy for Peptic Ulcer Surgery 22:239, 1947.
- Illingworth, C. F. W. *Peptic Ulcer*. Edinburgh, E. & S. Livingstone, 1939.
- Kramer, J. B. and Palmer, W. L. *The Problem of Peptic Ulcer*. *Ann. J. Med.* 13:615, 1952.
- Oberhelman, H. A., Jr., Woodward, E. R., Zubins, J. M. and Dragstedt, L. R. *Physiology of the Gastric Antrum*. *Ann. J. Physiol.* 169:738, 1952.
- Pavlov, I. P. *The Work of the Digestive Glands*. London, Griffin, 1910.
- Porter, R. W., Morus, H. J., and French, J. D. Hypothalamic Influences on Hydrochloric Acid Secretion of the Stomach. *Surgery* 33:875, 1953.
- Ruffin, J. M., Baylin, G. J., Legerton, C. W. Jr., and Teater, E. C., Jr. *Mechanism of Pain in Peptic Ulcer*. *Gastroenterology* 33:232, 1953.
- Sandweiss, D. J. *Peptic Ulcer*. Philadelphia, W. B. Saunders Company 1951.
- Sauvage, L. R., and others. *The Relation between the Physiologic Stimulatory Mechanisms of Gastric Secretion and the Incidence of Peptic Ulceration. An Experimental Study Employing a New Preparation*. *Surg. Gynec. & Obst.* 98:127, 1953.
- Schmidler, R. *Gastritis*. New York, Grune & Stratton, 1947.
- Smith, F. H., Boles, R. S. Jr. and Jordan, S. M. *Problems of the Gastric Ulcer Reviewed*. *J.A.M.A.* 150:1505, 1953.
- Storer, E. H. and others. *Gastric Secretion in Heidenham Pouches Following Section of Vagus Nerves to the Main Stomach*. *Proc. Soc. Exper. Biol. & Med.* 8:325, 1952.
- Swynnerton, B. F. and Tanner, W. C. *Chronic Gastric Ulcer*. *Brit. M. J.* 2:841, 1953.
- Teater, E. C. Jr., and Barborka, C. J. *Effect of Carbonic Anhydrase Inhibitor Acetazolamide, upon Gastric Secretion in Man, Report of Its Use Separately and in Conjunction with Anticholinergic Drugs*. *Gastroenterology* 28:519, 1955.
- Teater, E. C., Jr. and others. *Coexistent Carcinoma of the Stomach and Hypertrophic Gastritis; Report of a Case with Review of the Literature*. *Gastroenterology* 24:579, 1953.
- Villarreal, R., Ganong, W. F. and Gray, S. J. *Effect of Adrenocorticotrophic Hormone upon the Gastric Secretion of Hydrochloric Acid, Pepsin and Electrolytes in the Dog*. *Am. J. Physiol.* 183:485, 1953.

ulation are surgically controllable the cephalic phase through vagotomy and the gastric phase by antrectomy (i.e., resection of the distal one-third of the stomach). By varying the level of distal gastrectomy different proportions of the acid-secreting target may be removed in addition to the hormone-secreting antrum. The quantitative relationship between the two phases of stimulation is interesting to contemplate and of practical importance in applying surgery to control ulcer. Unfortunately the relative importance of the two is not convincingly established. Dragstedt attributes duodenal ulcer to a deranged neurogenic phase and, by contrast, attributes gastric ulcer to the hormonal phase. He and his coworkers believe duodenal ulcer should be best controlled by an attack on the neurogenic phase of stimulation (i.e., vagotomy) and that gastric ulcer should be more directly controlled by ablation of the gastric (hormone) phase (i.e., antrectomy). This approach to duodenal ulcer however cannot be reconciled entirely with some of the experimental work of Storer Harkins and their associates. They have performed experiments on dogs with Heidenhain pouches to show that if the neurogenic avenue of stimulation is removed by vagotomy of the main stomach, there is an increase in secretion from the pouch, presumably due to a compensatory increase in the hormonal phase (Fig. 40). It is also

worthy of note that they found the gastric secretion to increase after gastroenterostomy. Evans Dragstedt and their associates have since confirmed that there is increased gastric secretion of acid after vagotomy "when not accompanied by gastroenterostomy." It should also be mentioned that Lillehei, Lewis and Wangenstein have shown that where vagotomy is combined with gastroenterostomy the quicker release of food from the stomach reduces the protective buffering effect of food during the delayed gastric phase of digestion, a phase that has been heightened, as just mentioned, by vagotomy and also by gastroenterostomy. In the Sauvage pouch preparation, antrectomy was a more potent factor than vagotomy in reducing the incidence of marginal ulcer in the connecting jejunal loop.

In summary, then the fundus and body of the stomach may be considered the secreting target and this target is normally stimulated to secretion at mealtime through neurogenic vagal impulses (cephalic phase) by a hormone secreted in the antrum (gastric phase) and by a hormone from the intestine (intestinal phase).

Experimentally Produced Ulcer The experimental production of ulcer is pertinent to its etiology. However the basic differences between the experimental animal, usually the dog, and man must be kept constantly in mind. In essence, all experimental

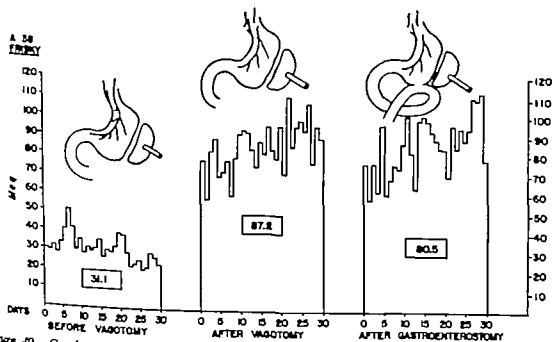


Figure 40. Graphs representation of free hydrochloric acid secretion (expressed in milliequivalents) from Heidenhain pouch of dog A-38 during control period (left) and collection periods after transthoracic section of vagus nerves to the main stomach (center) and subsequent gastrojejunostomy (right). (Schmitz, E. J., et al.: The Effect of Vagotomy of the Main Stomach on Heidenhain Pouch Secretion. *Surgical Forum*, 1963.)

These cells, either from an inherent immunity or otherwise, appear to be partially resistant to the digestive action of their own secretion, but the remainder of the gastrointestinal mucosa is more susceptible. As a possible protection from this acid, therefore there is provided a narrow rim of cardiac glands above and a three-inch sleeve of antropyloric glands below this acid secreting area. These two barrier zones secrete a highly alkaline neutralizing and mucinous fluid, presumably as a protection to the very vulnerable esophagus on the one side and to the intestine on the other. In addition, the cardiac sphincter and the angle of esophageal juncture with the stomach mechanically protect the esophagus from the reflux of digestive juices. The distal protection is reinforced in the second portion of the duodenum by the daily flow of approximately 700 cc. each of alkaline bile and pancreatic juices. Rarely does ulcer occur below this level of reinforcement.

The chemical neutralization is not the only local defense mechanism. A secreted layer of mucus mechanically protects the surface and, possibly most important of all, the food mgested at the height of acid secretion absorbs and buffers this acid. In the proximal duodenum a special layer of cells referred to as Brunner's glands secretes mucus and adds, therefore to this protective surface coating. They are strategically located at the point toward which the jet stream of acid is directed. Since so much experimental work in ulcer has been done in dogs, it should be remarked that the occurrence of these protective glands is significantly less in the dog's duodenum than in the human.

To provide sufficient acid for digestion of the meal experiments suggest there are three phases of gastric secretion, the cephalic, the gastric and the intestinal phase. Neurogenic impulses by way of the vagi, elicited by the sight or odor or taste of food, bring about the immediate hypersecretion of acid, the so-called *cephalic* or *neurogenic phase* of digestion. This conditioned reflex was described by Pavlov in 1900. The observations of Beaumont on his patient, Alex St. Martin, and the more recent observations of Wolf and Wolff in patients with gastroscopic windows demonstrated the profound influence on the appearance and function of the gastric mucosa effected by the changing emotional states mediated through the vagal nerves.

The second phase is induced by a hormone secreted by the gastric antrum and

called gastrin by Edkins in 1905. Gastrin is a histamine-like substance which reaches the corpus and fundus by way of the blood stream and in this delayed manner stimulates the target parietal cells to secretion, the so-called *gastric phase* of digestion. It is noteworthy that this phase is delayed, as contrasted with the immediate cephalic phase. Secretion of the hormone gastrin by the antrum is stimulated by local contact with food or by mechanical distention of the antrum. This is presumably through stimulation of the submucosal sensory receptors since the application of cocaine to the antral mucosa prevents the release of gastrin even in the presence of food or distention. Dragstedt's studies have shown that hypersecretion of gastric juice may be induced through the endocrine influence of the antrum even after the antrum has been transplanted into the colon as a diverticulum. This secretion is abolished if the colon containing the antral diverticulum is excluded from gastrointestinal continuity and washed free of fecal material or food. Obstruction of this isolated segment on the other hand, produced vigorous secretion, apparently by virtue of the peristaltic pressure alone.

There is a third phase of peptic digestion, referred to as the *intestinal phase* which is stimulated by a hormone similar to but less powerful than gastrin. This hormone is assumed to be secreted by the mucous membrane of the small intestine when in contact with food. The character of this hormone has not been well defined. The subtle balance of physiologic checks and counterchecks effective in the control of secretion is suggested again by recent experimental work which shows the antrum to exert an inhibitory as well as a stimulating effect on acid secretion—a factor which may prove of surgical significance. This inhibitory effect is initiated by local contact of acid on the antrum. Whether this inhibitory influence of the antrum is mediated through a local effect which simply interferes with the secretion of gastrin or whether it is accomplished by the liberation from the antrum of a gastric inhibitory hormone is being currently explored. Likewise, the duodenum upon contact with hyperacid chyme may exert an inhibitory role on acid secretion.

These brief physiologic facts form the basis of both the medical and surgical treatment of peptic ulcer. They have been supported in the experimental laboratory. Both the cephalic and the gastric phases of stim-

ected possibly in a lowered local resistance on the part of the duodenal mucosa. This local susceptibility to erosion could be due to a variety of factors including local vascular occlusive disease or vascular spasm with local ischemia, portal venous hypertension or to a number of as yet unexplained factors. This factor of local susceptibility must be added in order to explain the absence of ulcer in some patients with extremely high gastric acid secretion. In fact some of the highest acid values have been found in the so-called neurasthenic patient without any objective evidence or history of ulcer. Finally for years the emphasis has been placed on the local imbalance of acid secretion and its neutralization rather than on constitutional factors. However the exacerbation of ulcer with personal stresses and the increased incidence of ulcer in periods of general emotional stress as in times of economic depression and war and the gradually increased incidence with the increased tempo of the atomic era—all these facts, combined with the emphasis in the last decade on the vagal influence—are making clinicians more conscious of the constitutional and psychogenic disturbances leading to this disease. The point to be emphasized is that the surgical attack remains a *local* approach for a constitutional disease and therefore, leaves much to be desired.

ACUTE ULCERATION

Before the indications for surgery and the choice of surgical procedures for the control of chronic ulcer are considered, a warning should be sounded concerning *acute erosions and their complications and surgical implications* should be briefly reviewed.

Surgeons, pathologists and those interested in gastroscopy occasionally see other than the characteristically punched-out, indurated, chronic ulcer. They may see albeit more rarely the small superficial erosions, varying in size, but usually small, and often multiple. These transient ulcers may bleed so massively as to require emergency resection or they may heal and not be demonstrated after bleeding ceases. It is known that similar acute ulcerations may also perforate. The acute ulceration seen in association with burn (Curling) or with stress may be but more advanced examples of erosions that may transiently occur and create such emergency problems. Because such erosions do bleed and can, therefore, confuse the picture, there is reason, in treating such

erosions at least to bend every effort to control upper gastrointestinal hemorrhage by adequate or continued blood transfusions before resorting to surgery—particularly if chronic ulcer has not or cannot be documented. Gastric resection for the acute erosion instead of correcting a long standing physiologic imbalance (as in chronic ulcer) sacrifices an important organ for a transient blemish—although granted this expediency is occasionally necessary in the last resort to save life. Massive hemorrhage from acute gastroduodenal ulceration without previous ulcer history has been reported in the immediate postoperative period of patients undergoing unrelated surgery. While these are examples of uncontrolled hemorrhage from acute possibly "stress" ulcer they must represent the exception rather than the rule. In general if one treats the superficially bleeding erosions early in the hospital, and gives continuous blood transfusions they will usually stop bleeding—more often than will the penetrating calloused ulcer. The fact that acute erosions which may be quite superficial may also be associated with chronic ulcer must be borne in mind at emergency operation where the discovery of one bleeding point does not exclude others.

Likewise, in operations for acute perforations of the duodenum, simple closure is in order if the chronic ulcer diathesis cannot be documented. Such perforations may represent only an acute ulcer. The stomach should not be sacrificed for the transient lesion since the etiology of these acute erosions is not understood. Without a history of preceding chronicity it cannot be assumed that the ulcer will recur. Only for the perforation with calloused margin and a well-documented chronic history can primary gastric resection at the time of duodenal perforation be justified in the minds of those who approach the problem from a studied experience. The same is not true in gastric ulcer where primary resection of the perforated gastric ulcer is desirable, if not contraindicated by peritonitis. This differential is based upon the hazard of malignancy in the gastric ulcer and the greater difficulty in satisfactorily closing the perforation in the thicker gastric wall. For the calloused duodenal ulcer with a long documented history of chronic and recurring symptoms, primary resection at the time of perforation is thoroughly justified, provided, of course, generalized peritonitis does not make resection a prohibitive risk. Thorough peritoneal lav

ulcer has been produced by altering the normal balance between acid secretion on the one hand and the defense mechanism on the other.

Mann and Williamson were able to produce ulcer consistently by deflecting the protective alkaline secretions of the duodenum, pancreas and liver into the terminal ileum, leaving thereby the jejunum anastomosed to the stomach and exposed without protection to the action of the gastric secretions (Fig. 41). The possible additional factor in this experiment of removing the inhibitory influence on acid secretion, which was shown by Sokolov in 1904 to reside in the duodenum, has been recalled recently by Brackney, Thal and Wangenstein. It is noteworthy that the marginal ulceration in the Mann-Williamson dog is reduced by vagotomy although total protection against ulcer is not afforded. Further proof of the protective importance of the pancreatic secretions is the work of Elman and Hartman who found that external deflection of the pancreatic secretions produced peptic ulcer in 100 per cent of dogs. A seeming paradox to this, which present knowledge does not explain, is that Dragstedt found that in 300 animals maintained with suitable insulin replacement after total pancreatectomy (as contrasted with deflection of the pancreatic secretions) the incidence of ulcer was only 1.3 per cent. The influence of the rare alpha cell tumor of the pancreas on ulcer formation may be in some way related to these observations.

The second method of producing experimental ulcer was reported in 1940 by Code and Varco. Whereas heretofore experimental ulcer had been produced by deflecting the protective secretions, they produced experimental ulcer for the first time as a result of prolonged hypersecretion of acid. Where separately injected doses of histamine had failed to produce ulcer the subcutaneous implantation of beeswax saturated with his-

Paraphrasing and aside from experimentally produced ulcer the protective importance of the alkaline biliary and pancreatic secretions is demonstrated clinically by the occasional chronic duodenal ulcer seen associated with carcinoma of the pancreas which has obstructed the biliary and pancreatic ducts and prevented these protective secretions from reaching the duodenum. I treated a patient who died from a massively bleeding marginal ulcer three years after a Whipple resection for carcinoma of the pancreas presumably the result of erosion by unneutralized acid. The latter case illustrates the importance of considering a higher gastric secretion in the Whipple operation done for cancer if the preoperative gastric acid secretion is in the higher range.

EXPERIMENTALLY PRODUCED ULCER Deflection of alkaline defenses (Mann-Williamson, dog 1923)

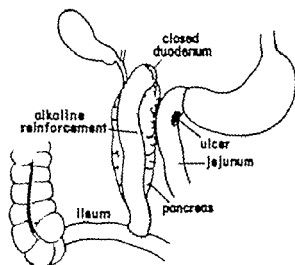


Fig. 41

tamine produced a sustained stimulation which resulted in ulcer. This experimental ulcer is not prevented by vagotomy because it is produced by stimulating the gastric phase of acid secretion rather than the cephalic phase.

Several other methods of producing ulcer have profound clinical significance. The ulcerating effect of retained unbuffered acid was demonstrated by Shay and his group. In fasting rats they ligated the pylorus, and multiple ulcers in the obstructed stomach consistently resulted. In a subsequent experiment, Harkins and Hooker showed that these rats are protected by vagotomy. The important buffering effect of food in the defense mechanism is demonstrated in the Fremont dog. The stomach is taken out of continuity but the blood and nerve supply are preserved. In the absence of food in this isolated stomach, these dogs consistently develop ulcers but are protected by vagotomy as shown by Dragstedt.

ETIOLOGY OF PEPTIC ULCER

The work cited is but a fraction of the experimental workers' contributions to the understanding of the pathophysiology associated with chronic peptic ulcer. Nevertheless at present, we must admit that the exact etiology of chronic benign ulcer is unknown, although obviously two factors are quite generally accepted. These factors are (1) the hypersecretion of acid and pepsin not alone in the normal manner at mealtime, but particularly the sustained secretion of this acid and enzyme in the interim between feedings and (2) a constitutional factor re-

fected possibly in a lowered local resistance on the part of the duodenal mucosa. This local susceptibility to erosion could be due to a variety of factors including local vascular occlusive disease or vascular spasm with local ischemia, portal venous hypertension or to a number of as yet unexplained factors. This factor of local susceptibility must be added in order to explain the absence of ulcer in some patients with extremely high gastric acid secretion. In fact some of the highest acid values have been found in the so-called neurasthenic patient without any objective evidence or history of ulcer. Finally, for years the emphasis has been placed on the local imbalance of acid secretion and its neutralization rather than on constitutional factors. However the exacerbation of ulcer with personal stresses and the increased incidence of ulcer in periods of general emotional stress as in times of economic depression and war and the gradually increased incidence with the increased tempo of the atomic era—all these facts, combined with the emphasis in the last decade on the vagal influence—are making clinicians more conscious of the constitutional and psychogenic disturbances leading to this disease. The point to be emphasized is that the surgical attack remains a local approach for a constitutional disease and, therefore, leaves much to be desired.

ACUTE ULCERATION

Before the indications for surgery and the choice of surgical procedures for the control of chronic ulcer are considered, a warning should be sounded concerning acute erosions and their complications and surgical implications should be briefly reviewed.

Surgeons, pathologists and those interested in gastroscopy occasionally see other than the characteristically punched-out, indurated, chronic ulcer. They may see, albeit more rarely the small superficial erosions varying in size, but usually small and often multiple. These transient ulcers may bleed so massively as to require emergency resection or they may heal and not be demonstrated after bleeding ceases. It is known that similar acute ulcerations may also perforate. The acute ulceration seen in association with burn (Curling) or with stress may be but more advanced examples of erosions that may transiently occur and create such emergency problems. Because such erosions do bleed and can, therefore, confuse the picture, there is reason, in treating such

erosions at least to bend every effort to control upper gastrointestinal hemorrhage by adequate or continued blood transfusions before resorting to surgery—particularly if chronic ulcer has not or cannot be documented. Gastric resection for the acute erosion instead of correcting a long standing physiologic imbalance (as in chronic ulcer) sacrifices an important organ for a transient blemish—although granted this expediency is occasionally necessary in the last resort to save life. Massive hemorrhage from acute gastroduodenal ulceration without previous ulcer history has been reported in the immediate postoperative period of patients undergoing unrelated surgery. While these are examples of uncontrolled hemorrhage from acute possibly stress ulcer they must represent the exception rather than the rule. In general if one treats the superficially bleeding erosions early in the hospital, and gives continuous blood transfusions they will usually stop bleeding—more often than will the penetrating calloused ulcer. The fact that acute erosions which may be quite superficial may also be associated with chronic ulcer must be borne in mind at emergency operation where the discovery of one bleeding point does not exclude others.

Likewise in operations for acute perforations of the duodenum, simple closure is in order if the chronic ulcer diathesis cannot be documented. Such perforations may represent only an acute ulcer. The stomach should not be sacrificed for the transient lesion since the etiology of these acute erosions is not understood. Without a history of preceding chronicity it cannot be assumed that the ulcer will recur. Only for the perforation with calloused margin and a well-documented chronic history can primary gastric resection at the time of duodenal perforation be justified in the minds of those who approach the problem from a studied experience. The same is not true in gastric ulcer where primary resection of the perforated gastric ulcer is desirable, if not contraindicated by peritonitis. This differential is based upon the hazard of malignancy in the gastric ulcer and the greater difficulty in satisfactorily closing the perforation in the thicker gastric wall. For the calloused duodenal ulcer with a long documented history of chronic and recurring symptoms, primary resection at the time of perforation is thoroughly justified, provided, of course, generalized peritonitis does not make resection a prohibitive risk. Thorough peritoneal lav

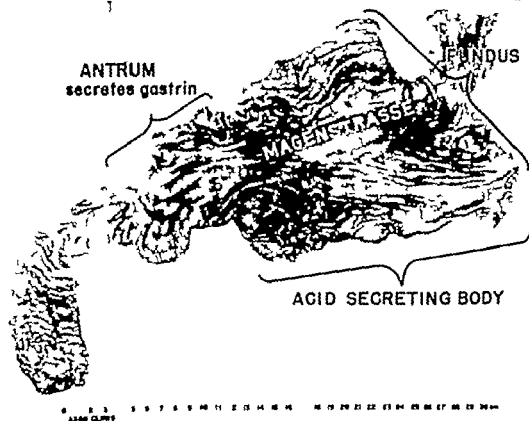


Figure 42. Photograph of a normal stomach opened longitudinally showing the terminal end of the esophagus and the first and second portions of the duodenum.

age with normal saline to prevent pocketing of foreign material is an important part of the operation for perforated viscus.

When primary resection is done either for perforation or hemorrhage in the patient with acute ulcer the long-term result will not be gratifying and the "dividend" to the patient not nearly so apparent as in the patient who has suffered long from chronic ulcer. Should he develop any of the untoward sequelae of resection, they would prove particularly objectionable to him. It should also be pointed out that vagotomy has no established place in the control of these acute stress ulcers. Zubiran, Dragstedt and associates, for example have shown that cortisone stimulation of gastric secretion occurs even after antrectomy and vagotomy. In dealing with the acute ulcer one should look for the recent administration of corticoids and all factors conducive to stress. A relationship between acute erosion and chronic peptic ulcer has not been established. It is possible that acute ulcer may be the forerunner of chronic ulcer—at least in the vulnerable patient with hypersecretion of acid. It is known that chronic ulcers are generally limited in their occurrence to certain locations, that is, the first portion of the

duodenum, the antrum and the lesser curvature of the stomach and the lower esophagus—anatomic areas in which the mucosa is smooth and rather tightly adherent to the muscularis in contradistinction to the fundus of the stomach where the mucosa hangs in folds and is loosely adherent to the underlying musculature (Fig. 42). Ogilvie calls attention to this fact and suggests that if abrasions occur as a result of trauma, infections, dietetic indiscretions and the like in the fundus, where the mucous membrane is loose the edges of the abrasion fall together as the stomach empties and it heals as rapidly as would a small burn on the eyelid, but if it occurs where the mucous membrane is fixed, the defect remains open as would a small burn on the bridge of the nose and acid digestion occurs before the abrasion can heal. In these, then, a chronic ulcer might result. The difference in significance in the acute and chronic ulcer is portrayed by Ogilvie when he says "If we were able to inspect the lining of our whole alimentary canal with the constant scrutiny and minute attention that a girl devotes to studying her complexion, we should probably see that our inner surface like our outer one is constantly showing small blemishes."

sions are multiple and may occur anywhere and as a rule are of no significance unless because of their location combined with the patient's vulnerability they become chronic or unless they massively bleed or perforate. The fact that on x ray examination duodenal ulcer is found approximately nine times as commonly as gastric ulcer yet in autopsy subjects the incidence of ulceration in the duodenum and stomach is identical, lends weight to this philosophical hypothesis.

TREATMENT OF CHRONIC DUODENAL ULCER

The pathogenesis and, therefore, the etiology of chronic ulcer are better understood than those of acute ulcer. Suffice it to say that when the ulcer is situated in its more common location, that is, the first portion of the duodenum, a medical regimen should prove adequate in at least 80 per cent of cases. In most clinics, surgery is advised only because of the complications of ulcer or for those which prove chronically intractable to adequate medical treatment. Such treatment should encompass the principles of acid control, anticholinergic drugs, spacing buffering food in the stomach throughout the twenty four hours, abstinence from the temperate use of stimulants (nicotine, alcohol and coffee) combined with sedatives, mental and physical rest and recreation. A mistake that may be made in the medical management is to allow susceptible patients to become markedly overweight by the required frequent feedings. This is seen occasionally in the intractable case where the patient comes late to surgery. The weight factor then adds to the risk as well as to the mechanical problems of surgery.

SELECTION OF THE PATIENT FOR SURGERY

From 30 to 75 per cent of gastric ulcers come to surgery because of the high recurrence rate when treated medically and because of the generally good results from surgical therapy and particularly because resection is the only reliable way to exclude malignancy. By contrast, the ulcer in the duodenum, being unrelated to malignancy, requires surgical assistance in its management in percentages varying from only 10 to 30 per cent, the figures quoted from most medical centers. At The Mason Clinic, surgery has been applied in percentages varying from 10 to 15 per cent. In the last three years it has averaged 14 per cent. In other words, the duodenal ulcer is primarily a

medical and not a surgical disease. Elective surgery should be considered for the patient who has (1) intractable symptoms unrelieved by an adequate medical regimen (2) unrelieved or recurring obstruction and (3) repeated hemorrhage.

Emergency surgery is required for acute perforation or uncontrolled hemorrhage. While surgery for intractability and obstruction is more often required in the fifth and sixth decades of life it has been observed that the ulcer which first becomes manifest in the older patient and that which arises very early in life as in the second decade represent the more severe diathesis and are most prone to prove intractable. The old dictum of applying simple gastroenterostomy in the older patient with "burned-out ulcer scar" is no longer tenable. Gastroenterostomy per se is no longer considered adequate treatment for ulcer certainly not for the limited number which justify surgical interference.

In selecting patients for surgery, the surgeon should deny acceptance for surgery as long as possible the asthenic migrainous patient who is a finicky eater and who is underweight. This introvert finally exhausts his physician's patience and surgical assistance is sought. With any treatment there will be a high percentage of unsatisfactory results in these patients and an increased percentage of the dumping syndrome and other untoward sequelae after surgery. Such patients should not have surgery of any kind without prolonged observation on the part of the surgeon as well as the internist. To accept properly his responsibility the surgeon must know firsthand the patient's history, habits in work and recreation, his worries, his reactions to possible marital or business problems, the inciting factors in the exacerbations of his symptoms and his cooperation or lack of it in following his physician's instructions. Before recommending surgery he will be wise to discuss with the patient the possible untoward sequelae of surgery in a manner that does not necessarily plant the seed for introspective complaints but rather provides a protective understanding should they occur. Such sequelae will then be accepted not as a result of a mistake on the part of the surgeon, but rather as an irreducible premium for control of the more crippling ulcer symptoms. By the same token, the internist or the patient's general physician should not be guilty of indifferent procrastination in advising surgery in the severe forms of the disease. Pos-

sibly the internist, in defining intractability should place less reliance on the response of the patient to in hospital treatment and more significance on the early relapse after hospital discharge when the patient resumes his occupation in the habitual environment in which he must continue to work. Unjustified and prolonged delay in advising surgery not only taxes the patient unfairly but also favors penetration of the ulcer advanced fibrosis and a more extensive inflammatory reaction, factors which make more difficult the anastomosis or closure of the duodenal stump and thereby increase the technical hazards of surgery.

SELECTION OF THE SURGICAL PROCEDURE

When the refractory course or the complications of duodenal ulcer clearly justify surgery the choice from a number of advocated surgical procedures places on trial the surgeon's judgment and his technical skill. As in medical management, all methods of surgical attack are directed at the reduction or subtotal elimination of gastric acid secretion, without which peptic erosion does not occur. This can be accomplished in varying degree by one or a combination of methods of altering gastric physiology (Fig. 43).

Basically these methods are (1) vagotomy thereby removing the cephalic phase of stimulation (2) resection of the antrum thereby removing the source of gastrin which is responsible for stimulating the delayed gastric phase of acid secretion, and which by experience and some experimental work might be interpreted to be the more influential of the two phases and (3) resection of varying amounts of the acid secreting

body of the stomach, thereby reducing the acid secreting target* of parietal cells which can respond to any of the three avenues of stimulation. Simple drainage procedures, such as gastroenterostomy or pyloroplasty, are no longer considered adequate in their own right as a primary attack, although they are used as complementary procedures to the above.

DISTAL SUBTOTAL GASTRIC RESECTION

This operation began to supplant simple gastroenterostomy and pyloroplasty in the United States some thirty years ago having been introduced from the European clinics by Berg and advocated by Strauss as early as 1917. At the time of its initial use, the physiologic rationale was not as well understood as now. It seemed a radical departure to subtotally remove an important organ for an ulcer no more than a centimeter in diameter. Its proponents were criticized and it was compared to the absurdity of amputating a leg to relieve a varicose ulcer. Contrast this attitude with the report in 1932 of the Committee of the American Gastroenterological Association which, after several years of study reported 95 per cent satisfactory results following subtotal gastric resection.

The Physiologic Basis for the Procedure. Distal gastrectomy in removing the antrum, ablates the delayed gastric hormonal (gastrin) phase of acid secretion and, at the same time depending on the level of resection, removes a varying percentage of the acid secreting target, or body of the stomach. Those using the method advocate from 60 to 80 per cent resection. It is essential to remove the entire antrum down to and beyond the pylorus an accomplishment that may not be easy in the advanced penetrating ulcer. That the resection has extended beyond the pylorus into the duodenum, particularly on the posterior wall should be insured by a recorded histologic confirmation.

Among those who advocate subtotal gastric resection there is a distinct difference of opinion as to the proximal extent of resection that is, the amount of target remaining to be exposed to the stimulating vagal and other physiologic stimuli. The majority speak of a 75 per cent subtotal resection. Unfortunately it is difficult to accurately determine the amount of stomach that is left. Priestley uses a formula developed by Heinrich, while Moore and Harkins have developed a method utilizing planimetry

VARIETIES OF SURGICAL ATTACK FOR PEPTIC ULCER

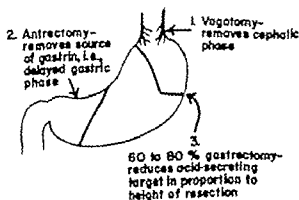


Figure 43. See text for the combinations of the surgical attacks illustrated above.

and weight. This is designed to estimate more accurately the percentage of stomach that is left. Hart prefers a 60 to 65 per cent resection, preserving thereby more gastric capacity and presumably better postoperative nutrition. He believes that clinicians should be more liberal in advising surgery for the patient who cannot live a reasonably satisfactory life with his ulcer but that surgeons should be more conservative in this primary resection reserving the more radical resection for the few who suffer recurrence. He believes that all surgical patients should not be inflicted with a high subtotal resection and the increased risk of undernutrition and "dumping" in order to protect better the few who are apt to develop recurrent ulcer. It does seem reasonably established that the higher the resection, the lower the incidence of recurrent ulcer but that the higher resection may result in a greater percentage of the "small stomach syndrome" and "dumping."

After gastric resection the restoration of *gastrointestinal continuity* presents a separate consideration. Whereas resection is designed to control ulcer by contrast the various methods of re-establishing continuity are aimed at reducing to a minimum the untoward sequelae therefrom, although some also claim enhancement of ulcer control by the particular anastomosis of their choice. Essentially the choice is between an end-to-end gastroduodenostomy (Billroth I) on the one hand, and an end-to-side gastrojejunostomy (Billroth II) on the other. Certainly the accomplished surgeon will be versed in the variations of both methods so that he will not be forced to stretch a single method beyond its reasonable limitations.

Gastrojejunostomy After resection for duodenal ulcer gastrojejunostomy (Fig. 44) remains the most widely used anastomosis. This is for two reasons. In the first place it can be accomplished without tension on the suture line regardless of the height of the

METHODS OF ANASTOMOSIS AFTER PARTIAL OR SUBTOTAL GASTRECTOMY

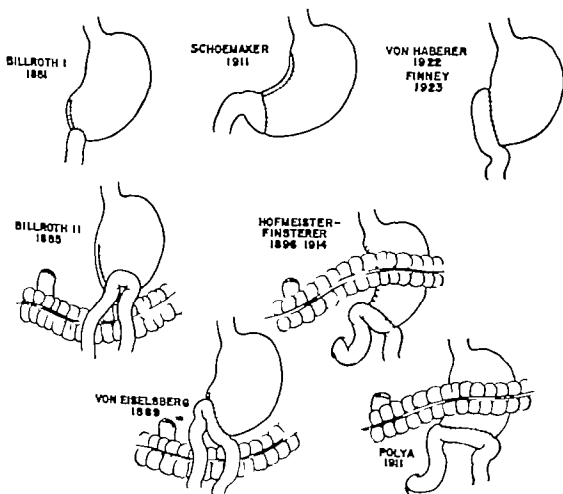


Fig. 44

resection and, in the second place the deformed duodenum may not lend itself well to anastomosis and the surgeon is compelled to use the jejunum. In those clinics where surgery for ulcer is restricted to the more complicated cases, the surgeon has to deal with a relatively large number of advanced deformities of the duodenum. He may therefore for technical reasons have to forego the physiologic and mechanical advantage of the Billroth I gastroduodenostomy in many of these. Actually in the larger series of followed cases reported, the difference in untoward sequelae for the two methods has not been impressive and Ordahl and his associates surprisingly found a higher incidence of recurrent ulcer to follow the Billroth I than the Polya anastomosis where the same extent of gastric resection was done for duodenal ulcer.

In anastomosing the end of the gastric stump to the jejunum the entire cross section of the stump may be anastomosed (Polya 1911 and Moynihan 1923) or the lesser curvature side may be closed using a lesser width for anastomosis (von Eiselsberg 1889 and Hofmeister 1896). For the higher lesser curvature ulcer there are technical advantages in closing rather than anastomosing the lesser curvature. The functioning diameter of the anastomosis will depend upon the diameter of the distal jejunal loop and it has been pointed out by some that a larger anastomosis could enhance the chance for "dumping" into the proximal loop. Prohaska, Govostis and Kirsteins have suggested that stomal dysfunction following subtotal gastrectomy is caused, not by stomal edema, but rather by an atonic gastric pouch in which the efferent outlet is placed above the afferent inlet. It was found that this combination occurred more frequently in the "anti-peristaltic" end-to-end gastrojejunostomy in which the distal loop of jejunum is placed in continuity with the lesser curvature of the stomach. If a retrocolic anastomosis is made proponents point out two advantages. A shorter loop of jejunum (1 to 3 inches distal to Treitz ligament) can be used and one avoids the mechanical disadvantages of pulling a longer loop of jejunum around in front of a heavy greater omentum in the obese patient. Some would meet this last objection by removing the greater omentum in the extremely obese. The shorter the jejunal loop employed, the higher will be the concentration of alkaline, pancreatic and biliary secretions which bathe and protect the anastomosis. That a longer loop may

predispose to recurrent ulcer has been explained by Kiriluk and Merendino on this basis rather than on the basis of an increasing inherent vulnerability of the more distal jejunal mucosa. The posterior anastomosis is technically a little more difficult in that the margins of the surgical aperture in the transverse mesocolon (which should always be made to the left of the midcolic vessels) must be sutured in a collar like manner to the stomach above the anastomosis. If this is not done, the aperture could descend and strangle the anastomosis and create obstruction to either or both the afferent and efferent loops. The anterior anastomosis may be preferable in a secondary resection where the transverse mesocolon is indurated from marginal ulceration or where its vascular arcades are not sufficiently wide to permit an adequate opening. In either the antecolic or retrocolic anastomosis one should try to see that the anastomosis lies transversely without tension and that the aperture of the afferent loop is not lower than the efferent loop. Some would insist that the proximal or afferent loop be anastomosed to the lesser curvature and some prefer this in the postcolic anastomosis. However in the antecolic anastomosis this may at times be reversed and the proximal loop be anastomosed to the greater curvature, if by so doing it seems to assure a better transverse position without tension.

Gastroduodenostomy. The Billroth I (1881) or variations thereof is used by the majority of surgeons following resection of benign gastric ulcer because (1) a lesser resection is usually performed for gastric ulcer and (2) the uninvolved duodenum lends itself well to anastomosis. There are staunch advocates of the method and it is used also after resection for duodenal ulcer. When the deformed stump does not permit end-to-end anastomosis an end-to-side gastroduodenostomy (von Haberer 1922 and Finney 1923) is employed (Fig. 44). Experimentally the Harkins group found restoration by gastrojejunostomy following partial gastrectomy to result in a higher level of gastric secretory activity than after gastroduodenostomy. Marginal ulceration in the animals occurred earlier and oftener after gastrojejunostomy. There is experimental evidence suggesting that excess acid in the duodenum acts as an inhibitory influence on the further secretion of acid by the stomach, probably through a hormone influence. This had been suggested by Sokolov as early as 1904. The work of Harkins group confirmed

that of Dragstedt's group that this protective influence by contrast is lost after gastrojejunostomy. Everson has shown in the experimental animal at least that the assimilation of fat and protein is reduced less after the Billroth I than after the Billroth II procedures. Also it is pointed out that the duodenal anastomosis is closer to the protective alkaline reinforcement of pancreatic and biliary secretions and that the duodenum, as contrasted to the jejunum, is guarded by Brunner's glands which secrete additional alkali and a protective mucus.

Mechanically of course an end-to-end anastomosis approaches the normal more than an end-to-side. There is one less suture line, the lower compartment of the abdomen is not soiled during the anastomosis and one does not have to deal with the problems of the greater omentum and the mesentery of the colon. These advantages might be considered overwhelmingly in favor of gastroduodenostomy. However in actual clinical practice the follow-up results while hard to evaluate because of the many variables introduced, nonetheless fail to convince many authorities either as to its advantage in ulcer control or in reduction of untoward sequelae. They would warn against overextending the indications for its use particularly where technical hazards make the anastomosis difficult. Coliglier Moir and Wrigley found the ulcer recurrence rate in a three year follow-up rose from 2 to 17 per cent when the Billroth I was substituted for the Polya procedure, a 75 per cent resection having been done in each series.⁶ Ordahl, Ross and Baker found the Billroth I to be preferable for gastric ulcer but to be followed by a 28.6 per cent incidence of recurrent ulcer when used for duodenal ulcer. On the other hand, in a larger series of cases, Harkins and Nyhus reported a recurrence rate (proved and unproved cases) of 3.5 per cent. While it is true that technically if one frees the entire greater curvature and divides the left gastric artery and mobilizes the duodenum, the anastomosis can be accomplished in most cases without tension in the hands of the more expert, the less experienced may be ill advised to attempt it. There does not seem to be sufficient proved advantage in the Billroth I anastomosis to justify stretching the reasonable indications for its use. To do so could tempt one to do an inadequate resection of the stomach or to establish the anastomosis under tension. The ideal end result after gastric resection, regardless of the type of anastomosis

is a small stomach having a capacity of a pint or less with low acid secretion limited to the mealtime and a gastric emptying time of about one-half an hour.

VAGOTOMY

The transmission by way of the vagus nerves of influences important to gastric secretion was pointed out by Rokitsansky a hundred years ago. Vagotomy as a modality of surgical treatment for peptic ulcer was introduced by Dragstedt and Owens in 1943. Heretofore the attack on ulcer had been a local one. Vagotomy by contrast, takes into account the psychosomatic factor in the etiology of ulcer in that it blocks the neurogenic avenues of stimulating gastric secretion and accomplishes more completely the purpose for which the anticholinergic drugs have long been used. Furthermore, it was shown that vagotomy presumably on the basis of acid reduction relieved the pain of ulcer. It had the advantage of not sacrificing the volume capacity of the stomach. But on the other hand, it may be argued that it affords no direct attack for the complications of ulcer (perforation, obstruction and hemorrhage). It is to be remembered that in most centers it is these complications that constitute the chief indication for surgery. In general it may be said that vagotomy alone is no longer considered an adequate primary attack for primary ulcer.

One of the more undisputed indications for vagotomy *per se* is secondary gastrojejunal ulceration. The Committee of the American Gastroenterological Association found a favorable response from vagotomy in 81 per cent of patients with recurrent ulcer following simple gastroenterostomy and in 72 per cent with ulcer following gastric resection. Walters, Chance and Berkson reported similar results. Whether one should be content in applying vagotomy alone for recurrent ulcer may require individualization. Healing of the very indurated ulcer may be prevented by its scar and resection of the ulcer should be done in addition to the vagotomy. One should also search for the cause of previous failure of ulcer control, such as a retained portion of the gastric antrum, inadequate resection of the body of the stomach, a too-long jejunal loop or a poor placement of the gastroenterostomy. Any of these deficiencies will require correction in addition to vagotomy. In recurrent ulcer a search for an islet alpha cell tumor of the pancreas should also be made.

VAGOTOMY COMBINED WITH GASTROJEJUNOSTOMY OR PYLOROPLASTY

Because vagotomy impairs the motor function of the stomach and because the healing of an active ulcer after vagotomy has also resulted in obstructive retention, it is now considered best to add either gastroenterostomy or pyloroplasty to the vagotomy in order to produce better gastric emptying. But it was pointed out by Lillehei that by this combination the stomach is left unprotected by food during the delayed gastric phase of hypersecretion. Two individually inadequate procedures it may be argued, are thus combined and the patient exposed to the untoward effects of each protracted diarrhea being the most objectionable. There is no justification to add vagotomy to an adequate 75 per cent gastric resection because this has not afforded additional protection, according to the follow-up report of the Committee of the American Gastroenterological Association.

However it should be mentioned again that Dragstedt has emphasized the possible specificity of vagotomy for the control of duodenal ulcer. His experimental work suggests to him that duodenal ulcer results from the neurogenic phase of hypersecretion, in contrast to gastric ulcer which he believes

should be more specifically controlled by an attack on the hormonal phase of hypersecretion. It would seem, however, that some of the earlier advocates have forsaken gastrojejunostomy in combination with vagotomy and have substituted antrectomy and gastroduodenostomy as an auxiliary procedure to vagotomy. The generally accepted situation in which a number of surgeons prefer simple gastroenterostomy and vagotomy arises when there is a penetrating duodenal ulcer or a markedly deformed duodenum which makes pyloroplasty and a Billroth I gastroduodenostomy difficult or hazardous to accomplish. The incidence of recurrent ulcer is approximately twice that following a two-thirds gastric resection being about 10 per cent (5 per cent proved and 5 per cent suspected). However the mortality in vagotomy and gastroenterostomy when applied to the difficult penetrating ulcer would be appreciably less than that following resection of the ulcer.

Technical Considerations. Vagotomy may be accomplished supradaphragmatically by way of a transthoracic approach or subdiaphragmatically by way of an abdominal approach. It is ineffective unless all of the vagal fibers are divided. The original 10 per cent failure to accomplish complete vagotomy may be reduced with greater experi-

GASTROJEJUNOSTOMY & PYLOROPLASTY

Note that these operations previously discarded are now employed in conjunction with vagotomy

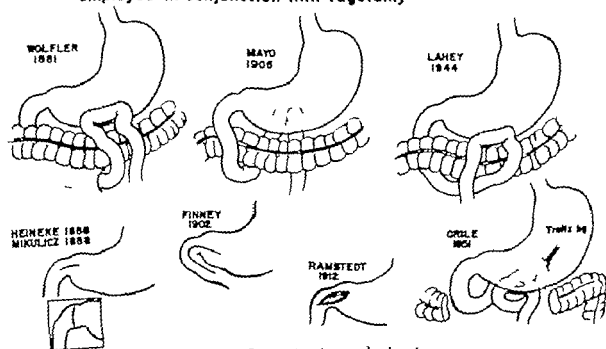


Figure 45. Gastroenterostomy and pyloroplasty

ence of the surgeon and a better appreciation of the varying anatomic course of the vagal nerve fibers. In performing the concomitant gastrojejunostomy Dragstedt has stated that it should be placed in the antrum within 4 or 5 cm. of the pylorus. Crile has emphasized placing it distally in the dependent portion of the stomach and warns against an inversion by multiple layers of suture (Fig. 45)

ANTRECTOMY COMBINED WITH VAGOTOMY

Physiologic and Surgical Considerations. According to present knowledge if the entire antrum of the stomach is resected, the source of the hormone gastrin would be thereby removed and the gastric hormonal phase of gastric hypersecretion would be eliminated. If in addition both vagus nerves are divided, the neurogenic or cephalic phase of secretion is also destroyed, leaving only the intestinal hormone (third phase) to stimulate the remaining gastric fundus to secretion. This should, theoretically at least, accomplish total or near total achlorhydria and peptic digestion cannot occur in the absence of hydrochloric acid. According to present concepts this should, therefore, accomplish near perfect control of the ulcer diathesis. It should be technically easier to perform than is a high subtotal gastric resection, particularly in the fat or barrel chested individual with a small stomach situated high beneath the diaphragm. By removing only the antrum a greater gastric capacity is retained and Zollinger believes this an important advantage in the patient who is preoperatively below normal weight.

The chief objection to this combination is the fact that it has not as yet had sufficiently wide application and patients have not been followed long enough for dependable evaluation. Many surgeons prefer to await the results of longer study from the large, qualified experimental centers before adopting this method. Subtotal gastrectomy by contrast has been used in this country for thirty years and, while the results are by no means perfect, it has afforded approximately 95 per cent good results. It remains one of the more satisfactory abdominal operations. Whether the resulting total achlorhydria of vagotomy and antrectomy is completely desirable is a question also raised by some and, in this connection, the association of polyps and pernicious anemia with achlorhydria has been cited. A diminished absorption of fat has been found to follow vagotomy

The vagus nerves supply not only the stomach, but also the entire small intestine at least part of the colon the liver and the pancreas. Whether total vagotomy at the diaphragmatic level for benign peptic ulcer therefore will in the long term follow up prove justifiable is still held by some as open to question.

Furthermore in the severely penetrating duodenal ulcer or in the deformed duodenum a Billroth I gastroduodenostomy may not be feasible and then the antrectomy must be completed by gastrojejunostomy or else the antrectomy substituted by simple gastroenterostomy. Actually the results in the few centers which have reported on this procedure have uniformly shown an absence of recurrent ulcer except in a few instances where vagotomy was incomplete. Most reports speak of a "50 per cent" or "limited" gastrectomy in combination with the vagotomy rather than limiting the resection strictly to antrectomy. This should be borne in mind by those adopting the method. In general, the antrum can be recognized grossly by its smooth surface and adherent submucosa in contradistinction to the loose redundant rugal folds of the fundus (Fig. 42). It requires a resection of from 40 to 50 per cent to insure complete removal of the antrum. Since the antral cells extend up the lesser curvature, the resection should be higher on the lesser curvature side. The untoward effects have been about the same as following the high subtotal gastrectomy. In any surgical approach depending on vagotomy alone or in combination there is the hazard of failure from incomplete vagotomy. This was found to be as high as 10 per cent in the earlier series.

MISCELLANEOUS SURGICAL MODALITIES

Numerous combinations of surgical procedures for duodenal ulcer have been applied or recommended and some of these are currently being studied in centers which by virtue of their organized follow-up ulcer clinics are in a position to supply an eventual evaluation. Wangensteen has forsaken his so-called tubular gastric resection in which the fundus and greater curvature were resected longitudinally and the lesser curvature preserved as a reconstructed tube (Fig. 46). This approach was designed to resect all of the acid-secreting target. He now is employing a "segmental three-quarter mid gastric sleeve resection with anastomosis between the remaining small fundic pouch and the antrum, combined with a Heineke-Mi-

VAGOTOMY COMBINED WITH GASTROJEJUNOSTOMY OR PYLOROPLASTY

Because vagotomy impairs the motor function of the stomach and because the healing of an active ulcer after vagotomy has also resulted in obstructive retention, it is now considered best to add either gastroenterostomy or pyloroplasty to the vagotomy in order to produce better gastric emptying. But it was pointed out by Lillehei that by this combination the stomach is left unprotected by food during the delayed gastric phase of hypersecretion. Two individually inadequate procedures it may be argued, are thus combined and the patient exposed to the untoward effects of each protracted diarrhea being the most objectionable. There is no justification to add vagotomy to an adequate 75 per cent gastric resection because this has not afforded additional protection according to the follow up report of the Committee of the American Gastroenterological Association.

However it should be mentioned again that Dragstedt has emphasized the possible specificity of vagotomy for the control of duodenal ulcer. His experimental work suggests to him that duodenal ulcer results from the neurogenic phase of hypersecretion, in contrast to gastric ulcer which he believes

should be more specifically controlled by an attack on the hormonal phase of hypersecretion. It would seem, however, that some of the earlier advocates have forsaken gastrojejunostomy in combination with vagotomy and have substituted antrectomy and gastroduodenostomy as an auxiliary procedure to vagotomy. The generally accepted situation in which a number of surgeons prefer simple gastroenterostomy and vagotomy arises when there is a penetrating duodenal ulcer or a markedly deformed duodenum which makes pylorotomy and a Billroth I gastroduodenostomy difficult or hazardous to accomplish. The incidence of recurrent ulcer is approximately twice that following a two-thirds gastric resection being about 10 per cent (5 per cent proved and 5 per cent suspected). However the mortality in vagotomy and gastroenterostomy when applied to the difficult penetrating ulcer would be appreciably less than that following resection of the ulcer.

Technical Considerations. Vagotomy may be accomplished supradaphragmatically by way of a transthoracic approach or subdiaphragmatically by way of an abdominal approach. It is ineffective unless all of the vagal fibers are divided. The original 10 per cent failure to accomplish complete vagotomy may be reduced with greater experi-

GASTROJEJUNOSTOMY & PYLOROPLASTY

Note that these operations previously discarded are now employed in conjunction with vagotomy

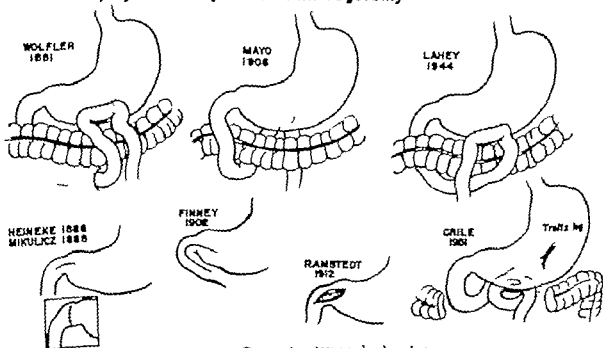


Figure 45. Gastroenterostomy and pyloroplasty

ence of the surgeon and a better appreciation of the varying anatomic course of the vagal nerve fibers. In performing the concomitant gastrojejunostomy Dragstedt has stated that it should be placed in the antrum within 4 or 5 cm. of the pylorus. Crile has emphasized placing it distally in the dependent portion of the stomach and warns against an inversion by multiple layers of suture (Fig. 45).

ANTRECTOMY COMBINED WITH VAGOTOMY

Physiologic and Surgical Considerations. According to present knowledge if the entire antrum of the stomach is resected, the source of the hormone gastrin would be thereby removed and the gastric hormonal phase of gastric hypersecretion would be eliminated. If in addition both vagus nerves are divided, the neurogenic or cephalic phase of secretion is also destroyed, leaving only the intestinal hormone (third phase) to stimulate the remaining gastric fundus to secretion. This should, theoretically at least, accomplish total or near total achlorhydria and peptic digestion cannot occur in the absence of hydrochloric acid. According to present concepts this should, therefore, accomplish near perfect control of the ulcer diathesis. It should be technically easier to perform than is a high subtotal gastric resection, particularly in the fat or barrel chested individual with a small stomach situated high beneath the diaphragm. By removing only the antrum a greater gastric capacity is retained and Zollinger believes this an important advantage in the patient who is preoperatively below normal weight.

The chief objection to this combination is the fact that it has not as yet had sufficiently wide application and patients have not been followed long enough for dependable evaluation. Many surgeons prefer to await the results of longer study from the large, qualified experimental centers before adopting this method. Subtotal gastrectomy by contrast has been used in this country for thirty years and, while the results are by no means perfect, it has afforded approximately 95 per cent good results. It remains one of the more satisfactory abdominal operations. Whether the resulting total achlorhydria of vagotomy and antrectomy is completely desirable is a question also raised by some and, in this connection, the association of polype and pernicious anemia with achlorhydria has been cited. A diminished absorption of fat has been found to follow vagotomy

The vagus nerves supply not only the stomach but also the entire small intestine, at least part of the colon, the liver and the pancreas. Whether total vagotomy at the diaphragmatic level for benign peptic ulcer therefore will in the long term follow-up prove justifiable is still held by some as open to question.

Furthermore in the severely penetrating duodenal ulcer or in the deformed duodenum a Billroth I gastroduodenostomy may not be feasible and then the antrectomy must be completed by gastrojejunostomy or else the antrectomy substituted by simple gastroenterostomy. Actually the results in the few centers which have reported on this procedure have uniformly shown an absence of recurrent ulcer except in a few instances where vagotomy was incomplete. Most reports speak of a "50 per cent" or "limited" gastrectomy in combination with the vagotomy rather than limiting the resection strictly to antrectomy. This should be borne in mind by those adopting the method. In general, the antrum can be recognized grossly by its smooth surface and adherent submucosa in contradistinction to the loose redundant rugal folds of the fundus (Fig. 42). It requires a resection of from 40 to 50 per cent to insure complete removal of the antrum. Since the antral cells extend up the lesser curvature the resection should be higher on the lesser curvature side. The untoward effects have been about the same as following the high subtotal gastrectomy. In any surgical approach depending on vagotomy alone or in combination, there is the hazard of failure from incomplete vagotomy. This was found to be as high as 10 per cent in the earlier series.

MISCELLANEOUS SURGICAL MODALITIES

Numerous combinations of surgical procedures for duodenal ulcer have been applied or recommended and some of these are currently being studied in centers which by virtue of their organized follow-up ulcer clinics are in a position to supply an eventual evaluation. Wangenstein has forsaken his so-called tubular gastric resection in which the fundus and greater curvature were resected longitudinally and the lesser curvature preserved as a reconstructed tube (Fig. 46). This approach was designed to resect all of the acid secreting target. He now is employing a "segmental" three-quarter mid gastric sleeve resection with anastomosis between the remaining small fundic pouch and the antrum, combined with a Heineke-Mi-

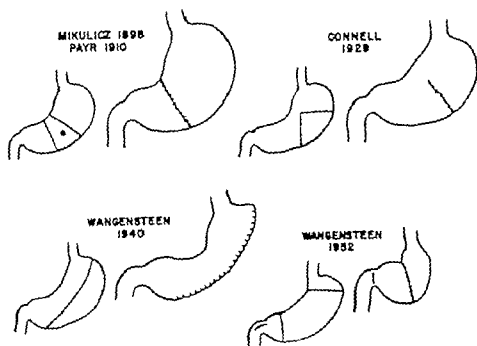


Figure 46 Miscellaneous surgical modalities.

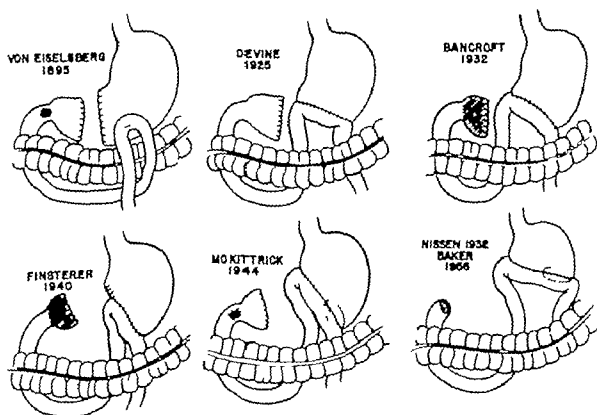


Figure 47 Miscellaneous surgical exclusion operations.

ulcer pyloroplasty (Fig. 45) By this method the greater portion of the acid-secreting body of the stomach and a part of the hormone-secreting antrum are removed and at the same time some of the antrum is intentionally left. Wangensteen and Berni, who has used a similar surgical approach have suggested that any remaining acid secretion will thereby contact the remaining antrum and invoke the inhibitory function of the latter against further acid secretion. Any antianemic factor of the antrum is likewise preserved as well as its protective mucus and alkaline secretions. Waddell and Bartlett have revived antral exclusion and combined it with vagotomy presumably expecting the latter to protect against the high incidence of recurrent ulcer seen in former years when antral exclusion was suggested by Devine (Fig. 47) and adopted by others as a technical solution for the difficult penetrating duodenal or pyloric ulcer. Since it requires a long period to evaluate the effective ulcer control and untoward effects of any surgical procedure, the majority of surgeons may prefer to await longer follow-up reports before applying these newer methods.

FACTORS IN THE SELECTION OF THE SURGICAL PROCEDURE

A follow-up study of results has shown that each surgical procedure so far applied to duodenal ulcer leaves something to be desired in some patients both from the standpoint of failure to control ulcer and because of untoward sequelae. Even those who are partial to a particular procedure may at times substitute another procedure either for (1) *technical reasons* or (2) because of certain *clinical considerations*. In the latter category Zollinger and Smithwick are among those who, for the underweight patient, would prefer to preserve more gastric capacity. Therefore, instead of applying subtotal gastric resection, they would apply either vagotomy with gastroenterostomy or vagotomy with antrectomy and a Billroth I anastomosis. Zollinger observed that whereas the majority of his patients who were overweight prior to surgery would hold their ideal weight after an extensive gastric resection of the Hofmeister type, this was not true for the majority of the patients who were below their ideal weight at the time of operation, whether or not they had at one time been above this weight. For the latter he would not do an extensive resection. For those deemed to be unusually sus-

ceptible to psychogenic influences, the addition of vagotomy may take on added importance. Those favoring gastric resection may vary the extent of the resection from 60 per cent to 80 per cent, depending upon the adjudged severity of the disease, preserving a greater gastric capacity in the milder cases.

Bruce adopting Kay's method of measuring quantitatively gastric acid in response to histamine has attempted to evaluate the severity of the ulcer diathesis and to fashion individually the type of surgical procedure to be applied in each case. Kay found that a dose of 0.04 mg. of histamine per kg. of body weight will elicit the maximum secretory response from the entire population of parietal cells and that this response varies widely with different patients with ulcer. But since it is a maximum response, it is constant on repeated tests for the same individual. Greater doses of histamine beyond that stated did not further affect secretion. The patient is protected from side effects of the large doses of histamine by antihistamine drugs and by gastric tube the secretion of acid is measured for the forty-five-minute period after histamine is given. Bruce has considered the test as a measure of the severity of the ulcer diathesis and, on histologic examination of stomachs resected for ulcer he has found a correspondingly greater density of parietal cells per unit of mucosa in those with the greater secretory response. By means of the test he divides the patients requiring surgery for duodenal ulcer into three clinical groups. For those with only moderate elevation of secretion he would apply vagotomy and gastroenterostomy particularly where technical hazards or impaired risk argues for this technically easier procedure. For those with more marked elevation of secretion he would apply 60 per cent subtotal resection, and for those with very high secretion, 75 per cent resection plus vagotomy. He finds that the 60 per cent distal resection will reduce by 45 per cent the milliequivalent level of preoperative secretion. He expects by surgery to reduce the maximum secretion to within the range found in normal patients without ulcer.

In the main, *technical problems* become the more common reason for altering the surgical attack. Priestley refers to the overweight, barrel-chested patient with a well rounded belly and a firm, thick, abdominal wall, who commonly has a small stomach situated high and deep in the abdomen in a virtually transverse direction. He points



A



B

Figure 48 A, Method of gastric resection for the deformed duodenum with ulcer perforating into the pancreas. The duodenum is transected distal to the pylorus without regard to the large, perforating ulcer which is bordered by dense scar and thickened pancreatic capsule. A frozen section is made from the posterior distal end of the specimen to prove duodenal mucosa and to insure for the record that all of the antrum has been removed. B Fine interrupted silk is used to approximate the seromuscular layer of the anterior duodenal wall to the thickened pancreatic capsule and to the proximal margin of the ulcer. The mucosal edge is thereby inverted without suture (Baker J. W., Boyd, R. S. and Foster R. A.: *Ann Surg.*, vol. 142)

out that in such a patient an adequately high gastric resection is more difficult. The greater omentum and the mesenteries infiltrated with fat add to the hazards of any of the Billroth II modifications. Such patients, of course should be reduced in weight before surgery. Unfortunately they have such intractable symptoms that their ulcer management of increased milk and cream intake granted it has been prolonged too long, has added to their weight. In such instances some surgeons would do an antrectomy and vagotomy the lesser resection with a Billroth I anastomosis being much easier than a higher resection.

Resection of the peptic ulcer which has perforated through the duodenum into the pancreas is fraught with technical hazards, predisposing to increased morbidity and mortality. The danger to the common bile duct, the pancreas and its ducts, and an insecure duodenal closure have influenced some staunch resectionists to substitute other measures for gastrectomy. In these patients, vagotomy and gastroenterostomy, antral exclusion and gastroenterostomy or a two-stage gastric resection have been employed. The technical hazards can be overcome by excluding the ulcer into the duodenal stump. This can be accom-

plished without sacrificing the physiologic requirements embodied in resection (Fig. 48). The importance of proving histologically that the resection has been carried beyond the pylorus to insure removal of all of the antrum must be stressed. The method is not applicable in emergency resections done to control bleeding from the duodenal ulcer. Such exclusion was first suggested by Nissen and is to be distinguished from the antral exclusion of Devine (Fig. 47) which has proved unsuccessful in ulcer control, and from the Bancroft or Finsterer technique in which the antral mucosa is rimmed out but the seromuscular coats are preserved for closure. This latter technique meets the physiologic obligation of removing the antrum, but is less accurate in removing all of the antral mucosa. Furthermore, it is technically more difficult to accomplish with neatness and safety. Baker compared a followed series of 122 patients, in which resection was accomplished by leaving the ulcer and excluding it into the duodenum, with 156 patients, in whom the ulcer was resected. He found the mortality in the excluded group to be 0.8 per cent (one patient) and in the resected group to be 1.9 per cent (three patients).

recurrent ulcers suspected in each series, only one in each series requiring secondary surgery.

Duodenostomy In occasional instances the surgeon may have elected to resect the duodenal ulcer only to find, as the resection progresses, an unanticipated difficulty which prevents a convincingly secure closure of the duodenal stump. In order to convert a possible leak into a controlled fistula, the duodenal stump may be closed around a small soft rubber catheter which is brought through the abdominal wall as a duodenostomy. It is best to reinforce this closure of the stump by tacking the adjacent omentum over the exit of the tube. Most surgeons leave either a cigarette or suction drain in the subhepatic space adjacent to the duodenostomy catheter. Welch first called attention to the advantage and safety of duodenostomy after gastric resection and later reported ten cases in which the procedure was used without a death. He prefers to combine the duodenostomy with a double catheter jejunostomy after the manner of Allen and Donaldson. Priestley, Ross and Warren, Hoerr and Caudell, Garrison and Lee are among those who endorse the use of duodenostomy for the rare instance in which closure of the duodenal stump presents a problem. The tube is clamped about the eighth day and removed on the tenth day; the drain site usually healing promptly thereafter. Stomal obstruction will require leaving the tube longer. Measured replacement of the fluid and electrolyte loss is obviously important. Complications include leakage around the duodenal catheter, an anastomotic ulcer in the immediate post-operative period presumed due to loss of the protective alkaline pancreatic and biliary secretions, and prolonged drainage from the sinus tract.

It should be remembered that a temporary duodenostomy may be more simply accomplished by placing an indwelling nasogastric tube at the time of the gastrojejunostomy into the proximal loop well beyond Treitz ligament. In such instances, additional holes may be cut in the tube to simultaneously suction the remaining stomach. The disadvantage of this method is the discomfort of the patient and the other complications of nasogastric tubes which limit their usefulness beyond one or two days post-operatively.

If the dilemma which makes duodenostomy advisable can be anticipated before irrevocable resection of the ulcer has been

joined it is obviously better and safer to substitute either a two-stage resection or vagotomy combined with gastrojejunostomy.

ULCER COMPLICATED BY OBSTRUCTION

Gastric retention may complicate pyloric or duodenal ulcer as a result of either (1) spasm (2) edema or (3) organic deformity. This may be discovered only upon roentgenologic examination or it may be clinically evident because of vomiting. Retention of gastric acid prevents healing of the ulcer and may become associated with secondary gastric erosions. Persistent vomiting may lead to hypochloremic alkalosis through the loss of electrolytes—particularly chloride ions—and prolonged vomiting to hypokalemia. The state of nutrition may suffer and total blood volume may be reduced.

The treatment is directed at gastric decompression either by continuous gastric suction or by aspiration of the stomach night and morning, combined with a liquid diet. To be emphasized is the fact that anticholinergic drugs are contraindicated because they serve to increase the retention. Usually, forty-eight hours of continuous suction will relieve any superimposed edema and the subsequent plotting of twenty-four hourly oral intake versus gastric output will determine the degree of any persisting organic obstruction. During this trial period, serum electrolyte determinations should be made and deficiencies gradually corrected. Unrelieved obstruction is an indication for surgery. However surgery should not be undertaken until the superimposed edema and gastric distention has been relieved by several days of suction and until the electrolyte imbalance has been at least partially restored. Surgery done before this is accomplished may result in hazardous resection and insecure anastomosis. An anastomosis may become smaller than desired when elimination of edema and restoration of gastric tone are accomplished after rather than before operation. On the other hand, in the face of unrelenting obstruction, gastric suction and parenteral support should not be pursued more than two to three days. The depletion may be increased rather than improved and surgical risk will not be lessened by further delay. In an occasional extreme instance of this kind, when ideal preparation cannot be attained by parenteral feedings, it may become advisable to proceed with gastric resection and at the time of surgery to perform concomitant jejunos-

tomies after the manner of Allen and Donaldson. These surgeons recommend that two catheters be placed into the jejunum below the gastrojejunostomy with the one catheter extending beyond the anastomosis into the gastric stump for decompression and the second catheter extending downward into the jejunum for postoperative feeding. This will afford the malnourished patient protection against postoperative edematous obstruction and at the same time allow early enteral feeding. This will be seldom necessary but the point should be emphasized that parenteral feeding does not substitute for enteral feeding over a long period. Furthermore the prolonged use of nasogastric tubes is to be avoided.

GASTROSTOMY

For decades gastrostomy has been employed for the purpose of feeding the patient with esophageal obstruction either from cancer or from stricture. Gastrostomies for this purpose may be a double purse-string inversion of a mushroom or plain catheter after the manner of Stamm, or a tubal valvular gastrostomy after the manner of Spivack, or other variations therefrom. At present a more common use for gastrostomy is as an adjunct to extensive abdominal surgery when gastric or intestinal distention can be anticipated, such as after radical resection of obstructing carcinomas of the colon and total colectomies for ulcerative colitis. Some surgeons use gastrostomy after limited gastric resection combined with vagotomy.

Farris and Smith use a special Foley catheter inserted into the anterior wall of the stomach and brought out through a stab incision at a point where the stomach is in normal apposition with the abdominal wall, insuring fixation of the visceral and parietal peritoneum with several sutures. This is done at the time of major surgery and suction is maintained in the postoperative period until the hazard of ileus is terminated. The patient is more comfortable than with nasogastric suction, particularly when the need for suction is prolonged. Placing this large and rather stiff rubber tube upward (cephalad) in the stomach serves to decompress the distended stomach and its excess contents yet does not withdraw the small volume of gastric content from the distal end of the stomach. This residue in the distal stomach may pass through the pylorus as normal peristalsis recovers.

ULCER WITH FREE PERFORATION

Peptic erosion may penetrate through the duodenal or gastric wall to produce a free perforation with escape of duodenal or gastric content into the peritoneum. By contrast, the posterior duodenal ulcer and occasionally the posterior gastric ulcer perforate into the pancreas without creating the emergency of peritoneal spill. When the acid chyme strikes the peritoneal surface a dramatic clinical picture results. The generalized upper abdominal pain is so severe that the patient becomes afraid to move, for the slightest motion increases the pain. He may vomit at the onset, the abdominal muscles become boardlike and leukocytosis and a slight elevation of temperature invariably follow. While in the first six to twelve hours the picture may be mistaken preoperatively for acute cholecystitis, acute pancreatitis or closed loop intestinal obstruction, after six hours or more the picture may be more often mistaken for acute appendicitis. The agonizing upper abdominal pain and rigidity by then may have become replaced by localized pain and tenderness over the right lower quadrant and the leukocytosis will have increased. This is due to gravitation of the free duodenal content down the right iliac gutter with beginning peritonitis in this dependent area.

When perforation occurs on an empty stomach, the peritoneal spill is less than when it occurs after a meal, a factor influencing the need for lavaging the peritoneum at the time of operation. Usually a careful history, absence of liver dullness on percussion, and x rays of the upper abdomen in the upright and left lateral decubitus positions demonstrating free air under the diaphragm will confirm the diagnosis. Air under the left diaphragm suggests perforation of a gastric ulcer. Severe pain presenting in the shoulders or root of the neck suggests perforation of the esophagus and this possibility should be ruled out before the abdomen is incised. A broadened mediastinum, a pneumothorax or hydropneumothorax, as demonstrated by chest x ray may be associated with rupture of the esophagus. The serum amylase level should be determined to rule out pancreatitis although a moderate elevation can occur from peritoneal absorption of this enzyme following spillage from the perforated duodenum, particularly if perforation follows a heavy meal.

Treatment. As in the case of any perforated abdominal viscus, the earlier surgical

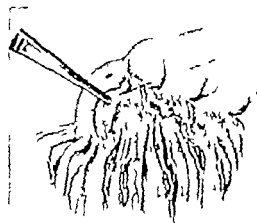
intervention is accomplished the better. Except for uncontrolled hemorrhage, there is no abdominal surgical emergency in which each of the early hours of delay is more costly. Unlike perforations farther along the intestine the acid chyme is not primarily infective, although secondary bacterial invasion becomes superimposed upon the chemical or enzymatic peritonitis. The insult to the peritoneum is roughly proportionate to the amount of the spill (greater if the perforation occurs after a meal) and the time interval between the perforation and its surgical correction. Within the first six hours, in an otherwise normal risk patient, the mortality is negligible. After this interval the mortality becomes appreciable as the peritoneum becomes more extensively involved and the patient becomes more depleted. Within the early period little preparation is necessary. In an exceptional case shock persisting beyond relief of pain may suggest coincident bleeding. This only intensifies the emergency and adds the requirement for blood transfusion. It is wise to place a nasogastric tube before surgery if there is abdominal distention. By a consideration of the preoperative history combined with the operative findings, the surgical problem will be resolved into a preference between (1) simple closure of the perforation and (2) a more definitive primary attack for the ulcer diathesis usually subtotal gastric resection including the perforated ulcer. In either case the peritoneum of the upper and lower abdominal fossae and pelvis should be carefully lavaged with normal saline to remove the acid, enzyme and food particles. Also irrespective of the surgical procedure selected, preoperative and postoperative therapy must include repair of the metabolic derangement caused by the peritonitis which results from the escape of gastric and duodenal contents. Cope has emphasized the importance of plasma therapy and the adequate replacement of potassium and nitrogen losses. The adequacy of the fluid therapy program is best judged by the renal output.

Simple closure is applied in perforated duodenal ulcer when (1) there is historically an absence of chronicity and at laparotomy the edges of the perforation are soft rather than firm or cartilaginous (2) the duodenum is not deformed to suggest recurrent episodes of ulceration or (3) the duodenal perforation is judged to be an acute erosion rather than the result of a chronic ulcer diathesis. Simple closure is also resorted to

for either the duodenal or gastric perforations seen late after diffuse peritonitis and a deteriorated state of the patient make gastric resection too hazardous. A technical consideration of importance is not to attempt a purse-string closure of the perforation. In the first place this suture is difficult to place in the inflamed edematous and friable duodenal wall and of greater importance invagination may result in obstruction of the duodenum during the postoperative period. An easier and more secure method is that of Roscoe Graham of tacking an omental graft over the perforation. The duodenocolic appendage of omentum adjacent to the inferior curve of the duodenum (between the duodenum and hepatic flexure of the colon) is useful in this procedure (Fig. 49). This is brought over the perforation and tacked with a few fine silk sutures to the duodenal wall. This seems preferable to a free omental graft and the sutures do not have to be taken close to the ulcer where the wall is friable. This appendage has proved so expeditiously useful in closing perforations and protecting duodenal stump closures after resection that I have termed it the "providential omentum." The peritoneum should be thoroughly lavaged and the abdomen closed without drains. A gastrostomy or a nasogastric tube may be used to protect the closure and as a therapeutic measure if peritonitis and ileus are present.

Primary gastric resection including the perforation is preferred to simple closure (1) if an ulcer diathesis of long standing has been well documented, (2) if by previous hemorrhage obstruction or intractability to a proper regimen the ulcer would qualify for resection and (3) if the perforation at laparotomy is bordered by firm scar or deformed duodenum and provided advanced generalized peritonitis does not rule against this more involved surgery. Coincident hemorrhage and obstruction are also indications for primary resection.

If the perforated gastric ulcer is not resected, at least biopsy should be performed. Because gastric ulcers are more difficult to close and because malignancy is always a possibility in gastric ulcer preference is for primary resection for the perforation proximal to the pylorus. Simple closure has been more commonly employed in the past in this country. In most reported series it has been found that about 50 per cent of patients with simple closure will have no more trouble whereas the other 50 per cent will require further treatment and often secondary



TRANSVERSE COLON

PROVIDENTIAL OMENTUM

*J. J. Moore*

Figure 49 For closing over the perforated duodenal ulcer the tag of gastrocolic omentum adjacent to the duodenum is tacked in a circumferential manner over the perforation with interrupted, fine, nonabsorbable sutures.

surgery. When there is reasonable doubt as to the necessity for resection, it would seem wise to apply simple closure even though a few patients may require a second operation. It is often difficult to evaluate satisfactorily the clinical picture in the emergency and this is particularly true in the average patients seen in the charity hospital where a high percentage of ulcer operations are either for perforation or massive hemorrhage.

The third alternative nasogastric suction and supportive treatment as a substitute for surgery has been explored by Seeley and Campbell. It has been adopted by others for the patient seen late after perforation and when the clinical picture is then one of advanced peritonitis rather than perforation. However I do not subscribe to the method even in these cases. The best treatment for advanced peritonitis associated with flous, a spiking temperature and leukocytosis is emergency preparation of the patient by gastric suction and antibiotic, electrolyte and fluid support, followed promptly, by laparotomy. Laparotomy is now primarily for evacuating fossae and loculated interintestinal pockets of pus. This is accomplished by stripping the intestine and irrigating the peritoneum with copious amounts of saline to which has been added penicillin and streptomycin. The perforation which by now has been sealed over with exudate is nevertheless securely closed over by omentum and gastrostomy is performed to relieve the distention of the peritonitis. The mortality in these desperately late cases is extremely

high, but some patients may be saved by these heroic methods—more it is believed, than by gastric suction alone. This method of treating peritonitis has been described by Burnett and others following perforative appendicitis is supported by my experience in a limited number of similar cases and should apply. I believe for peritonitis following perforated ulcer. It is admittedly unorthodox and will require more than present accumulated experience to prove the superiority that I prophesy for it.

GASTRODUODENAL HEMORRHAGE

General Considerations. Approximately 20 per cent of peptic erosions bleed and 5 per cent bleed massively. Melena is evidence of active penetration of the ulcer. It is not in itself an indication for surgery. When associated with evidence of shock, such as clammy perspiration or faintness a dangerous amount of blood (1500 cc. or more) may have been suddenly lost. Uncontrolled hemorrhage or recurrent episodes of severe bleeding are indications for surgery. Often the patient is brought to the emergency room with the story that he has fainted in the bathroom after "vomiting a bowlful of blood." The physician then faces the problem of determining the site of bleeding and the volume and rate of blood loss. He must promptly pursue a dependable course which will control the emergency. This can be a challenging responsibility. The inherent difficulty is in determining which patients will cease bleeding by conservative measures and which will die from lack of surgical control

of the hemorrhage. This must be accomplished before the emergency state of shock becomes irreversible. Sources of potentially fatal bleeding are for the most part in the upper gastrointestinal tract. *i.e.*, above the ligament of Treitz. For practical purposes they may be divided into three groups: (1) esophageal and gastric varices resulting from portal hypertension; (2) acute transient erosions and hemorrhagic gastritis for which gastric resection represents an unjustified sacrifice except as an emergency control of the hemorrhage to save life, and (3) the chronic peptic ulcer.

Carcinoma rarely causes massive hemorrhage although it may cause melena. Ulcerating lymphosarcoma of the stomach, however, not infrequently bleeds massively. It is estimated that 90 per cent of all upper gastrointestinal bleeding is from peptic ulcer. This is in keeping with the prevailing high incidence of peptic ulcer. The surgeon who has seen penetration and erosion into an artery in the crater of an ulcer can appreciate that such bleeding can be fatal. Blackford, Smith and Affleck showed that in a six year period in Seattle, with a population at the time of 350,000 there were 151 deaths from hemorrhaging ulcer. Allen simultaneously pointed out that hemorrhage in the older patient was often fatal.

In general, hemorrhage has been defined as massive when the hematocrit value drops as low as 30 per cent and the erythrocyte count to three million or less. It is obviously massive if shock is associated. With the patient horizontal in bed, incipient shock and the degree of blood loss may not be appreciated but may be demonstrated by elevating the head of the patient—an observation referred to as the "tilt test" by Green and Metheny. An application of this test is seen when the patient with considerable acute blood loss into the intestinal tract may not appear shocked until taken to the x ray room and placed upright for fluoroscopy.

Treatment. A few working principles are applicable at the bedside. Hemorrhage must be stopped before hemoglobin hypoxia results in irreversible damage to such vital organs as the brain, kidneys, liver and heart. If bleeding cannot be controlled by medical measures, of which early and continuous blood transfusion is the most important, then control by surgical operation must be considered. During this trial period, the gastric acid must be continuously aspirated or buffered by frequent feedings. Gastric aspiration by tube not only serves to with-

draw the acid but may prove a guide to continued bleeding as well. Local application of thrombin preparations has not proved helpful. The older the patient the more arteriosclerotic and the more susceptible to hemoglobin hypoxia his vital organs may be and, therefore, the shorter the interval period before an irreversible state is reached.

Costello has shown that early and adequate blood replacement appreciably reduced the high mortality of an earlier period when transfusions were used less often. He also found that in some who were operated upon late for rapid deterioration, the bleeding vessel had in reality thrombosed and the critical picture was the result of irreversible shock rather than continued bleeding. The importance of immediate massive blood replacement for massive loss is apparent. It protects the patient from hemoglobin hypoxia and the measure of such replacement, as correlated with repeated blood counts, is the most dependable indication of continuing hemorrhage. If surgery then should become necessary for control the mortality will be less if hemoglobin hypoxia has been prevented during the evaluation period. The patient whose blood counts continue to fall in spite of generous blood replacement during the early hours of observation, particularly the patient in the older arteriosclerotic age group, must be seriously considered for emergency surgery.

Unfortunately there is no laboratory test which can be used as a guide to the cessation of hemorrhage. The best rule to follow is constant observation of the patient. If the pulse remains fast, the blood pressure lowered, the hematocrit value lowered and if the patient is perspiring despite continuous blood replacement, then common sense indicates that bleeding continues. The internist should evaluate the general risk of the patient and the surgeon should be in constant consultation until the emergency is controlled, either by medical measures or by surgical intervention. Patients over age fifty and particularly those with obvious arteriosclerosis, should be candidates for early emergency surgery. Age is not the sole factor in this decision. Other indications besides age and arteriosclerosis which lead one to individualize in favor of surgical intervention are (1) history of previous hemorrhage or perforation (2) the repeated vomiting of large quantities of blood, attesting the erosion of a large artery (3) the patient with a long ulcer history to suggest extensive scarring which

might interfere with clot retraction (4) the patient with proved gastric ulcer (5) the patient whose hemorrhage is associated with pyloric obstruction and (6) the patient who starts to bleed again within the hospital on a good regimen. Under these conditions, massive hemorrhage is less likely to stop.

Recurrence of bleeding after a temporary cessation is a bad omen and probably results from acid or mechanical dislodgment of a pin point clot. It should be remembered that it is difficult to revive a shocked patient twice. The consulting team by sustained observation should decide within the first twenty four hours or at the most forty eight hours, which policy is to be followed. Surgery after this golden period carries a proved higher mortality than an adequate medical regimen. It has been shown that often when an operation is performed late the vessel has thrombosed and the symptoms of persisting shock, which prompted late surgery were actually symptoms of cerebral hemoglobin hypoxia and late surgery only hastened death. If driven by the deteriorating state of the patient to consider surgery late in the course of massive bleeding, certainly one should first prove by continuous gastric suction that the patient is still bleeding.

Of practical importance in timing surgery is the local availability of the patient's type of blood. Should he prove to be one of the rarer Rh negative types the clinician will be less prone to temporize. To give other than the ideal type blood without removing the source of bleeding would be hazardous for the patient's future. The question of recently administered adrenocortical hormone should be explored both from the standpoint of its being a factor in the hemorrhage and for necessary substitution hormone therapy should surgery be performed.

In those patients in whom emergency surgical intervention is decided upon to control bleeding, the surgeon is in most instances committed to subtotal gastric resection as the only means of accomplishing control, even if the point of hemorrhage cannot be demonstrated. Because of this the surgeon should be certain of several points before sacrificing two-thirds or more of the stomach. These points should have been clearly established before operation: (1) that hemorrhage does arise from the upper gastrointestinal tract, (2) that it is not from esophageal varices, (3) that it is not the result of a blood dyscrasia and (4) that there is x ray evidence of an ulcer.

The first of these, that the bleeding is above the ligament of Treitz, can be determined, if not already proved by frank hematemesis by Levin tube intubation and aspiration of blood. Gastric ulcer and esophageal varices are usually associated with frank hematemesis whereas duodenal ulcer may be manifested only by melena. The second point, that the bleeding is not from esophageal varices can be determined by emergency fluoroscopy reinforced by a careful history and tests of liver function. Bleeding from this source is no less an emergency but the surgical approach is different. Emergency fluoroscopy in addition to assisting in ruling out varices will often show the deformity of an ulcer if not the ulcer crater and in the opinion of most authorities the test is not a greater hazard than a blind search by the surgeon at the time of laparotomy. It is no more tragic for the patient to die in the x ray room than for him to die in his hospital bed or the operating room. Adequate blood replacement is his best protection and blood can be administered in each of these places while the diagnosis is being established.

The more fortunate patients in whom an ulcer has been previously documented by x ray examination need not be subjected to the additional hazard of further x-ray examination. Blood dyscrasia can on occasion be detected by platelet count and bleeding, clotting and prothrombin time. Instances of severe gastrointestinal bleeding associated with thrombocytopenic purpura have been observed. These basic tests should be made at the initial hospital admission when the blood is drawn for the first crossmatching.

Anesthesia. When operation is decided upon, the anesthesiologist should join the team of consultants and help with the choice of the anesthetic agent. The patient with an ulcer which is bleeding slowly or intermittently and whose blood volume and blood pressure can be maintained easily by blood transfusions may be operated upon with use of either a regional block technique or general anesthesia. On the other hand, the anesthetic most often employed for the patient whose ulcer is bleeding profusely and whose blood volume and pressure cannot be maintained easily is general anesthesia with or without a muscle-relaxant drug. Regional techniques which dilate the vascular system in the area anesthetized and thereby cause a blood pressure fall are usually contraindicated in the patient who has lost a great deal of blood.

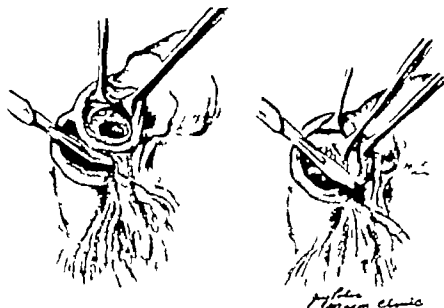


Figure 50. The technique of Alfred Strauss for resecting the posteriorly perforating duodenal ulcer. The anterior wall of the duodenum is divided distal to the pylorus, the posterior ulcer is visualized, the posterior wall of the duodenum is then transected distal to the ulcer and the proximal edge of the transected duodenum is retracted upward as the surgeon dissects the ulcer from the pancreas in a proximal direction (Strauss, A. A., Strauss, S. F., Schwartz, A. H., and Kram, D. D., J.A.M.A., vol. 140)

Regardless of the type of bleeding or the type of anesthetic technique employed, precautions must be instituted to assure means of administering fluids, blood and drugs and to prevent aspiration of stomach contents into the tracheobronchial tree and facilitate oxygenation of the patient.

The patient should be intubated with a cuffed endotracheal tube while he is awake and under local anesthesia. The endotracheal tube assures a means of oxygenating the patient while the inflated cuff on the tube prevents aspiration of blood or blood clots should they be regurgitated from the stomach.

At the end of the operation, the patient should remain in the operating room until (1) he is awake, (2) his respirations are judged to be adequate and (3) his mouth, pharynx and tracheobronchial tree have been cleansed by suction of mucus and/or regurgitated stomach contents.

At laparotomy the surgeon must remember that even when an ulcer of the stomach or duodenum is demonstrated, there may still be smaller erosions which may be the primary or an additional source of bleeding. To control the emergency, the ulcer should be resected. If it has penetrated through the duodenal wall into the pancreas, the duodenum must be separated from the pancreatic crater base.

A technical variation which may prove helpful in this trying situation is that of

Strauss (Fig. 50). In contradistinction to elective resection, a bleeding ulcer cannot be safely excluded into the duodenum. Transfixation of the end of an eroded artery leaving the ulcer crater exposed to the digestive juices, may result in delayed recurrent hemorrhage. Likewise, the posterior surface of the duodenum should be carefully explored for secondary points of bleeding and the gastric stump that is preserved should have its mucosal folds carefully visualized all the way into the cardioesophageal juncture. This is best accomplished with an endoscope. Unfortunately, a large number of patients with massive hemorrhage are obese individuals in whom this ideal is difficult to accomplish. Before the abdomen is closed search should be made for a coincident Meckel's diverticulum, which might harbor heterotopic gastric mucosa and give rise to secondary bleeding. Evidence of cirrhosis of the liver enlargement of the spleen or an engorged portal venous system suggesting portal hypertension should be searched for and these findings or their absence should be carefully recorded as a helpful reference should there be recurrent hemorrhage.

RECURRENT GASTROJEJUNAL ULCER

The development of gastrojejunal ulcer is evidence of failure in the individual case of the procedure applied. The incidence of recurrent ulcer has been reported as high as

34 per cent following gastroenterostomy and in the United States has averaged between 5 and 35 per cent. This led eventually to the abandonment of gastroenterostomy as a primary attack for ulcer. However the fact that no procedure today gives completely dependable control in all patients is attested by a continuing incidence of recurrent ulcer in the neighborhood of from 2 to 4 per cent following 70 per cent distal gastrectomy and approximately 5 to 10 per cent after vagotomy combined with gastroenterostomy. Recurrent ulceration, according to Thompson, is prone to manifest itself earlier after gastrectomy than after gastroenterostomy.

In the treatment of recurrent ulcer medical measures should be of course tried first. Recurrence may be due to a period of unusual emotional stress. Medical treatment, favored by the altered physiology already accomplished by surgery may suffice in some of these minor recrudescences. In general, however recurrence indicates an unusually severe ulcer diathesis and reoperation may become necessary. In reviewing 617 operations performed on 586 patients at the Mayo Clinic for gastrojejunal ulcer Walters, Chance and Berkson concluded that vagotomy can be expected to give excellent results in 70 per cent of those ulcers which follow adequate gastric resection. It carries a low operative risk and whether the trans-thoracic or transabdominal route is used makes little difference as far as the ultimate result is concerned, although they prefer the abdominal approach for more thorough exploration. When the recurrent ulcer follows simple gastroenterostomy they found gastric resection to be superior to vagotomy yielding excellent results in 86.5 per cent of 111 traced cases as compared to 77.8 per cent for vagotomy alone. Whereas the mortality was only 1 per cent following simple vagotomy in the postresected cases re-resection in these cases carried an operative mortality rate of 15 per cent. It is the belief of some surgeons however that re-resection should be done when the ulcer crater is found to be indurated. In such instances, healing would be slow and unsure despite the control of hypersecretion by vagotomy. When re-resection is necessary vagotomy should still be added and, according to Walters will not increase the mortality.

Before operation for recurrent ulcer as in all severely intractable cases the clinician and surgeon should consider the possibility of hyperfunctioning islet alpha cell tumor of the pancreas. Since Strom first described

this condition, Zollinger and Ellison have collected from their own and other experience forty-three cases in which islet cell tumors were related to peptic ulcer. Since these are non insulin producing islet cell tumors it is believed that the hyperglycemic glycogenolytic factor of the pancreas, glucagon may be the factor stimulating the gastric secretion to abnormal levels. Earle, Cahill and Hoar have published contrary observations on man and animals showing that glucagon administered over short intervals reduced blood pepsinogen levels as well as the milliequivalents of gastric acid secretion. This effect was apparently not dependent on intact vagi since vagotomized animals showed a similar inhibition. While evidence points to a potent pancreatic ulcerogenic factor of some form, this work tends to disprove glucagon as of any importance in peptic ulcerogenesis. Preoperative laboratory tests are not of help and only careful surgical exploration will discover this rare cause of intractable ulcer.

Likewise, at operation, in addition to careful exploration of all surfaces of the pancreas for such a tumor the surgeon should look for other causes of failure of the previous gastric resection, such as an overlooked portion of excluded gastric antrum (persisting hormone stimulation) an inadequate resection of the body of the stomach (too large a residual target) incomplete vagotomy in those cases in which the primary attack was based on removing the neurogenic phase of stimulation, and a long proximal jejunal loop (less concentrated alkaline protection of the anastomosis). If the primary attack was vagotomy the Hollander insulin test or the balloon motility test may be used as a guide to the completeness of the previous vagotomy before operation is performed for recurrent ulcer.

GASTROJEJUNOCOLIC FISTULA

This condition is seen with less and less frequency since simple gastroenterostomy has been abandoned. It occurs as a result of a marginal ulcer usually situated at the anastomosis or in the efferent loop just distal to the anastomosis penetrating through the wall into the overlying colon. With penetration through this barrier a fistula between the stomach or jejunum and colon is thereby established. This results in a fecal odor to the breath and rapid transit of food through the intestinal tract because of the irritation to the small bowel by the regurgitant feces. A marked loss of weight usually results. The

diagnosis is discovered more readily by colon x-ray than by x ray of the stomach. In advanced cases, the diagnosis is evident from history alone.

Correction of this condition involves the take-down of the fistula plus gastric resection to control the severe ulcer diathesis. After diagnosis, some of these patients may by blood transfusions and parenteral feedings, have some of the lost weight restored and be eligible with the aid of antibiotics for a one-stage resection of the fistula and subtotal gastrectomy.

Prior to present-day preoperative and postoperative adjuncts it was more often advisable to stage the procedure. This requires diverting the fecal stream proximal to the fistula, which is in the transverse colon and thereby preventing the feces from entering the stomach and jejunum and from traversing the small bowel. It can be accomplished in several ways. Pfeiffer and Kent were the first who recorded staging by performing a colostomy on the ascending colon. Baker independently accomplished the same with an ileostomy of the terminal ileum and Lahey avoided the exteriorized diversion by an anastomosis of the terminal ileum end to side with the descending colon distal to the fistula. Since this is a permanent anastomosis the entire colon proximal to the fistula is resected at the second-stage operation. In the procedures of Baker and Pfeiffer no bowel is sacrificed and the ileostomy or colostomy is closed at the second-stage operation when the fistula is resected and a subtotal gastrectomy done.

CHRONIC GASTRIC ULCER

Pathogenesis and Surgical Treatment. As contrasted with those in the duodenum, ulcers less often become chronic in the stomach, although the equal distribution of erosions in the duodenum and stomach on routine antispasmodics suggests that erosions often form in the stomach and heal without becoming chronic. The incidence of chronic gastric ulcer is but one-ninth that of chronic ulcer of the duodenum. Also it is of some differential significance that the chronic gastric ulcer by contrast with the duodenal ulcer is associated with little if any elevation in the normal acid secretion.

Dragstedt and his associates attribute gastric ulcer to an exaggeration of the hormonal gastric phase of secretion and the duodenal ulcer to an overactive and prolonged neurogenic phase. For this reason, they believe that vagotomy is not the preferred operation

for gastric ulcer and it should not be substituted for distal gastrectomy. It is established that distal gastric resection for gastric ulcer is seldom complicated by recurrent ulcer even though the resection is not more than 50 per cent. For gastric ulcer therefore, many authorities substitute a partial 60 per cent distal resection for the usually higher resection (75 per cent) used in duodenal ulcer. Whereas in duodenal ulcer there is no concern for malignancy, this is a major consideration in gastric ulcer and one of the cardinal requirements in the surgical treatment, therefore is the removal of the ulcer for histologic study.

To protect against recurrent peptic ulcer the other requirement is removal of the entire antrum and a portion of the target body. Since more of the stomach is preserved in resection for gastric peptic ulcer and since the uninvolved duodenum lends itself well to anastomosis gastroduodenostomy (Billroth I) is the customary method of restoring continuity. The mortality following gastric resection for gastric ulcer is lower (less than 1 per cent) than for duodenal ulcer since the resection carries through normal unscarred duodenum which can be more safely closed or anastomosed. Resection gives more dependable control of the ulcer diathesis than is true when applied for duodenal ulcer and, possibly, since a lesser resection suffices and an end-to-end gastroduodenostomy is more easily applicable untoward sequelae may at least theoretically be reduced.

One word of caution should be sounded in reducing too generously the resection for gastric ulcer. The gastric ulcer may be associated with or be secondary to duodenal ulcer. In such an instance the more extensive resection done for duodenal ulcer should apply. Johnson finds gastric ulcers or their scars associated in some 10 per cent of patients subjected to gastrectomy for duodenal ulcer. These patients have the hypersecreting duodenal type rather than the hyposecreting hypomotile gastric ulcer. In two-thirds of these, there was pyloric stenosis and gastric retention and he suggested that this retention of acid is an etiologic factor in the development of such associated gastric ulcer. Because of the fact that the two may be associated, plus the fact that recurrence after gastric resection for gastric ulcer is occasionally seen, the surgeon should exercise caution in reducing the amount of resected stomach below 60 per cent. If a long-standing history of ulcer and previous roentgenologic evidence of duodenal deformity sug-

gest previous coincidental duodenal ulcer then the resection should be less conservative particularly if preoperative testing shows the gastric secretion is excessive.

In general, the technical problems are not as great in resection for gastric ulcer and the mortality is less than after resection for duodenal ulcer. The exception to this is the penetrating ulcer high on the lesser curvature close to the esophageal juncture. This situation has prompted some surgeons to resort in these instances to gastroenterostomy and vagotomy, an attack which may be as Dragstedt believes, without physiologic support. Others, for the same technical reasons have done a distal gastric resection leaving the high ulcer. Both of these methods fail to rule out malignancy. Certainly total gastrectomy is not justified in such instances without histologic proof by frozen section of malignancy. I have not encountered an ulcer that, after adequate mobilization of the entire stomach, could not be resected in the Hofmeister manner leaving some stomach. In these instances the Hofmeister clamp if placed above the high ulcer might encroach upon the esophageal juncture and for this reason the use of the clamp is omitted (Fig. 51 a and b). Instead, the stomach is freed entirely on the greater and lesser curvatures the field is isolated by packs, a clamp is put part way across the greater curvature side marking the level of anastomosis the greater curve side of the stomach is transected distal to the clamp and then a freehand vertical resection of the lesser curvature, including the ulcer is accomplished. The open margins of the lesser curvature are then closed under direct vision in layers so as not to encroach upon the opening of the esophagus. The greater curvature side, which has been held by the clamp is then anastomosed in the usual manner.

Indications for Operation for Gastric Ulcer. Whereas for duodenal peptic ulcer surgery is advised only for the 15 per cent who suffer from the complications of chronic recurrence, by contrast in gastric ulcer the incidence of cancer which is mistaken for peptic ulcer leads the clinician to advise surgery in from 30 to 75 per cent. It is generally agreed that the gastric ulcer deserves surgical consideration as long as it remains unhealed. The all important question is whether or not all gastric ulcers should be promptly subjected to surgery. Reference here, of course, is to those ulcers which cannot be diagnosed by x ray or other measures as being frankly malignant.

In favor of such a policy would be the argument that the issue would thus be rapidly and, from the standpoint of cancer more safely, settled. Against this argument is the fact that gastric resection is not without mortality and morbidity and, further, more medical treatment does prove effective in healing probably 50 per cent of these ulcers. Precipitate surgery would result in some instances in gastric resection for acute erosions which are not actually chronic ulcer and which do not justify sacrifice of the stomach. Certainly as in duodenal ulcer a medical trial is worth while in a considerable number of previously untreated gastric peptic ulcers. Marshall found that in 411 gastric ulcers without gross evidence of cancer 37 per cent came to surgery and 15 per cent proved to be malignant. It is remarkable that 50 per cent of the patients who proved to have malignant ulcer gave a history of ulcer distress for five or more years. From this study Marshall concluded that probably 50 per cent of gastric ulcer patients deserved immediate surgery. In 161 cases of gastric ulcer without gross evidence of cancer which Jones Clements and Pearson reviewed, 55 per cent were operated upon and of those 9 per cent proved to be malignant. Of the seventy-three patients (45 per cent) who were treated medically twenty-five had recurrence of their ulcer and five subsequently came to surgery. One of these proved to be malignant. Forty healed and remained well but required follow up x ray examination for insurance against recurrence.

Preoperative differential diagnostic methods for malignancy should include, of course a careful history and physical examination to discover distant metastasis such as Virchow's lymphatic metastasis in the neck, a nodular liver or a cul-de-sac implant presenting as a rectal "shelf." The absence of free gastric acid secretion on repeated anals is strongly in favor of malignancy although the presence of free acid does not rule out malignancy. The roentgenologist can catalogue the definitely malignant and a large portion of the definitely benign, but in the borderline group between these two extremes he will not give assurance.

In most centers histologic evidence of cancer is found in 10 per cent or more of ulcers which have preoperatively been considered benign. Radiologic signs favoring malignancy are the meniscus resulting from the rolled border an ulcer greater in diameter than 2 cm., particularly when it is with-

out proportionate penetration and the ulcer's situation on the greater curvature or prepyloric region rather than on the magenstrasse. Cytologic studies on the gastric aspirate may if the findings are positive

prevent delay in surgery. As far as the threat of malignancy in the gastric ulcer is concerned in borderline cases the majority of internists and surgeons insist on from ten to fourteen days of rigid medical control,

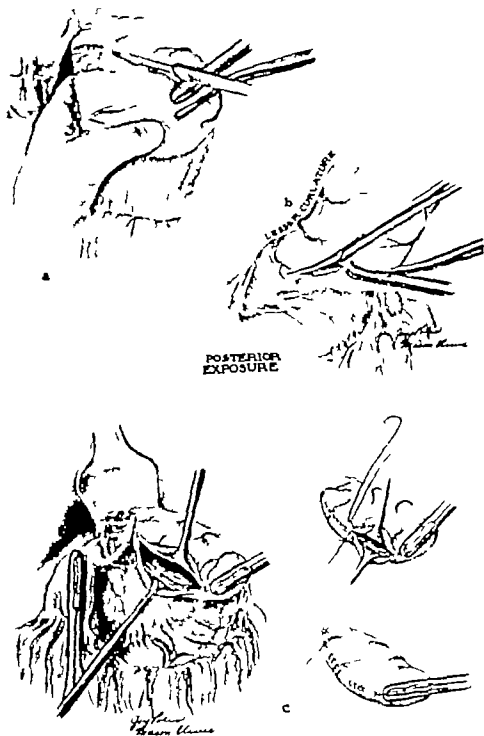


Figure 51 A method of resecting the ulcer high on the lesser curvature of the stomach. After the stomach is freed and the duodenum transected, the level of resection and anastomosis is marked on the greater curvature by a Payr clamp. Then, without the aid of vertical clamps which might encroach upon the esophagus, the ulcer and lesser curvature are removed by sharp dissection (a) anterior view (left) and (b) posterior view (right) c, This vertical lesser curvature incision is then closed with two rows of sutures under direct vision and the greater curvature stump is anastomosed to either the duodenum or jejunum.

with bed rest, hourly feedings and antacids. If 50 per cent healing cannot be demonstrated, the patient should have surgery. The majority of these ulcers will not prove to be malignant but nonetheless they represent a complicated peptic ulcer which should have the benefit of surgery before such complications as penetration and hemorrhage increase the hazard.

Patients with gastric ulcer are in general, poorer surgical risks than patients with duodenal ulcer. They average ten years older than patients with duodenal ulcer and will have a proportionate increase in associated arteriosclerosis and other constitutional diseases. Those ulcers which do show satisfactory partial healing in two weeks should be proved to have completely healed in six weeks and should be x rayed several times in the first year thereafter to prove that they remain healed. If the ulcer should recur surgery is preferable to further confusing delay.

Even at operation differentiation between cancer and chronic peptic ulcer may still be difficult in these borderline cases. The ulcer in the distal stomach presents a lesser problem in this regard than does the ulcer situated high in the stomach because the resections for benign and malignant ulcer in the prepyloric area are essentially the same except that in cancer the greater omentum is included in the resection and an attempt is made to remove more duodenum and to include the subpyloric and regional lymphatics.

On the other hand, for the higher ulcer near the esophageal juncture resection is done without clamps (Fig. 31c). If frozen section proves the ulcer to be cancer a total resection of the remaining stomach is justified to insure a greater margin against local recurrence, unless lymphatic metastasis is also discovered. In the latter case the chance for control may not be adjudged sufficient to justify the additional mortality and morbidity of total gastrectomy.

The radical cancer operation is not justified in borderline cases without first having histologic proof of cancer. It should also be remembered that large gastric ulcers penetrating the pancreas or lesser curvature are more often benign. They should be stripped off the pancreas without injury to that organ.

ESOPHAGITIS AND ESOPHAGEAL PEPTIC ULCER

Peptic ulcer of the esophagus occurs with extreme rarity as compared with either gas-

tric or duodenal ulcer for the simple reason that the esophagus is not exposed to the gastric acid secretions except under unusual situations. One of these occurs by regurgitation when the esophageal sphincter mechanism is deficient, particularly as in sliding hiatal hernia. The second, and much rarer situation is when heterotopic gastric secretory mucosa occurs above the cardiac sphincter.

The esophagus is more vulnerable to peptic erosion than is the rest of the gastrointestinal tract. It is protected physiologically against acid reflux by a sleeve of alkaline secreting cardiac glands and mechanically by the angulation of the entrance of the esophagus and by the sphincter action of the cardioesophageal junction. Transient esophagitis or erosions may follow reflux from repeated episodes of vomiting or occur when the sphincter is made incompetent by esophageal varices and by the prolonged use of indwelling nasogastric tubes. Esophagitis from prolonged exposure to acid peptic secretion may become symptomatic with or without erosion. The erosions from reflux are more often linear and stenosis may eventually result. The circular punched-out crater similar to that seen in chronic peptic erosion of the duodenum is found more often when the terminal esophagus is lined by columnar epithelium similar to that in the stomach. As in the duodenum such an ulcer may bleed or it may by repeated recurrence and healing, lead to stenosis and obstruction. At this stage it may be difficult to determine whether the stenosis is the result of regurgitant esophagitis alone or recurring peptic ulceration. It might even be confused by the clinician with the obstructive achalasia referred to as cardioesophagus, although the roentgenologic characteristics of this entity including the greater esophageal dilatation, serve to differentiate it.

Surgical treatment may require (1) simple repair of a sliding hiatal hernia to eliminate reflux, (2) an operation to control hyperacid secretion, (3) bougienage for or the resection of, a stenotic terminal esophagus, (4) the interposition of a loop of jejunum or colon to replace an incompetent sphincter or (5) a combination of one or more of these procedures.

For symptomatic esophagitis associated with sliding hiatal hernia but without stenosis or associated duodenal or gastric ulcer the correction of the reflux may be all that is required. Medical treatment, including weight reduction, antacids and elevation of

the head of the bed, should relieve the injury. In about 30 per cent surgical correction of the hiatal hernia will be justified. If the disorder is intractable to adequate medical treatment, surgery should not be indefinitely postponed because the complications of stenosis and hemorrhage in the elderly patient are graver problems. When esophagitis is associated with a hiatal hernia and in addition there is a duodenal or gastric ulcer repair of the hernia should be combined with one of the operations previously described to drain the stomach and control the hypersecretion of acid.

Even after obstruction of the terminal esophagus has complicated the picture, Wengenstein and Levin found that gastric resection relieved several patients previously treated by dilatations. Barrett has described a chronic peptic ulcer in the terminal esophagus which is encompassed by columnar rather than the usual squamous epithelium. Whether the ulcer was incited at the beginning by congenital ectopia of gastric mucosa in the terminal esophagus above the cardiac sphincter mechanism or whether the columnar epithelium is heterotopic resulting from repeated healing of ulceration secondary to acid regurgitation, is by then of no importance. Surgical attack for such a cardioesophageal ulcer requires resection of the involved terminal esophagus, plus an attack designed to reduce acid secretion. An additional procedure is necessary to protect the terminal esophagus, which will thereafter by virtue of the loss of the cardiac sphincter be more exposed than before.

Ellis has advocated a sleeve resection of the involved terminal esophagus with reanastomosis of the terminal esophagus to the cardia of the stomach. This is then supplemented by distal resection of the antrum of the stomach, vagotomy and Billroth I gastroduodenostomy. Since 1955, Ellis has applied this procedure in twenty-seven patients. There was one operative death and one patient has required repeated dilatations. The others have had satisfactory results, fifteen excellent, eight good (occasional nausea and vomiting) and three are too recent to evaluate. Twenty four of these patients had developed their esophageal lesion secondary to sliding hiatal hernia. Three had undergone previous cardioplasties of various types for cardiospasm.

When the esophageal stricture is not associated with duodenal ulcer or deformity after resecting the stenotic or incompetent cardioesophageal juncture Merendino preserves the stomach and protects the esoph-

agus by the interposition of a jejunal segment between the esophagus and the stomach, combined with bilateral vagotomy and Finney pyloroplasty. He and others have shown that once the sphincter mechanism is lost and the esophagus is then anastomosed to the stomach, the esophagus becomes vulnerable to acid regurgitation and ulcer and hemorrhage are frequent sequelae. They find the interposed jejunal loop adequately substitutes for this sphincter mechanism and they prefer this to attempts to reduce acid secretion. In the thirty-three operations performed, there have been four operative deaths a mortality of 12.1 per cent. The patients varied in age from ten to seventy five years and the series included not only patients with peptic esophagitis and stricture but also patients with cardiospasm and esophageal varices (extrahepatic type) and lye stricture. Of the twenty patients with peptic esophagitis and stricture, all are satisfied with the result. They report no failure to cure the esophagitis, no recurrent ulceration and no new ulceration of the upper gastrointestinal tract. The operation is prolonged, difficult and the hazard of an insufficient blood supply to the transposed jejunum can be a major consideration.

UNTOWARD RESULTS OF SURGERY

Mortality Except in the case of uncontrolled hemorrhage, unrelieved obstruction or perforation, peptic ulcer in itself is not a fatal disease. Therefore, any surgical procedure applied should be critically appraised on the basis of mortality.

Operative mortality stems, for the most part, either from technical imperfections which result in leaking gastrointestinal anastomoses or leaking duodenal stumps, from peritonitis and from pulmonary complications or from cardiovascular accidents. In some patients with massive hemorrhage, exsanguination and hypoxia may be responsible for death. Pulmonary complications, a frequent cause of death twenty five years ago have been appreciably reduced by use of antibiotics, preoperative suction of the stomach to prevent pulmonary aspiration and postoperative tracheal suction. Cardiovascular accidents have been reduced by improved anesthesia, anticoagulants, blood replacement and pharmacologic support of blood pressure. These will continue to be responsible for an irreducible minimum of mortality whereas technical imperfections by good judgment and selection of surgical methods may be individually improved.

The mortality rate reported by specialists

in this field varies from less than 1 to as high as 3 per cent following gastric resection for ulcer and from 0.5 per cent to 1.5 per cent after vagotomy. The mortality is understandably higher in charity hospital wards where surgery is more often done for uncontrolled hemorrhage or perforation than for intractability. Furthermore patients in charity wards are usually poorer surgical risks than are private patients.

Possibly the more representative figures on mortality are those collected by the Ohio Chapter of the American College of Surgeons from twenty-nine hospitals in the state of Ohio and cited by Crile. The state wide mortality for elective gastric resection was 4.9 per cent, whereas the mortality for operations exclusive of resection (mostly vagotomy with a gastric drainage procedure) was 1.7 per cent. These figures did not include deaths from emergency operations for bleeding or perforation. On the basis of these figures, Crile suggests that the over all mortality would be much less if vagotomy and gastroenterostomy (or pyloroplasty) were done primarily and gastric resection reserved for cases of recurrent ulcer. The surgeon whose mortality is high with gastric resection should consider this advice. Certainly resection of the larger posterior penetrating ulcer should be individualized. In these cases, an appreciable reduction in the mortality of gastric resection can be accomplished by excluding the difficult ulcer into the closed duodenal stump (Fig. 48A).

In a comparison by Baker of subtotal gastrectomy with resection of the ulcer and of selective exclusion of the difficult posterior penetrating ulcer into the duodenal stump mortality was found to be reduced from 1.9 per cent to 0.8 per cent by employing the exclusion method in the more difficult cases. It is noteworthy that of the three deaths in the 156 resected ulcers, two were from leaking stomps which should not have occurred with gastroenterostomy. On the other hand, there were no deaths due to technique in the 122 cases of difficult ulcer treated by exclusion, the single death being that of an eighty year old man operated upon on the fifth day of a massive hemorrhage and dying of cerebral hypoxia. By application of this selective principle of exclusion, the last 140 gastric resections performed since completion of the previous study have been accomplished with only one death, an elderly patient operated upon in an emergency for exsanguination. It should be noted that the exclusion method was not followed by a

higher incidence of recurrent ulcer there being one proved and three suspected recurrences in each series. In the difficult situation the surgeon should apply a method which has proved safest in his hands, even though it is associated with a higher rate of recurrent ulcer and even though in these few cases secondary operations be required.

The reported mortality of resection for gastric ulcer is approximately one-half that for duodenal ulcer mainly because the normal duodenum presents no technical problems either in closure or anastomosis.

Incidence of Recurrent Ulcer The next most serious complication to fatality is failure of the surgical procedure to control permanently the ulcer diathesis. Gastroenterostomy alone has been followed by gastrojejunal ulcer in up to 33 per cent, gastroenterostomy protected by vagotomy has been followed by gastrojejunal ulcer in from 5 to 10 per cent. Gastric resection has been followed by recurrent ulcer in from 2 to 4 per cent, the higher recurrence rate following the more conservative resection. A true 75 per cent gastric resection has a minimal recurrence rate, being less than 1 per cent in most reported series. The recurrence rate after antrectomy and vagotomy is less than 1 per cent. The recurrent ulcer is rare after 65 per cent gastric resection for gastric ulcer.

Dumping Syndrome. This term has been used to describe the tachycardia, palpitation, faintness, perspiration and sense of warmth or flushing some patients who have undergone gastric surgery experience shortly after meals. It more often follows ingestion of sweet liquids such as milk shakes and syrups although it may follow any food. It probably should be differentiated from the "small stomach syndrome," in which the patient for the first few weeks after operation feels uncomfortably full after eating.

The incidence of "dumping" varies with different authors ranging from 5 to 100 per cent. It probably is present, if searched for, in a minimum of from 15 to 20 per cent of patients for the first few weeks or months after gastric resection, and in from 1 to 5 per cent it may persist to a disabling degree. Its incidence will depend in part upon the individual's psyche (the migrainous patient, the fastidious eater the introvert) and will depend more upon the extent of resection than upon the type of anastomosis employed. The extensive 75 to 80 per cent resection has been followed by a greater incidence of dumping than has the 60 per cent resection however individual susceptibility remains

an important factor and each patient should be carefully screened preoperatively with this in mind.

The actual cause of dumping remains obscure. Distention of the stomach or jejunum the pull of the full stomach and jejunum on unsupported mesenteries too rapid emptying of the stomach and reflux pooling of the blood in the splanchnic bed with resultant systemic hypovolemia have all been suggested as mechanisms. One of the more attractive recent theories is that of Roberts, Randall and Farr. They postulate that the osmotic pull of the jejunal contents after a carbohydrate meal produces fluid compartment and electrolyte shifts which deplete the circulating blood volume and thus creates this picture of mild shock. Certainly the clinical syndrome is one closely simulating syncope or shock.

Many methods of treatment have been recommended. Probably the most effective is to reassure the patient that the complication is neither serious nor permanent, to see that he carefully avoids taking liquids and/or sweet foods on an empty stomach and instead starts the meal with dry toast, poached eggs and other proteins. The majority of patients are relieved shortly after lying down. Reassurance seems the best treatment and time the best cure.

Nutrition. Failure of the patient to maintain his preoperative weight occurs in about one-fourth of patients after subtotal gastrectomy. A follow up which was made of 78 patients showed 55 per cent to maintain their normal weight, 20 per cent to become overweight and 25 per cent to be under their preoperative weight. Why some patients should lose weight is not completely understood, but there are at least two responsible factors. First the patient does not eat enough, either because of early satiety from the reduced gastric capacity or because of symptoms of dumping when larger meals are eaten. As a result, the caloric intake is frequently at such a level that weight cannot be gained or even maintained. This is probably the most common cause of weight loss in these patients. Second, assimilation studies have revealed that the absorption of both fats and proteins is reduced after gastric resection and even after simple vagotomy. This is probably of significance in only 10 to 15 per cent of these patients. The physiologic basis for malabsorption is probably inadequate stimulation of the biliary and pancreatic secretions as well as inadequate admixture of the food with these secretions.

Both presumably result from the rapid transit of food through the stomach and intestine with limited contact of the food with the intestinal mucosa. The reduced transit time is explained in part by the reduced size of the stomach and the fact its outlet is no longer guarded by the pyloric sphincter. If the duodenum is bypassed, as in gastrojejunostomy the loss of assimilation should be even greater than after gastroduodenostomy. In fact, studies confirm that the fecal fat and nitrogen loss is actually less after the Billroth I gastroduodenostomy than after the Billroth II gastrojejunostomy. Dietary supplements of pancreatic enzymes and bile salts are usually ineffective. The treatment, therefore is purely dietary and consists of careful attention toward providing sufficient calories in the form of frequent meals of those foods which do not elicit dumping. Anticholinergic drugs may be of help in reducing the rapid transit of food through the intestine.

READING REFERENCES

- Baker J. W. Ileostomy Preliminary to Resection of Gastrojejunocolic Fistula. *Northwest Med.* 39:398-403, 1910.
- Baker J. W., Boyd, R. S., and Foster R. A. Gastric Resection with Exclusion of a Complicated Duodenal Ulcer: Analysis of 122 Cases. *Ann. Surg.* 142:519-531, 1935.
- Barrett, N. R. Chronic Peptic Ulcer of the Oesophagus and Oesophagitis. *Brit. J. Surg.* 33:175-182, 1950.
- Beaumont, W. Experiments and Observations on the Gastric Juice and the Physiology of Digestion. Plattsburgh, F. P. Allen, 1833.
- Berg, A. A. The Radical Surgical Cure of Gastric and Duodenal Ulcer. *S. Clin. North America* 5:49-91, 1910.
- Blackford, J. M., Smith, A. L., and Affleck, D. H. Peptic Ulcer Emergencies. Study of Massive Hemorrhages and Acute Perforations Treated during Diagnosis of 916 Private Cases Suffering from Peptic Ulcer. *Am. J. Digest. Dis.* 4:646-650, 1937.
- Brackney, E. L., Thal, A. P., and Wangenstein, O. H. Role of Duodenum in the Control of Gastric Secretion. *Proc. Soc. Exper. Biol. & Med.* 88:302-306, 1955.
- Code, C. F. and Varen, R. L. Chronic Histamine Action. *Proc. Soc. Exper. Biol. & Med.* 44:475-477, 1940.
- Coffey R. J. and Lazaro, E. J. Vagotomy Hemigastrectomy and Gastroduodenostomy (Finney on Haberer) in the Treatment of Duodenal Ulcer. *Ann. Surg.* 141:862-869, 1955.
- Cope, O., and Wight, A. Metabolic Derangements Imperiling the Perforated Ulcer Patient. *The Plan of Therapy Arch. Surg.* 72:571-582, 1956.
- Crisle G. Jr. Choice of Operations for Duodenal Ulcer. *Postgrad. Med.* 14:454-455, 1953.
- Cushing, H. Peptic Ulcers and the Interbrain. (Bal-

- four Lecture) Surg Gynec and Obst 33 1-34, 1932.
- Davis, H. A. Wetzel, H., and Davis, L. Acute Upper Alimentary Tract Ulceration and Hemorrhage Following Neurosurgical Operations Surg Gynec & Obst 100 51-58 1955.
- Dragstedt, L. R. Pathogenesis of Gastroduodenal Ulcer Arch. Surg. 44 438-451 1942.
- Dragstedt, L. R., Oberhelman, H. A., Jr., Evans, S. O., and Hugler, S. P. Antrum Hyperfunction and Gastric Ulcer Ann Surg. 140 306-404 1954.
- Dragstedt, L. R. and Owens, F. M. Jr. Supradiaphragmatic Section of Vagus Nerves in Treatment of Duodenal Ulcer Proc Soc Exper Biol & Med. 53 152-154 1943.
- Dragstedt, L. R., and others. Quantitative Studies on the Mechanism of Gastric Secretion in Health and Disease. Ann. Surg. 133 626-640 1950.
- Earle, A. S., Cahill, C. P. Jr. and Haur, C. S. Jr. Studies on the Relationship of Cholecystin (HGP) to Blood Pepsinogen Concentrations Ann. Surg. 146 124-130 1957.
- Edwards, L. W., and others. Duodenal Ulcer Treatment by Vagotomy and Removal of Gastric Antrum Ann. Surg. 145 738-752, 1957.
- Ellis, F. H. Jr. Andersen, H. A., and Clagett, O. T. Surgical Management of the Complications of Reflux Esophagitis Arch Surg 73 578-589 1956.
- Elman, R. and Hartmann, A. F. Spontaneous Peptic Ulcers of Duodenum after Continued Loss of Total Pancreatic Juice Arch Surg. 23 1030-1040 1931.
- Eyama, S. O. Jr., and others. Stimulating Effect of Vagotomy on Gastric Secretion in Hendenham Pouch Dogs. Am. J. Physiol. 174 219-225 1953.
- Eyerson, T. C. Experimental Comparison of Protein and Fat Assimilation after Billroth II Billroth I, and Segmental Types of Subtotal Gastrectomy Surgery 36 525-537 1954.
- Farmer, D. A., and Southwick, R. H. Hemigastrectomy Combined with Resection of the Vagus Nerves. New England J Med 247 1017-1022, 1952.
- Farris, J. M. and Smith, C. K. An Evaluation of Temporary Gastrotomy—A Substitute for Nesogastric Suction. Ann. Surg. 144 475-486 1956.
- Gallagher, I. C., Moor, P. J. and Wrigley, J. H. The Billroth-I and Polya Operations for Duodenal Ulcer a Comparison. Lancet 1 220-222, 1956.
- Green, D. M. and Mothmy, D. Estimation of Acute Blood Loss by the Tilt Test. Surg. Gynec. & Obst. 84 1045-1050, 1947.
- Harkins, H. N., and Hooker, D. H. Vagotomy for Peptic Ulcer. Experimental and Clinical Studies. Surgery 22 239-245, 1947.
- Harkins, H. N. and others. The Billroth I Gastric Resection. Experimental Studies and Clinical Observations on 291 Cases. Ann. Surg. 140 405-427 1954.
- Harrison, R. C., Leley, W. H., and Hyde, H. A. The Production of an Acid Inhibitor by the Gastric Antrum. Ann. Surg. 144 441-449 1956.
- Jordan, P. H., Jr., and Sand, B. F. A Study of the Gastric Antrum as an Inhibitor of Gastric Juice Production. Surgery 42 40-49 1957.
- Jordan, S. M., Chairman. Report of Committee on Surgical Procedures of National Committee on Peptic Ulcer of American Gastroenterological Association on Study of Vagotomy Study of Gastric Resection, Comparative Study of Vagotomy and Gastric Resection. Gastroenterology 22 297-469, 1952.
- Kay, A. W. Effect of Large Doses of Histamine on Gastric Secretion of Hydrochlorides Assessed Histamine Test. Brit. J. Med. 2 77-80 1953.
- Kiribik, L. B. and Merendino, K. A. Experimental Study of Buffering Capacity of Contents of Upper Small Bowel. Surgery 35 532-537 1954.
- Kinikuk, L. B. and Merendino, K. A. The Comparative Sensitivity of the Mucosa of the Various Segments of the Alimentary Tract in the Dog to Acid-Peptic Action. Surgery 35 547-566, 1954.
- Lillichi, C. W., Lewis, F. J., and Wangenstein, O. H. The Limitations of a Gastric Drainage Operation upon the Effectiveness of Vagotomy. Gastroenterology 15 467-497 1950.
- MacLean, L. D. and others. Nutrition Following Subtotal Gastrectomy of Four Types (Billroth I and II, Segmental, and Tubular Resections) Surgery 35 703-718, 1954.
- Mann, F. C., and Williamson, C. S. The Experimental Production of Peptic Ulcer Ann. Surg. 77 409-423, 1923.
- Marshall, S. F. The Relation of Gastric Ulcer to Carcinoma of the Stomach. Ann. Surg. 137 691-903 1953.
- McKistrick, L. S., Moore, F. D., and Warren, A. Complications and Mortality in Subtotal Gastrectomy for Duodenal Ulcer. Report on a Two-Stage Procedure. Ann. Surg. 120 531-561 1944.
- Merendino, K. A., and Dillard, D. H. The Concept of Sphincter Substitution by an Interposed Jejunal Segment for Anatomic and Physiologic Abnormalities at the Esophagogastric Junction, with Special Reference to Reflux Esophagitis, Cardiospasm and Esophageal Varices. Ann. Surg. 142 486-508, 1955.
- Moore, H. G. Jr. and Harkins, H. N. A Critical Evaluation of the Billroth I Gastric Resection. Surgery 32 408-423, 1952.
- Oberhelman, H. A., Jr., Woodward, E. R., Zahara, J. M., and Dragstedt, L. R. Physiology of Gastric Antrum. Am. J. Physiol. 160 768-748, 1952.
- Ogilvie, H. The Problem of Peptic Ulceration. Lancet 1 555-560 1953.
- Pavlov, I. P. The Work of the Digestive Glands, 2nd ed. London, Charles Griffin & Co., 1902. Translated by W. H. Thompson, 1910.
- Prestley, J. T. Reflections on Surgical Treatment of Duodenal Ulcer Arch. Surg. 69 455-463, 1954.
- Prohaska, J. V., Govatsis, M. S., and Klinefelter, A. Mechanism of the Efferent Stomach Dysfunction Following Subtotal Gastrectomy Arch. Surg. 63 491-499 1954.
- Saenger, L. R., and others. The Relation between the Physiologic Stimulatory Mechanisms of Gastric Secretion and the Incidence of Peptic Ulceration: An Experimental Study Employing a New Preparation. Surg. Gynec. & Obst. 93 127-142, 1953.
- Sealey, S. F., and Carey, D. Nonoperative Treatment of Perforated Peptic Ulcer: A Further Report. Surg. Gynec. & Obst. 102 435-446 1956.
- Storer, E. A., and others. Gastric Secretion in Hendenham Pouches Following Section of Vagus Nerves to Main Stomach. Proc. Soc. Exper Biol. & Med. 80 325-327 1953.
- Strass, A. A., and others. Results of Subtotal Gas-

- trectomy for Gastric and Duodenal Ulcers since 1917. J.A.M.A. 149 1093-1101 1932.
- Waddell, W. R., and Bartlett, M. K.: Antral Exclusion with Vagotomy for Duodenal Ulcer: Acid Secretory Studies on 50 Patients. Ann. Surg. 140: 3-11 1937.
- Walters, W., Chance, D. P., and Berkson, J.: A Comparison of Vagotomy and Gastric Resection for Gastrojejunal Ulceration. A Follow-up Study of 301 Cases. Surg. Gynec. & Obst. 100 1-10, 1935.
- Walters, W., Chance, D. P., and Berkson, J.: The Surgical Treatment of Gastrojejunal Ulceration. Arch. Surg. 70 526-532, 1935.
- Wangenstein, O. H., and Levin, N. L.: Gastric Resection for Esophagitis and Stricture of Acid Peptic Origin. Surg. Gynec. & Obst. 69 560-570 1919.

- Welch G. E., and Rodkey G. V.: A Method of Management of the Duodenal Stump after Gastrectomy. Surg. Gynec. & Obst. 98 370-379 1931.
- Wolf S. and Wolf H.: Human Gastric Function. An Experimental Study of Man and His Stomach. London, Oxford University Press 1913.
- Zollinger R. M., and Ellison, E. H.: Nutrition after Gastric Operations. J.A.M.A. 151 811-814 1954.
- Zollinger R. M., and Ellison, E. H.: Primary Peptic Ulcerations of the Jejunum Associated with Islet Cell Tumors of the Pancreas. Ann. Surg. 142 709-728 1955.
- Zubiran, J. M., and others: Peptic Ulcer and the Adrenal Stress Syndrome. Arch. Surg. 65 809-815 1932.

Tumors of the Stomach

By SAMUEL F. MARSHALL, M.D.

SAMUEL FREDERICK MARSHALL was educated at Johns Hopkins University and received his surgical training under one of the early Hopkins surgical residents who had become the Chief Surgeon of the Henry Ford Hospital. He joined the Lacey Clinic Staff in 1935 and his surgical skill and judgment have added to the reputation of that organization.

Tumors of the stomach consist of benign tumors and malignant tumors but by far the larger number of gastric neoplasms comprise the carcinomas or gastric cancers. Malignant gastric tumors, moreover, may be carcinomatous or sarcomatous—the sarcomas accounting for approximately 3 per cent of all types of gastric neoplasms.

BENIGN TUMORS

The incidence and types of tumors, as ascertained by microscopic study of 1700 tumors consecutively removed by operation are shown in Table I. It will be noted that benign tumors account for 4.8 per cent of the total number of tumors found at the operating table but the number of different benign tumors which occur in the stomach is small. It is probably true that they occur more frequently than noted at operation because they rarely give symptoms and thus are recognized less frequently than the malignant tumors which sooner or later always produce digestive distress.

One of the commonest benign gastric tumors is the simple adenoma of the stomach

which may occur as a single tumor or more rarely as a multiple polypoid form of tumor. Ewing pointed out that rarely these tumors are submucous in origin. The tumors ordinarily remain small and seldom give symptoms unless situated near the pyloric orifice where, when pedunculated, they may prolapse through the pylorus and cause obstructive symptoms. The common site of origin

Table I Classification and Incidence of 1700 Gastric Tumors

	NO. OF CASES	PER CENT
Carcinoma	1567	92.2
Sarcoma	51	3.0
Lymphoid tumors	35	
Leiomyosarcoma	16	
Benign tumors	82	4.8
Leiomyoma	28	
Single polyps	31	
Multiple polyps	6	
Lipoma	2	
Miscellaneous	3	
Aberrant pancreas	12	
Total	1700	

is in the antral area. They are frequently accompanied by achlorhydria and minor grades of anemia.

Polyps may be multiple and produce diffuse gastric polyposis. They may be either neoplastic or inflammatory but in most cases they are adenomatous. Multiple polyps, which occur as a result of inflammation and are related to advanced hypertrophic gastritis produce diffuse gastric polyposis. Multiple discrete polyps may be scattered over the entire mucosal surface but more often are localized in the lower third of the stomach (Fig. 52). We have seen instances in which they involved the entire stomach and on several occasions have found malignant degeneration occurring in multiple polyps which are true adenomatous polyps in character. Other benign tumors encountered are the fibromas, fibroadenomas, lipomas, hemangiomas and occasionally a neurofibroma, but these are extremely uncommon. Feldman has stated that 2.5 per cent of gastric tumors are benign tumors and Stewart observed seventy-eight benign tumors of the stomach in 11,000 post-mortem subjects (0.7 per cent). There is considerable difference of opinion in the literature as to whether adenomatous polyps eventually become malignant. It appears, however, that while gastric polyps do not have the same tendency to degenerate into cancer as do polyps of the colon and rectum, malignant degeneration does undoubtedly take place and is a real danger.



Figure 52 Multiple polyposis of the stomach. One area showed adenocarcinoma.

There are no definite characteristic criteria by which malignant and benign polyps can be differentiated clinically or by roentgenologic examination. The only safe method, therefore, is surgical removal of all polypoid tumors of the stomach. A final decision regarding the malignancy or benignancy can be made only by pathologic study of the small tumor. Rigler and Erickson have stressed the precancerous nature of polyps and at the Lahey Clinic about 30 per cent of the gastric polyps removed at operation have proved to be malignant. Many of these tumors are pedunculated and can be excised locally and an immediate pathologic examination made of a frozen section. Should the tumor prove to be malignant, resection of the stomach should be carried out.

The commonest benign tumor is a *leiomyoma* which in some cases is very similar in structure to the leiomyoma of the uterus. This tumor almost always is single; it arises from the smooth muscle layer of the stomach and usually projects into the lumen of the stomach. Many of these tumors are small and rarely grow to a size that may prove to be of clinical significance. On the other hand, they may attain a very large size and in one of our patients the tumor was as large as a man's head. Boyd has stated that these tumors may undergo malignant change, forming leiomyosarcomas. Meissner is of the opinion that there is no proof that a malignant leiomyosarcoma arises from a pre-existing benign leiomyoma. He could find no evidence of such changes occurring in any of the specimens of smooth muscle tumors, benign or malignant, which he studied. We have had twenty-eight patients who have had operations for benign leiomyomas. There are no signs or symptoms which are pathognomonic of these tumors. The clinical course depends on the size of the tumor, whether the origin is near the gastric orifice where obstruction may be produced or whether ulceration occurs in the mucosa (Fig. 53) with resulting hemorrhage. It is impossible to determine clinically or roentgenologic examination, or even by direct visualization of the tumor at operation whether these tumors are sarcomatous or benign. Because of this they should be removed surgically. Resection is preferable to local excision unless the tumor is very small, uncomplicated and easily removed, provided also that an immediate microscopic examination verifies the absence of a malignant smooth muscle tumor.

The lipomas, fibromas, angiomas and neu-



Figure 33. a, Leiomyoma of stomach partially resected stomach. Note ulceration of tumor b Discrete rounded intraluminal filling defect, leiomyoma of stomach.

leiomyomas originate in any part of the gastric wall and are extremely rare: differentiation cannot be made on the basis of clinical or roentgenologic findings. The true pathologic state of these tumors cannot be recognized until they are excised and histopathologic examination is made.

One rare type of tumor arising in the stomach is *aberrant pancreatic tumor*. These tumors are of practical significance only because they produce filling defects as noted on the roentgenogram and may be confused with gastric polyps, peptic ulcer or even gastric carcinoma. Most of these tumors are located in the pylorus and the first part of the duodenum and are asymptomatic. They ordinarily arise on the greater curvature and are usually discovered incidentally upon roentgenologic examination of the gastrointestinal tract. Aberrant pancreatic tumors arising in the stomach have been noted in twelve patients. If gastrointestinal symptoms are present, they suggest peptic ulcer, pyloric obstruction, gallbladder disease or functional indigestion. Gastric surgery is indicated in these subjects to rule out the possibility of a malignant tumor or a prepyloric ulcer which may carry the danger of malignant degeneration. In the majority of the patients, the tumor can be locally excised, provided a pathologic examination is carried out immediately and confirmation obtained of the benign nature of the tumor. Once these tumors are seen at the operating

table, the surgeon can, in the majority of cases, make a diagnosis from the gross appearance inasmuch as they are in the gastric wall and are submucosal in origin.

MALIGNANT TUMORS

Carcinoma is the most frequent form of malignant neoplasm arising in the stomach and comprises 90 to 95 per cent of all gastric tumors. Other tumors arising in the stomach include the sarcomas, or the malignant mesenchymal tumors, which account for about 3 per cent of the tumors of the stomach.

Sarcoma. Sarcoma of the stomach is not a common gastric neoplasm but must be considered in the differential diagnosis of tumors arising in this location. Fifty-one such tumors were noted in the group of 1700 gastric tumors. The reported incidence of sarcoma of the stomach varies. Ewing estimated that sarcomas comprised about 1 per cent of all gastric neoplasms. D'Aunoy and Zoeller found only 335 cases of sarcoma recorded in the literature through 1929.

Theoretically sarcoma of the stomach may arise from any of the mesenchymal tissue components of the organ. Malignant sarcomatous tumors arising from the fibrous tissue, fat and blood vessels however are so rare that they are merely surgical curiosities. For practical purposes, there are two types of gastric sarcomas: those arising in the smooth muscle and those arising from

Table 2. Pathologic Classification of Malignant Neoplasms of the Stomach

I. Carcinoma

- 1 Carcinoma in polyp
- 2 Adenocarcinoma
- 3 Carcinoma simplex
- 4 Mucinous carcinoma
- 5 Adenocanthoma

II Sarcoma

- 1 Lymphoma (unclassified)
 - a. Hodgkin's disease
 - b. Reticulum cell sarcoma
 - c. Lymphosarcoma
 - d. Lymphocytoma
 - e. Macrofollicular lymphoma
- 2 Leiomyosarcoma

the lymphoid tissue elements. The pathologic classification is given in Table 2. The general term includes the malignant lymphomas and these may be subdivided according to histopathologic criteria laid down by Warren and Lulenski. The most frequent types of these tumors however are the lymphosarcomas and the Hodgkin tumors arising in the stomach. These localized lesions in the stomach may represent instances in which a primary focus of a generalized lymphoma is detected before widespread involvement is recognized. The tendency for malignant lymphomas to remain circumscribed in the gastrointestinal tract is commonly recognized. Malignant lymphomas may arise in any of the various segments of the gastrointestinal tract but occur most often in the stomach. Warren has reported that thirty out of forty-nine gastrointestinal lymphomas arose in the stomach.

The clinical behavior of malignant lymphomas of the stomach is similar to that of carcinomas occurring in this organ. The symptoms in general are those of gastric carcinoma, chiefly epigastric pain, simple indigestion and anorexia. The roentgenograms are usually diagnostic of a malignant lesion involving the stomach and the roentgenologic examination presents no characteristic picture on which a diagnosis of sarcoma can be made with any degree of certainty (Fig. 54). In a group of forty-one patients having sarcoma of the stomach, the preoperative diagnosis was made in only one subject in whom the tumor arose from lymphoid tissue, but was suspected in two other patients.

The only treatment of value in these patients with gastric sarcoma is radical resection of the stomach which, when the tumor arises on lymphoid tissue, should be followed by deep irradiation treatment. Sarcomas of



Figure 54. Reticulum cell sarcoma of stomach proved by exploration and biopsy. Marked deformity of entire media of stomach with coarsening of mucosal folds suggests lesion other than carcinoma.

lymphomatous origin will respond to x-ray therapy which should be used four to six weeks after resection, but x-ray treatment may also be effective in those tumors of lymphoid origin which are too extensive to warrant resection. One of our patients with Hodgkin's disease of the stomach, who had x-ray therapy only lived seven years after exploratory operation and biopsy. Resection was not possible because the tumor was too extensive. Irradiation treatment should not be used before resection is carried out in those patients with suspected malignant lymphoma, since irradiation of a hollow viscus containing a large ulcerating tumor may entail the danger of gross hemorrhage and perforation. If at operation resection is not possible and biopsy only is done to determine the nature of the tumor this risk of x-ray therapy must, of course, be taken.

The resectability rate for sarcoma is higher than for carcinoma and the five-year survival after resection is surprisingly good—44 per cent of our group survived five years or longer after resection.

Leiomyosarcoma. In a total of fifty-one sarcomas of the stomach, sixteen arose from smooth muscle to form leiomyosarcoma. These tumors, similar to the benign leiomyomas, exhibit a marked tendency to hemorrhage and thus produce marked secondary anemia and yet often they produce surprisingly little gastric distress, even though the tumor is large, unless there is hemorrhage con-

sues. One such resected tumor weighed 50.50 gm. Many of these sarcomas are resectable and the prognosis after resection is much more favorable than for carcinoma.

Leiomyosarcomas resemble the benign leiomyomas grossly and can be recognized only upon microscopic examination. There is no proof that any of the leiomyosarcomas arise from a pre-existing benign tumor. Pathologically, leiomyosarcomas arise from the gastric muscularis and are composed of spindle-shaped, relatively well-differentiated, smooth muscle cells in which scattered mitoses are present in each mass. The general impression is that these tumors are of rather slow growth and low grade of malignancy. They can be differentiated from the benign leiomyomas by evidence of invasion of normal tissue of more active growth. Invasion of blood vessels or lymph vessels is not found.

Leiomyosarcomas are considered to be slow growing and not prone to metastasize; they seldom recur after operation. We have noted metastatic lesions recurring in four patients with leiomyosarcoma, with metastasis to the lymph nodes, liver and the lungs. Results, in general, are good after resection of malignant smooth muscle tumors and, unless there is evidence of metastasis at the time of operation, cure can be expected. However these lesions can metastasize and cause death. One patient who had a large leiomyosarcoma on the greater curvature of the stomach died of extensive metastasis to the liver. At post-mortem examination the liver weighed 22 pounds and was almost completely replaced by tumor tissue. The primary growth also filled the entire left side of the abdominal cavity.

Irradiation treatment has no effect on sarcomas of smooth muscle origin.

Carcinoma. Carcinoma arising in the stomach is one of the most frequent forms of can-

cer in human beings and, similar to other forms of malignant disease, is found with greatest frequency in the older age group. It is estimated that 25 000 to 35 000 persons die each year in the United States from cancer of the stomach. It can be estimated further that approximately three or four times that number are alive with unrecognized disease or still living with diagnosed cancer either inoperable or successfully resected. The Bureau of Vital Statistics in the United States in 1952 reported total deaths from cancer of the stomach to be 23,466. This is a slight decrease over previous reports and represents 10.5 per cent of the total number of deaths from malignant disease (Table 3). The total number of deaths from neoplasms of the stomach for 1954, 1955 and 1956 (Table 4) is approximately the same for these years. Ninety-five per cent of the carcinomas of the stomach occur in patients more than forty five years of age and in the 1952 report from the Bureau of Vital Statistics, 92.2 per cent of patients were between the ages of fifty and 100. The age and sex distributions are shown in Table 5.

Pathology. There is no uniformity in the literature regarding the classification of gastric tumors. It is difficult to fit the microscopic features of a gastric carcinoma into a classification based on gross features. We use the classification as suggested by Warren and Meissner which is based on histopathologic features alone, and we find this classification satisfactory for the clinician and pathologist in discussing malignant neoplasms which arise in the stomach (Table 2). The terms medullary and ulcerating are used in describing the gross characteristics and indicate gross changes noted in carcinomas. Linitis plastica is a carcinoma simplex with diffuse infiltration of the entire stomach wall (Fig. 55). Warren and Meissner have not attempted to grade tumors on cellular char-

Table 3 Total Deaths in the United States, 1948 and 1952: Relation to Cancer Deaths

	1948	1952
Total population, estimated	146,571,000	155,767,000
Total deaths from all causes	1,444,337	1,496,838
Deaths due to cancer and other malignant tumors	197,042	222,511
Deaths due to cancer of stomach	26,215 (13.3%)	23,466 (10.5%)

Table 4 Malignant Neoplasms of Stomach—Mortality

YEAR	TOTAL DEATHS	MALES	FEMALES
1954	23,019		
1955	22,257		
1956	22,119	13,952 (63.1%)	8167 (36.9%)

Table 5 Malignant Neoplasms of the Stomach—Age and Sex Distribution
(U.S. Bureau of Vital Statistics—1932)

AGE, YEARS													
	1 to 9	10 to 19	20 to 29	30 to 39	40 to 49	50 to 59	60 to 69	70 to 79	80 to 89	90 to 99	100 and over	Not Stated	
Deaths	9	5	53	350	1393	3744	6927	7405	5290	271	6	13	23,466
61 per cent age 60 to 79 years													
92.2 per cent over the age of 50 years													
SEX DISTRIBUTION													
MALE PATIENTS						FEMALE PATIENTS							
	NUMBER		PER CENT			NUMBER		PER CENT					
	14,758		62.9			8,706		37.1					

acteristics because it is a difficult procedure and may vary with the individual pathologist. In their experience the gradation of tumors has assisted very little in the prognosis of carcinoma of the stomach. In a group of 633 patients who had a probable curative resection, there appeared to be little difference in the five-year survival rate based upon the microscopic diagnosis of the tumor except for those patients with mucinous adenocarcinoma.

Gastric cancer has a tendency to metastasize to regional lymph nodes, to exhibit a diffuse lymphatic spread in the gastric wall and also to show vascular invasion. This tendency to vascular invasion may have no relation to involvement of the lymph nodes or the lymphatic system, as pointed out by Meissner. Lymphatic spread may extend across the pylorus into the duodenum and has been noted 3 cm. distal to the pyloric vein. It may also involve the esophagus. Regional lymph node involvement is not uncommon and occurred in 61.8 per cent of 856 resected cases.

Carcinoma arising in the cardia along the lesser curvature has a marked tendency to involve the lower esophagus. There is also a tendency for tumors of the media to involve the lymph nodes of the splenic group and tumors of the lower part of the stomach, even on the left lesser curvature may show a strong tendency to spread to the lymph nodes of the splenic hilum. There is then a tendency to spread to the duodenum, esophagus and the regional lymph nodes. Moreover a significant portion of the immediate lymphatic drainage from the stomach occurs into the peripancreatic tissues, which at

operation may be technically inseparable from the pancreas. Routine elimination of lymph nodes along the major vessels and omentum, as well as removal of the spleen, undoubtedly improves the cure rate.

Symptoms. The fundamental inherent weakness of methods for early clinical detection of cancer of the stomach is due to the fact that the disease may be asymptomatic until it has spread to adjacent structures or has metastasized through blood vessels and lymphatics. Early symptoms may appear only after the disease is surgically incurable and may be vague and nonspecific. In a recent analysis of the earliest symptoms noted in 1708 consecutive patients with carcinoma of the stomach, we noted the frequency of early symptoms (Table 6). Epigastric pain and discomfort, anorexia and vomiting were among the earliest symptoms. Thus, epigastric discomfort and pain are



Figure 53. Linitis plastica—diffuse carcinoma simplex. Specimen removed at total gastrectomy. Note marked infiltration of gastric wall.

Table 6. *Earliest Symptoms of 1708 Patients with Gastric Carcinoma*

EARLIEST SYMPTOM	PER CENT
Epigastric pain	55
Anorexia	15
Vomiting	12
Weakness	9
Nausea	8
Dysphagia	7
Belching	5
Gas	5

very common early symptoms and when persistent and progressive demand early roentgenographic examination. Weight loss of some degree was noted in 90 per cent of patients. Great weight loss, severe anemia, vomiting, hematemesis and melena are evidences of advanced carcinoma and too often are manifestations of inoperable carcinoma of the stomach.

Diagnosis can be established early only upon the basis of suspicion of carcinoma on the part of the physician in all patients with indigestion beginning later in life and in particular in the male past forty-five years of age. Patients who show evidence of weight loss and marked anemia without evidence of gastric obstruction should be carefully screened for the possibility of gastric carcinoma. Bleeding from a gastric carcinoma may be evidenced by vomiting blood or by

passage of black tarry stools (Fig. 50) but this is not as common as in benign ulcer and again, occurs usually in advanced carcinoma.

Precursors of carcinoma. Earlier diagnosis will be made when the importance of the precursors or the precancerous lesions of the stomach, is well recognized. Many observers agree that chronic gastritis may be a precursor of cancer of the stomach. Not all stages or types of chronic gastritis can be considered precancerous, but chronic gastritis occurring in the prepyloric or antral region should be carefully differentiated from tumors. Warren and Meissner have emphasized the fact that the epithelial and infiltrative changes which occur in the gastric wall in chronic gastritis could very well be the precursors of carcinoma. Gastric polyps should be regarded as premalignant lesions. In our experience, 30 per cent of the gastric polyps have proved to be malignant upon excision and microscopic examination. Clinical evidence seems to support the conclusion that approximately 10 to 20 per cent of gastric ulcers which appear benign grossly on roentgenologic and clinical examination prove to be malignant on microscopic examination. We should therefore be very suspicious of cancer in the so-called benign gastric ulcer.

We should be suspicious of cancer in patients who have a family history of gastric

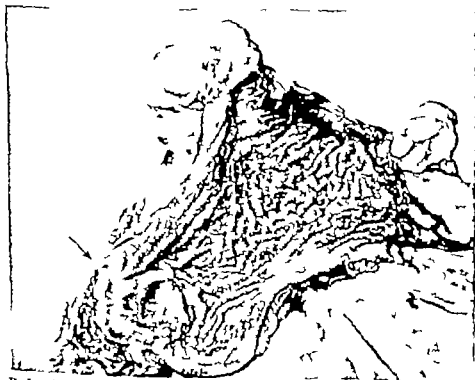


Figure 50. Early adenocarcinoma in antral area of stomach in a man seventy-one years of age. The only symptom was melena.

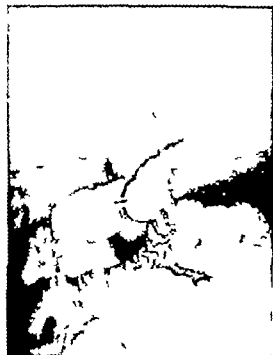


Figure 57 Extensive adenocarcinoma of the distal half of the stomach. At operation, the cancer was found to be nonresectable because of widespread metastases. The tumor was palpable in the epigastrium.

carcinoma and in those individuals who are known to have achlorhydria of several years duration. The incidence of carcinoma of the stomach in patients with pernicious anemia has been shown repeatedly in the literature to be three times as frequent as in other patients of the same age group. Laboratory tests such as gastroscopy and roentgenologic examination should be carried out carefully in the patient with pernicious anemia.

Diagnosis. Early diagnosis is predicated upon the basis of constant suspicion of carcinoma as the source of epigastric distress in patients past the age of forty-five years, particularly in the male. Appropriate laboratory studies should be carried out at once and palliative treatment for gastric distress should not be employed until the possibility of the presence of carcinoma is definitely excluded.

Roentgenographic diagnosis in our group of patients with gastric cancer was accurate in 94 per cent of the group Guiss, in a collective review of the end results of gastric carcinoma in 2891 patients, reported that x-ray examination was accurate in 90 per cent of the patients and in an additional 5 per cent the roentgenogram showed a gastric abnormality. Other laboratory procedures should be utilized when cancer is suspected. These include gastroscopic examina-

tion in patients with gastric ulcer gastric polyp or chronic gastritis. Esophagoscopy should be employed in all patients with carcinoma involving the cardia in order to determine its extension to the esophagus, and involvement of the esophagus should always be suspected when dysphagia is present. The cytologic techniques of Papanicolaou are of value and, in some instances, early diagnosis may be established when the roentgenographic examinations fail to indicate the nature of the lesion. Other laboratory data, such as determination of gastric acidity and the red blood count, may assist materially in early diagnosis. Achlorhydria occurred in 36 per cent of our patients and free acid of less than 20 was noted in 18 per cent. Acid values however may be normal in many patients. Anemia may not be an outstanding symptom except in far advanced carcinoma, and hemoglobin and blood count determinations may be of value as diagnostic procedures only when marked anemia is present.

On examination, a mass may be demonstrated in the mid or epigastric part of the abdomen (Fig. 57). In a group of 1708 patients palpable tumor masses were noted in 516 patients or 30.2 per cent. Too often this is evidence of advanced cancer. Large palpable tumors, however, may prove to be resectable at laparotomy and in 192 of this group of 516 they were resectable.

All tumors of the stomach, as evidenced by roentgenographic examination must be considered to be carcinomas, since about 95 per cent of gastric tumors prove to be this type of lesion. A few tumors such as leiomyomas or leiomyosarcomas and the polyp show characteristic roentgenologic findings, but any intrinsic defect must be regarded as carcinoma until proved otherwise. Differential diagnosis, therefore, must be made of benign tumors, chronic gastritis and gastric polyp. The possibility of gastric carcinoma must be excluded in patients who have symptoms arising from diseases of the pancreas, gallbladder and colon. Digestive disturbances may result from disease arising in other organs, but, on the whole, the possibility of the presence of such diseases can be excluded readily by roentgenologic and gastroscopic examination. Cancer can usually be differentiated correctly from gastric diverticula by roentgenologic examination and diverticula rarely require any surgical procedure.

Bezoars which are intragastric masses composed of hair (trichobezoar) vegetable

fibers and fruit seeds (phytobezoar) are encountered occasionally and may fill and form a complete cast of the stomach. In the majority of cases, the bezoar has a characteristic appearance roentgenologically.

Syphilis of the stomach is a very rare condition and, in view of this every individual with a filling defect in the stomach should have an exploratory operation even though the Wassermann reaction may be positive and there is a definite history of gastric syphilis. We have seen gastric syphilis in a young man remain unchanged with intensive antisyphilitic therapy over a long period, the patient finally coming to laparotomy in order that the presence of cancer could definitely be ruled out. Gastric syphilis may resemble gastric carcinoma, the two diseases can occur concurrently and the most likely diagnosis to be made will be carcinoma. Valuable time should not be lost by waiting for the effect of antisyphilitic treatment in making the differential diagnosis.

Operability. In the light of our present knowledge, the only method of treatment for gastric carcinoma is removal of the tumor by partial or total resection of the stomach. The primary object of this is to relieve the patient of gastric distress and obtain a cure of the carcinoma, if possible. In patients with nonresectable tumors, surgical treatment may prolong life or alleviate suffering by a palliative procedure if the disease is not too widespread in the abdomen. The inevitable result of untreated carcinoma is 100 per cent mortality. We believe that all patients with gastric cancer should have the benefit of laparotomy when there is no definite evidence of extragastric spread which would preclude removal by surgical resection. During the physical examination preoperatively the abdomen should be palpated carefully for a tumor and for the presence of an enlarged, nodular liver and ascites. Occasionally extension of the tumor to the umbilicus will be found and in such subjects palliative operation is not advisable or helpful. Careful rectal examination with the patient in the knee-chest position should be made to rule out the presence of peritoneal implants in the pouch of Douglas. Enlarged supraclavicular lymph nodes, the nodes of Virchow frequently indicate advanced cancer and operation is hopeless. If a Virchow node is present, a biopsy specimen should be obtained before any abdominal operative procedure is carried out. A roentgenologic examination of the chest should be made in all of these patients. If the various areas for

metastases of carcinoma are proved to be unaffected exploratory laparotomy should be performed regardless of the size of the tumor and, in the majority of subjects irrespective of the age of the individual operation can be carried out safely in the older age group provided the case is carefully evaluated preoperatively and the patient is properly prepared.

Operation. In a series of 1708 patients with carcinoma of the stomach observed at the Lakes Clinic from 1932 through 1954 the disease was considered to be too far advanced to attempt surgical resection in 810 and too advanced to attempt even exploratory laparotomy in forty-two. A total of 856 patients had resection and in 653 of these the resection could be considered curative. The term "curative resection" refers to a radical gastric resection total or high partial following which there is a reasonable hope of cure if all gross evidence of carcinoma is removed. We do believe, however, by employing exploratory laparotomy in all patients in whom metastasis cannot be demonstrated a higher rate of resection will be possible without an appreciable increase in mortality.

Operative treatment of cancer of the stomach from the standpoint of cure consists of a radical partial gastric resection or total gastrectomy. The first successful partial gastric resection for gastric cancer was done by Billroth in 1881. The first successful total gastrectomy for cancer was done by Schlotter in Zurich in 1897. In the United States the method used most frequently has been the Hofmeister modification of the Polya technique. This is an excellent method and permits high resection of the stomach with removal of all lymph nodes and the spleen. Reports of two important modifications of the Billroth II method were published in 1888 and 1889 the first by Krönlein and the second by von Eiselsberg. Von Eiselsberg closed the upper transected end of the stomach thus reducing the size of the gastric orifice and anastomosed the rest of the divided end of the stomach to the side of the jejunum. This method lends itself readily to radical partial resection of the stomach. These two procedures are often erroneously credited to Hofmeister and Polya, but were described by them in 1908 and 1911 respectively at a much later date than were the descriptions of the operations by von Eiselsberg and Krönlein. The Hofmeister modification places the anastomosis anterior to the colon.

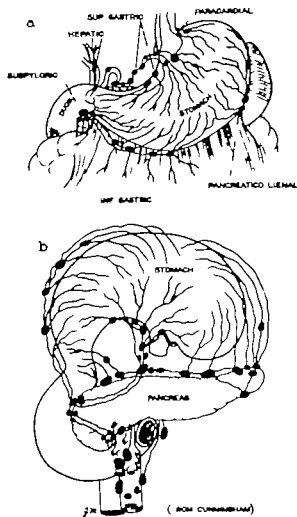


Figure 58 Distribution of lymph nodes of the stomach. Removal of all lymphatic drainage areas is part of a well-planned gastric resection.

Total gastrectomy should be employed in those patients whose cancerous process can not be removed by any method short of a total removal of the stomach. Cancers arising in the distal half of the stomach or in the prepyloric region may be radically removed by a high subtotal resection. In view of the demonstrated metastasis to the splenic lymph nodes in many patients having gastric cancers, the spleen is removed in all patients subjected to partial or total gastrectomy. The omentum is excised completely including the omentum between the spleen and the greater curvature of the stomach and also the gastrohepatic omentum, and, when partial resection is performed, only a small part of the stomach is permitted to remain, about 15 to 20 per cent. This more radical partial resection facilitates jejunal anastomosis, favorably affects the postoperative feeding problem and can be employed in many subjects rather than total gastrec-

tomy. All lymph nodes of the pyloric area, the celiac axis and curvature of the stomach should be included in the block dissection and the first part of the duodenum, almost to the level of the common bile duct, should be excised routinely. Removal of all lymphatic and vascular drainage areas (Fig. 58) is of far greater importance than is the benefit to be obtained from total gastrectomy alone, although total gastrectomy will often be necessary if the cardia is involved or if extensive carcinoma is present. Diffuse gastric cancer cancers extending high on the lesser curvature and those arising in the cardia necessitate total gastrectomy. Radical block dissection of all avenues of lymphatic spread is practically impossible but routine elimination of the lymph nodes along the major vessels and the omentum will undoubtedly improve the cure rate. Total gastrectomy for the majority of patients with gastric cancer has not yet been shown to increase the five-year survival rate, on the other hand, it does produce problems of nutrition and of anemia as a result of the disturbed physiology. After total gastrectomy many of these patients have considerable difficulty in obtaining sufficient caloric intake. Some patients, however do very well after total gastrectomy if the physician constantly supervises the diet, nutrition and general hygiene and prevents the development of anemia.

Exploratory laparotomy carries very little operative risk and is the only method of determining whether the tumor is resectable or is so advanced that extirpation would offer nothing to the patient. If the tumor cannot be removed even by total gastrectomy the best palliative procedure is partial gastrectomy. We believe that the ulcerating lesion can be removed safely with partial gastrectomy that this procedure carries no higher mortality than does gastroenterostomy and that the patient has a greater opportunity to obtain relief of his symptoms by a palliative gastric resection. Total gastric resection should not be employed as a palliative operation unless there is a reasonable chance of removing all carcinomatous tissue. We ordinarily use a type of partial gastric resection commonly known as the Hofmeister procedure (Fig. 59). The jejunum is brought anterior to the colon and the stomal orifice is made between a portion of the transected end of the stomach and side of the jejunum.

The Billroth I procedure (Fig. 60) is rarely used since a more radical removal of

Table 7 Percentage of Total Gastrectomies in All Resections for Malignant Disease of the Stomach 1944-1957

YEAR	CASES, NO.	RESECTION		
		PARTIAL NO.	TOTAL NO.	PER CENT
1944	38	30	8	21.0
1945	54	45	9	16.6
1946	34	26	8	23.5
1947	63	51	12	19.0
1948	43	32	11	23.5
1949	53	36	17	32.0
1950	60	38	22	36.6
1951	47	37	10	21.2
1952	42	37	12	24.4
1953	57	39	18	31.5
1954	30	25	5	16.6
1955	27	19	10	34.5
1956	24	17	7	29.2
1957	30	22	8	26.6
Total	611	454	157	

the duodenum, together with radical partial resection of the stomach, is more practical by the Hofmeister method.

The relative frequency of employment of total gastrectomy versus partial gastrectomy in the Lahey Clinic is shown in Table 7. Gastric resection can be accomplished most satisfactorily in the majority of patients through an abdominal incision—an abdominal approach is used in 90 to 95 per cent of our subjects. A left transrectus incision is employed, extending from the costal margin caudally to just below the level of the umbilicus.

The abdominal cavity can thus be explored and the decision made for partial or total gastrectomy (Fig 61). If total gastric resection is deemed necessary the incision can be extended and converted into a thoracoabdominal one by cutting across the costochondral arch into the fifth or sixth intercostal space. An incision of the diaphragm can be carried through the hiatus if a portion of the esophagus is to be excised. If desired and if preoperatively total gastrectomy is deemed necessary because of information obtained concerning esophageal

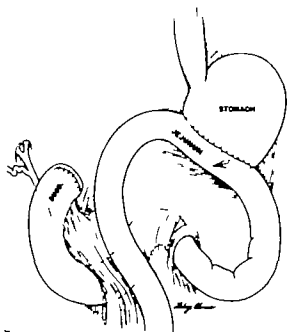


Figure 59 Hofmeister method of gastric resection, anastomosis is made anterior to the transverse colon.

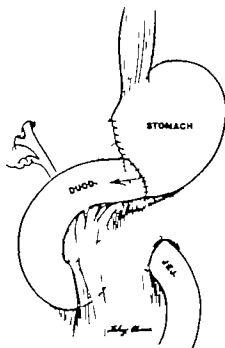


Figure 60 Billroth I, partial gastric resection.

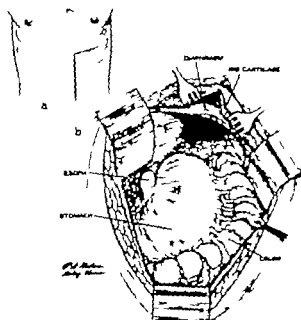


Figure 61 Abdominal incision for exploration and partial gastric resection. The incision may be extended to enter the left thoracic cavity by the division of the left costal margin, employed in total resection (Marshall, S F and Uram, H Surg. Gynec. & Obst., vol. 99)

involvement, a thoracoabdominal approach can be made through a low left thoracotomy incision (Fig. 62). The ninth rib is resected and the incision through the abdominal wall is extended in the direction of the thoracic incision by cutting the costochondral arch. In the hands of an experienced surgeon the risk of exploration through the transthoracic approach is no greater than that through an abdominal approach. If resection proves to be possible radical removal of the stomach can be carried out through the transthoracic approach and a more extensive excision of the involved esophagus can be performed. The mortality of total gastrectomy through a transthoracic incision is, perhaps, a little higher than when an abdominal incision is employed. Sweet reported fifteen deaths in a series of seventy seven patients who had total gastrectomy through a thoracic or abdominothoracic incision.

When the abdomen is opened, the extent of the gastric tumor is determined and the abdominal cavity is carefully explored. If no distant metastases are demonstrated, the omentum is detached from the transverse colon and the lesser omental cavity is opened (Fig. 63). With the stomach elevated upward and with the omentum completely detached from the transverse colon, the lesser omental cavity and pancreas are widely exposed. Extension of the malignant process to the pancreas the nodes of the

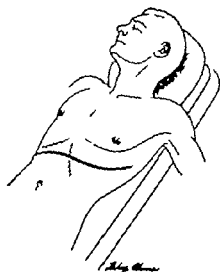


Figure 62 Thoracoabdominal incision for total gastrectomy, the ninth rib is resected, with extension of incision through the abdominal wall (Marshall, S F and Uram, H Surg. Gynec. & Obst., vol. 99)

celiac axis and to the mesocolon and colon can easily be determined and a decision made regarding operability. If the carcinoma of the stomach proves to be too extensive for resection, the operation can be terminated by dropping the colon and omentum back into the abdominal cavity. If resection is considered possible the stomach is mobilized, the principal arteries are ligated and the duodenum is divided at a level just above the ampulla of Vater. All regional lymph nodes are carefully removed, the splenic artery just above the upper border of the pancreas is followed to the splenic hilum, and the splenic artery and vein are ligated just proximal to the left gastrosplenic artery. This will permit ready removal of the spleen and will control bleeding from the short gastric vessel. If it is necessary to proceed then with total resection, this may be done easily (Fig. 64). Thus the stomach is almost completely mobilized following ligation of all the major vessels so that either a total or a high subtotal resection of the stomach can be carried out readily for removal of all of the tumor.

Table 3 Location of Gastric Carcinoma and Average Survival (Total Gastrectomy)

	NO. OF PATIENTS	AVERAGE SURVIVAL (MONTHS)
Lesser curvature	35	27.5
Greater curvature	13	26.0
Fundus	24	24.5
Medea	35	23.1
Prepyloric	16	17.3
Diffuse	41	15.4
Total	164	

If distant metastases are not found at operation, direct extension to adjacent organs does not prohibit resection of the left lobe of the liver and part of the pancreas and colon. The operative mortality has not been increased by extending the operation to include resection of adjacent organs.

Operative mortality The operative mortality in 127 patients having total gastrectomy at the Lahey Clinic over a ten year period (1944 to 1953 inclusive) was 8.7 per cent; previous to 1943 the mortality was 34.6 per cent. The operative mortality for partial gastric resection likewise has been reduced to 3.2 per cent (1950 to 1954). The over all mortality following operative procedures for gastric cancer is 5.7 per cent; this includes all types of operation—exploratory, palliative and partial and total resection.

In earlier years, the mortality in general was higher because of sepsis, which included peritonitis, mediastinitis and pneumonia. With the employment of chemotherapy and antibiotics, the operative mortality has decreased considerably during the past ten years in both procedures—total and partial gastrectomy. Good preoperative care with restoration of blood volume before operation and replacement of blood loss during operation has helped considerably in reducing the mortality in gastric surgery.

At the Mayo Clinic, the mortality rate following resection during the period from 1937 to 1949 showed a definite decline to a level of 8 per cent, which was about half that noted in 1937 (16.2 per cent). Wangen-

steen in 1919 also noted that as resectability increased the operative mortality of resection decreased over a period of ten years. McNeer and Pack reported that the operative mortality in total gastrectomy has been reduced from a high of 30 per cent to 11.4 per cent (1954).

Palliative treatment Great relief of symptoms can be obtained by palliative procedures among which partial gastric resection is most valuable. Gastroenterostomy may have to be used occasionally when the tumor is extensive and pyloric obstruction is present. We prefer partial gastric resection inasmuch as an ulcerating tumor can be removed and this obviates further blood loss with its consequent anemia, relieves obstruction more effectively than does gastroenterostomy, is more effective in obtaining a good state of nutrition and adds considerably to the physical comfort of the patient.

X-ray therapy has not proved to be of great value as a postoperative measure in patients with advanced gastric carcinoma.

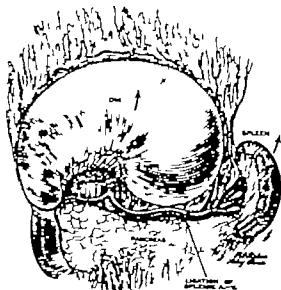


Figure 63 Exposure of the lesser omental cavity. The omentum is detached from the transverse colon and the stomach is elevated upward (Marshall, S. F., and Urrutia, H. Surg. Gynec. & Obst., vol. 90).

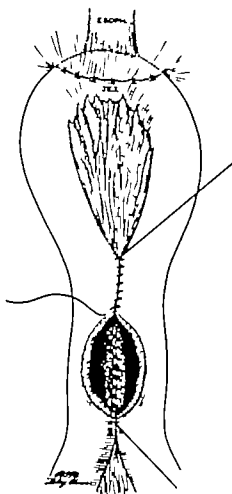


Figure 64 Total gastrectomy and esophagojejunostomy. The food pouch is made by employing a jejunojunctionostomy (Marshall, S. F., and Urrutia, H. Surg. Gynec. & Obst., vol. 90).

It may produce great discomfort, nausea and vomiting without materially affecting the tumor. It may be possible with the newer method, employing two million volt therapy to obtain more effective palliation with this type of irradiation treatment but to date, in a limited number of subjects, the results are inconclusive.

Medical treatment consists of drug therapy for relief of distress and pain. Sedatives will control early distress but, eventually as the disease progresses opiates in some form will be required and should be given as needed. Constant supervision of a dietary regimen may be effective in alleviating or preventing much digestive distress especially when drugs are added. Parenteral administration of fluids will do much to make the patient with advanced carcinoma more comfortable. Gastric lavage using a Levin tube also is helpful in patients with advanced disease in whom a palliative operation has not been possible. This insures decompression of an obstructed stomach with removal of retained food and other material.

Results of treatment In the United States for the year 1956 the total deaths from cancer of the stomach numbered 22,119 of this number 13,952 (63.1 per cent) occurred in males and 8167 (36.9 per cent) in females. By careful selection of all patients with gastric carcinoma, curative resection, either by partial or by total gastrectomy can be performed in 38 to 41 per cent. Over a period of twenty three years 653 (39.2 per cent) of a group of 1666 patients have had curative resections and their course after operation has been carefully followed. In a smaller group of patients it will be possible to do a palliative operative procedure which helps materially in making the patient more comfortable but in most instances does not prolong the length of life.

The five-year cure after total gastrectomy for carcinoma of the stomach in our hands is 14.1 per cent, 26.2 per cent of the patients lived three years or longer. It must be remembered, however, that total gastrectomy has been employed heretofore only in those patients in whom the carcinoma was extensive and could not be resected other than by total resection of the stomach.

Of the group of 653 cases of cancer of the stomach in which resection was possible the curability rate has increased notably. The five year survival rate after resection has risen from 15.4 per cent for the patients operated on during the years 1932 through

1934, and from 29.7 per cent (183 resected cases) for the period from 1945 through 1949 to 39.5 per cent (78 resected cases) for those operated on during 1950 and 1951.

During this same twenty year period (1932 through 1951) the ten year survival rate after curative resection increased from 7.7 per cent for the years 1932 through 1934 to 16.6 per cent for those patients operated on during the years 1941 through 1945. For a group of 1708 cases in which a diagnosis of gastric cancer was made, whether or not resection was performed, the absolute five year survival rate was 10.8 per cent. The survival rate after total gastrectomy in relation to the location of the gastric tumor is shown in Table 8. The survival rate with respect to the histopathologic state of the tumor is poor in patients with mucinous carcinoma, the average length of life of those who had resection being 14.9 months. The survival rate in patients with carcinoma simplex and adenocarcinoma was 21.6 months. The patients with adenocanthoma lived an average of 34 months.

Carcinoma of the stomach can be treated surgically with a low mortality rate—3.2 per cent for partial gastric resection and 8.7 per cent for total gastrectomy.

The treatment of gastric cancer is not hopeless. The curability rate can be elevated by earlier diagnosis through education of the public concerning the possibility of gastric cancer in those individuals who have persistent digestive distress, and by alertness on the part of the physician. Lowered mortality is dependent upon careful preoperative preparation, meticulous technique in gastric resection, the employment of antibiotics when needed, restoration of the volume and the chemical constituents of the blood to normal, and parenteral administration of fluids.

READING REFERENCES

- Berelson, J. Walters, W. Gray H. K., and Priestley J. T. Mortality and Survival in Cancer of the Stomach. Statistical Summary of Experience of Mayo Clinic. Proc. Staff Meet. Mayo Clin. 27: 137, 1952.
- Boyd, W. Pathology for the Surgeon, 7th ed. Philadelphia, W. B. Saunders Company 1955, 737 pp.
- D'Aunoy, R., and Zoeller A. Sarcoma of the Stomach: Report of Four Cases and Review of the Literature. Am. J. Surg. 9: 444, 1930.
- Eusterman, G. B. and Balfour D. C.: The Stomach and Duodenum. Philadelphia, W. B. Saunders Company 1935, 658 pp.
- Ewing, J.: Neoplastic Disease, 3rd ed. Philadelphia, W. B. Saunders Company 1928, 1137 pp.

- Feldman, M.: Clinical Roentgenology of the Digestive Tract, 2nd ed. Baltimore: Williams & Wilkins Company 1945. 709 pp.
- Goss, L. W.: Collective Reviews: End Results for Gastric Cancer: 2591 Cases. Internat. Abstr. Surg. 93:313, 1931.
- Kujper P. J.: The Spread of Gastric Cancer into Section Lines. Arch. chir. neerl. 4:253, 1932.
- Lefevre, H., and Lortat Jacob, J. L.: Indications et résultats de la gastrectomie totale dans le cancer de l'estomac. J. chir. 68:670, 1930.
- Marshall, S. F., and Melchner W. A.: Sarcoma of the Stomach. Ann. Surg. 131:821, 1930.
- Marshall, S. F., and Uram, H.: Total Gastrectomy for Gastric Cancer: Effect upon Mortality, Morbidity and Curability. Surg. Gynec. & Obst. 99:637, 1934.
- McNeer G., and Pack, G. T.: Postoperative Mortality after Total Gastrectomy. Cancer 7:1010, 1934.
- Melchner, W. A.: Leiomyoma of Stomach. Arch. Path. 33:207, 1941.
- Rigler L. G., and Erickson, L. G.: Benign Tumors of Stomach; Observations on Their Incidence and Malignant Degeneration. Radiology 26:6, 1936.
- Stewart M. J.: Observations on Relation of Malignant Disease to Benign Tumours of Intestinal Tract. Brit. M. J. 2:567, 1920.
- Sweet, R. H.: Total Gastrectomy by the Transthoracic Approach: a Subsequent Report. Ann. Surg. 138:267, 1933.
- Walters, W., Gray H. A., and Priestley J. T.: Carcinoma and Other Malignant Lesions of the Stomach. Philadelphia, W. B. Saunders Company 1942.
- Wangensteen, O. H.: Cancer of the Esophagus and the Stomach. Monograph for Physician. Sixth of a Series on Early Recognition of Cancer. 112 pp. New York: American Cancer Society 1931. pp. 99-100.
- Warren A. W.: Malignant Lymphomas of the Gastrointestinal Tract. S. Clin. North America 35:745, 1935.
- Warren, S., and Lulenaki C. R.: Primary Solitary Lymphoid Tumors of the Gastrointestinal Tract. Ann. Surg. 115:1, 1942.
- Warren S., and Melchner W. A.: Chronic Gastritis and Carcinoma of Stomach. Gastroenterology 3:251, 1914.

The Duodenum, Jejunum, Ileum and Appendix

By JAMES T. PRIESTLEY M.D. and
EDWARD S. JUDD M.D.

JAMES TAGGART PRIESTLEY was reared in Iowa because his grandfather the fifth great grandson of Joseph Priestley went west after his graduation from the University of Pennsylvania in 1874. Educated in liberal arts and in medicine at the University of Pennsylvania. Doctor Priestley is a Mayo Clinic product in surgery and carried on his graduate work there. He is one of the members of the surgical staff at the Mayo Clinic.

EDWARD STARR JUDD the son of a surgeon's surgeon, returned to his birthplace to practice his specialty. Educated at Dartmouth College and Rush Medical College he received his surgical training at the Mayo Clinic where he is now a staff member.

THE DUODENUM

Lesions of clinical and pathologic significance in the duodenum other than benign ulcer are relatively uncommon. This fact may explain in part some of the differences of opinion regarding the incidence, diagnosis, clinical importance and treatment of such lesions. Certain of these abnormalities do cause definite and serious symptoms,

however, so it is essential that their presence be suspected clinically and that they be diagnosed accurately and treated properly if the patient is to have a satisfactory result. Others may represent an embryologic abnormality or a benign pathologic change which produces no hazard to life and perhaps no symptoms. Therefore it is apparent that accurate evaluation of the findings re-

Table 9 Lesions of Duodenum Other Than Duodenal Ulcer Treated Surgically at the Mayo Clinic: 1949 to 1953 Inclusive

LESIONS	CASES
Obstruction	29
Diverticulum	11
Tumor	
Benign	6
Malignant	21
Fistula	35
Miscellaneous	18
Total	120

quires close cooperation between the internist, roentgenologist and surgeon if these lesions are to be properly diagnosed and if their proper clinical significance and appropriate therapeutic indications are to be determined.

To determine the type and relative frequency of lesions of the duodenum, other than benign ulcer which require surgical treatment, a review was made of records at the Mayo Clinic for the years 1949 to 1953 inclusive. Table 9 lists the conditions for which operation was performed during this period. There were of course many additional patients on whom the diagnosis of some type of duodenal lesion was made but for whom operation was not advised.

Certain anatomic features are of importance to the surgeon. The duodenum commonly is spoken of as having three parts, namely the first, second and third. Sometimes a fourth portion is described. The general conformation of the duodenum is that of the letter C or U with some variation in the length and curvature of its parts. The first part, usually about 2 inches in length, extends from the pylorus posteriorly and to the right and lies to the right and above the head of the pancreas. The hepatic artery courses on the cephalic side and the gastroduodenal artery common bile duct and portal vein lie behind this part of the duodenum. The first portion of the duodenum is commonly referred to by the roentgenologist as the duodenal cap or bulb. The second part of the duodenum extends for 3 or 4 inches and is directed vertically downward, with some concavity to the left in which lies the head of the pancreas. The common bile duct and the major and minor pancreatic ducts enter the posteromedial aspect of the midportion of the second part of the duodenum. Posteriorly this part of the duodenum is usually in close relation with the right adrenal gland, kidney renal ves-



Figure 65 Roentgenogram taken after the ingestion of barium. It reveals compression of the duodenum caused by carcinoma of the pancreas (Boeker, J. W., and Khrilín, B. R.: *Am. J. Roentgenol.*, vol. 57)

sels and inferior vena cava. The third, or transverse portion of the duodenum runs retroperitoneally for a distance of approximately 4 inches in a transverse direction at about the level of the third lumbar vertebra. Its anterior and inferior surfaces are covered by parietal peritoneum extending downward to the root of the transverse mesocolon and it is crossed by the superior mesenteric vessels and the root of the mesentery of the small intestine in which those vessels are located. Therefore, it is apparent that, unless one is careful, examination of this portion of the duodenum may be difficult or incomplete in the usual abdominal exploration. In its terminal portion, sometimes considered as the fourth part, the duodenum ascends along the left side of the aorta and then turns forward to join the jejunum. At this point it is fixed by the ligament of Treitz, which is mostly a fibrous band but which may contain some muscle fibers.

Obstructive Lesions. Other than benign ulcer a variety of causes exist for acquired duodenal obstruction. The clinical characteristics of these lesions and the indications for treatment naturally vary with the cause.

Acquired obstruction. Acquired obstruction of the duodenum, usually seen in adults, is most often caused by a malignant growth that arises near the duodenum, as in the pancreas, or by metastatic involvement of tissues adjacent to the duodenum, which in turn causes pressure on the duodenum (Fig.

(65) Primary tumors of the duodenum may also cause obstruction. Inflammatory processes, reactions incident to foreign bodies previous operation or some other intra-abdominal lesion likewise may cause duodenal obstruction. A nonspecific inflammatory reaction similar to regional ileitis occasionally may occur in the duodenum and cause disturbances in motility. Rarely a granulomatous reaction may be encountered in the duodenum. Pancreatitis, pancreatic cysts, postoperative adhesions, post-traumatic hematomas and post-traumatic stenosis of the duodenum also have been reported as causes of duodenal obstruction.

In all such cases, the symptoms and findings of duodenal obstruction are present in varying degrees although perhaps overshadowed by coexistent symptoms and findings. Appropriate treatment is determined after careful examination and evaluation of the patient. Operation often is advisable and its exact type usually can be determined only after the abdomen is opened and the true nature of the obstruction accurately assessed.

The diagnosis of "duodenal stasis" formerly was made with some frequency but it is relatively uncommon at present. The patient usually presents a variety of functional symptoms, anorexia, belching and "fullness" in the upper part of the abdomen usually are noted. Roentgenologically the duodenum appears rather large, but no definite cause of obstruction can be demonstrated. The cause and treatment of such a condition appear rather uncertain, but the management should be conservative except perhaps in the extremely rare case.

Tumors. Primary tumors of any type in the duodenum are uncommon. If lesions which are not true neoplasms, such as a pancreatic rest, are excluded, probably the majority of tumors in the duodenum are malignant. The great majority of malignant tumors in the duodenum are adenocarcinomas. It has been estimated that approximately half of all primary adenocarcinomas of the small intestine are found in the duodenum. Ewing has stated that malignant tumors of the duodenum constitute between 2 and 3 per cent of all neoplasms of the gastrointestinal tract.

Benign Tumors. The commonest benign tumor of the duodenum is an adenoma. This may or may not present as a polyp (Fig. 66). Probably the most frequently found polypoid lesion in the duodenum results from protrusion of gastric mucosa through

the pylorus into the duodenum. Other benign tumors of the duodenum, all of which are uncommon, include myoma, leiomyoma, lipoma, hemangioma, fibroma, cysts, angiosarcoma, fibroadenoma, fibromyoma and, if they are to be included in such a list, pancreatic rest and hypertrophy of the papilla of Vater. In most cases lesions of this type are small, cause no symptoms and are discovered incidentally. Occasionally they may become large, interfere with duodenal motility, possibly cause some obstruction of the common bile duct or bleed. The true nature of these benign lesions often cannot be determined with certainty without surgical exploration.

Management of lesions of this type varies with the individual patient. Some lesions are entirely symptomless and the roentgenologist is relatively certain that they are benign. Under these circumstances, especially if the patient is not a favorable candidate for a surgical procedure, it may be desirable to temporize and re-examine the patient in six or eight weeks. If the lesion has not changed roentgenologically at this time, operation again may be deferred and the patient re-examined in three or four months. In this manner the benign nature of the lesion can be determined with further certainty and operation avoided. Periodic examination in the future over a period of years would be desirable under such circumstances. Should the lesion evidence increase in size, opera-



Figure 66. Benign polypoid tumor of the duodenum evidenced roentgenographically after ingestion of barium.

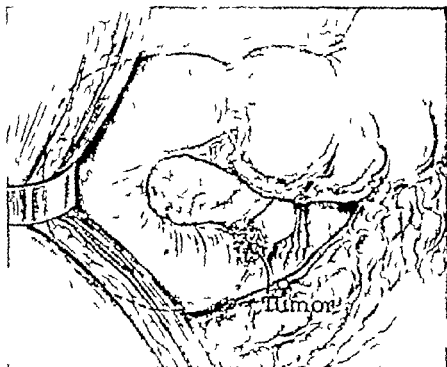


Figure 67 Carcinoma of the third portion of the duodenum. Note dilatation of the duodenum proximal to the lesion. This patient was alive and well five years after operation.

tion should be performed promptly. One always must be mindful of the possible development of malignant changes in an adenoma or a polyp and be guided accordingly in surgical judgment. In contrast, if the lesion causes symptoms or if the roentgenologist cannot be relatively confident of the benign nature of the lesion, exploration is advisable without delay.

At the time of operation for a tumor of the duodenum, the lesion should be examined and carefully inspected by transduodenal exploration. Its true pathologic nature should be determined by biopsy and immediate histologic study. If the lesion proves to be benign, simple local excision is all that is required.

Malignant tumors. Adenocarcinoma represents approximately 90 per cent of all malignant tumors found in the duodenum (Fig. 67) although sarcoma and leiomyosarcoma and a few other types have been reported. Among patients who have duodenal malignant lesions, males predominate over females in a ratio of 2 or 3 to 1. Malignant tumors of the duodenum may be classified as supra-ampullary, peritampullary or infra ampullary. In some series, the peritampullary group is said to be the largest, constituting 50 to 60 per cent of all lesions. However, it appears likely that, if lesions which actually arise in the ampulla of Vater are excluded, true primary duodenal malignant tumors

arise somewhat more often in the third portion of the duodenum than they do in either the first or second portion.

Most often symptoms caused by a malignant lesion in the duodenum appear gradually over a period of months. Four different types of symptoms may develop and direct attention to the lesion. These include those referable to duodenal obstruction, duodenal bleeding, duodenal perforation or obstruction of the biliary or pancreatic ducts. Obstruction may be the result of a polypoid or napkin ring type of neoplasm and may cause variable degrees of crampy pain, vomiting and proximal dilatation of the duodenum and stomach. Bile may or may not be present in the vomitus, depending on the location of the lesion. Bleeding may be massive but often is slow and of the gradual oozing type, so that the patient may present himself to the physician primarily because of weakness and anemia. Perforation is usually gradual and may result in pain of the type associated with a penetrating duodenal ulcer situated on the posterior wall or with pancreatic neoplasm. Actually pain of one type or another is the commonest chief complaint. Obstruction of the biliary or pancreatic ducts may result in jaundice or diarrhea or both.

A definite diagnosis usually can be made by a competent roentgenologist, although in describing the lesion he may use such

terms as filling defect "polypoid lesion" and intrinsic lesion." Occasionally an extensive neoplasm arising for example in the pancreas, may invade the duodenum secondarily and simulate a primary duodenal neoplasm. The presence of complete duodenal obstruction presents increased difficulty in accurate roentgenologic diagnosis.

Whenever possible surgical removal is the treatment of choice for a malignant lesion of the duodenum. The surgeon must determine at the time of surgical exploration whether it appears possible to remove the lesion with some hope of cure or whether involvement and extension of the malignant process are such that only some palliative procedure appears advisable. If the latter is the case, efforts should be directed to relieve symptoms as completely as possible with minimal operative morbidity and mortality rates. Various surgical procedures may be employed under these circumstances depending on the findings. These techniques include duodenojejunostomy, gastroenterostomy, low gastric resection of the Billroth II type and cholecystenterostomy or some other type of anastomosis to relieve obstruction of the choledochus and perhaps also the main pancreatic duct. Irradiation has not proved of value unless the lesion is of lymphomatous origin.

If it appears possible that the growth can be resected with some prospect of cure, this, of course, should be done. Any operation performed for removal of a malignant duodenal neoplasm should embody the principles of operations performed elsewhere in the body for malignant growths. This usually entails a Whipple type of operation with removal of the distal part of the stomach, the entire duodenum, the lower end of the common bile duct, the head of the pancreas and adjacent regions of lymphatic drainage from the duodenum. When the lesion is situated immediately below the pylorus, in the third portion of the duodenum, or at the duodenojejunal angle resection of only a portion of the duodenum and its related regions of lymphatic drainage may be reasonable. Thus an extremely extensive procedure with its attendant high morbidity and mortality rates may be avoided.

Diverticula. Diverticula probably occur more often in the duodenum than in any other part of the gastrointestinal tract except the colon. Among various adjectives used to describe duodenal diverticula are true, false, congenital, acquired, pseudo

primary and secondary. Actually two main types of diverticula are found in the duodenum namely the pseudo false or secondary diverticulum which results from constriction and deformity of the duodenum most often because of long standing benign duodenal ulcer and the true duodenal diverticulum. The former type is commonly found in the first portion of the duodenum, immediately below the pylorus, and is most often apparent along the superior or inferior border of the duodenum as an outpouching situated just proximal to a zone of narrowing. Such an abnormality is not a true diverticulum.

The incidence of true duodenal diverticula is variously reported in the literature to range from less than 1 per cent to almost 6 per cent, as detected on roentgenologic examination (Fig. 68). Most necropsy data indicate a definitely higher incidence which appears likely to represent a more accurate determination of incidence, since obviously not every diverticulum can be detected on roentgenologic examination, even by an expert roentgenologist. Careful studies at necropsy indicate an incidence ranging from 14 to 22 per cent.

Duodenal diverticula occur with equal frequency in both sexes. The largest number have been diagnosed in the sixth decade of life. They are uncommon prior to thirty years of age but have been found in the eighth or ninth decade of life.

Diverticula almost always are located along the mesenteric border of the duodenum. The reason for their occurrence in this region is commonly thought to be due to weakness in the muscular layer at the site where mesenteric vessels enter the intestinal wall. The wall of a duodenal diverticulum commonly consists of mucosa and muscularis mucosae; only seldom are other layers of the duodenal wall present. Diverticula vary in diameter from several millimeters to 5 cm. or even more. They are usually round but may be funnel shaped, bilobed or lobulated. Because of their location, they usually are imbedded in the pancreas, a fact which adds to the difficulty of their identification and treatment at the time of operation.

Most diverticula occur in the second portion of the duodenum reported series describe 56 to 85 per cent of them in this location. Thus it is not uncommon to find a diverticulum in direct relationship with the common bile duct or a pancreatic duct, a fact which must be kept in mind in the sur-

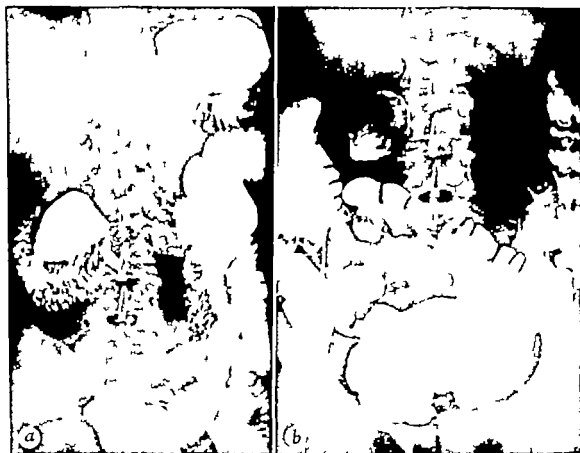


Figure 68 Roentgenogram revealing a duodenal diverticulum. a, Soon after ingestion of barium. b, Delayed film showing retention of barium in diverticulum.

gical treatment of such a lesion. Approximately 5 to 10 per cent of diverticula occur in the first portion of the duodenum and 20 to 30 per cent in the third portion. An occasional diverticulum has been reported in the fourth portion. Multiple diverticula occur in the duodenum in 15 to 30 per cent of cases, according to reports.

Usually pathologic changes are not associated with a diverticulum, however inflammatory changes, infection, ulceration, perforation, hemorrhage, calculi and tumors have been reported. If, as is usual, the diverticulum has a relatively wide mouth and there is no retention within the sac, it is likely that the mucosa lined cavity will evidence no pathologic change. If, on the other hand, the neck of the sac is small, retention may occur and infection, ulceration and associated changes may develop. A distended diverticulum may cause pressure on the biliary or pancreatic ducts and even obstruction of the duodenum has been reported, but all these complications are rare.

Most duodenal diverticula produce no symptoms. In the small percentage of patients in whom symptoms do occur they are

not necessarily pathognomonic of a diverticulum. It must be decided whether these symptoms actually are caused by the diverticulum or by some associated lesion, such as peptic ulcer or cholelithiasis, or a disorder such as an irritable bowel or functional dyspepsia. The differential diagnosis is not always easy but it is likely that the diverticulum should not often be incriminated. The most characteristic symptoms appear to be those of periumbilical pain, which may shift to the epigastrium or the right upper abdominal quadrant. The pain appears soon after meals and may be associated with a feeling of fullness or nausea. It may be relieved by belching or vomiting. Usually if these symptoms are caused by a diverticulum, the roentgenologist can demonstrate retention of barium in the diverticulum. Diarrhea or constipation and loss of weight also have been reported but are of little diagnostic value.

It is generally agreed that only the exceptional diverticulum requires surgical treatment, which includes probably less than 5 per cent of those discovered roentgenologically. In the large majority of patients,

this abnormality is an incidental finding of little if any clinical significance.

In a smaller group of patients there may be symptoms referable to the upper part of the gastrointestinal tract and careful investigation reveals no abnormal findings other than the diverticulum. The question then arises whether or not the diverticulum is the cause of the symptoms. Under these circumstances, appropriate medical treatment is recommended. This may consist of a bland diet, use of antispasmodics and alkalis and perhaps postural drainage or other procedures which appear to be indicated. Should the symptoms not be relieved, one then must determine if the evidence and symptoms appear to be sufficient to advise exploration with a view to operation on the diverticulum, if no other satisfactory explanation is found for the patient's symptoms.

In a recent study made at the Mayo Clinic, only thirty of 525 patients in whose records the clinician indicated the presence of duodenal diverticula underwent exploration of the upper part of the abdomen. Many duodenal diverticula incidentally discovered by the roentgenologist during this same time were not entered as diagnoses in the clinical records. In only eight of these 525 patients was duodenal diverticulum the main indication for exploration. Before deciding to proceed with an operation for duodenal diverticulum the physician not only must exclude organic disease elsewhere in the abdomen which might be responsible for the patient's symptoms and this may require careful and thorough investigation but he also must evaluate the magnitude and importance of the functional symptoms presented by many of these patients. Actually evaluation and exclusion of the latter group of symptoms are usually the most difficult part of arriving at a decision regarding indications for treatment.

A few patients who have duodenal diverticula are explored surgically in an effort to relieve disabling symptoms. Careful exploration is essential to determine if there may be any other possible explanation for the patient's symptoms. If such a condition is found, it should, of course, be corrected. In the absence of other findings and without evidence of considerable certainty that incriminates the diverticulum as the cause of trouble, it appears advisable that surgical attack on the diverticulum should be undertaken only when the anatomic findings are such that operation on the diverticulum can be accomplished with minimal risk to the

patient. Often this is not the case because of close association between the diverticulum and the biliary or pancreatic ducts or its complete inclusion in the pancreas.

There remains the exceptional patient in whom it appears definite from the history and clinical findings, such as retention of barium in the diverticulum, partial obstruction of the duodenum or pathologic changes in the diverticulum, that the diverticulum actually is the cause of the symptoms. Surgical treatment may be indicated for some of these patients.

Because of the common location of a diverticulum along the concave border of the duodenum the lesion usually is not apparent on initial exploration. Various procedures have been suggested to aid the surgeon to locate the diverticulum. Among these are previous ingestion of barium, which will present a whitish appearance through the wall of the diverticulum, injection of air into the duodenum to inflate the diverticulum and palpation of the neck of the sac with an exploring finger inserted into the duodenum through an appropriately placed incision for duodenotomy. Diverticula situated in the second, or descending, portion of the duodenum often are approached most satisfactorily by mobilizing the duodenum laterally and rotating it forward and to the left so that its mesenteric border is approached posteriorly. A diverticulum situated in the third, or transverse, portion of the duodenum requires mobilization of the retroperitoneal portion of the duodenum. Depending on whether the diverticulum is located in the proximal or distal portion of the third part of the duodenum it may be approached best from the right or the left. It may be more accessible by exposure through the gastocolic omentum or base of the transverse mesocolon.

When the diverticulum is located and exposed, one of various surgical procedures may be employed. Complete excision of the sac, with careful suture of its neck, is probably the procedure of choice when this can be accomplished without unwarranted trauma and hazard. Inversion of the sac into the lumen of the duodenum, with suture of the neck, may appear to be the treatment of choice in some subjects; however this may require as extensive dissection as does excision of the sac. Surgeons who have employed this method have not reported subsequent difficulty caused by protrusion of the sac into the duodenal lumen. In the exceptional case mere suture of the neck of

the diverticulum performed through a trans duodenal approach has been employed, although this would not often appear to be the most desirable treatment. Anastomosis between the diverticulum and the jejunum has been performed in a rare case but is probably seldom indicated. The choice of surgical procedure is influenced by the existence of significant pathologic changes within the diverticulum.

One always must be acutely mindful of important adjacent structures in any operation for duodenal diverticulum. Exposure and catheterization of the common bile duct usually are advisable prior to dissection of a diverticulum located in the region of the ampulla of Vater or extending into the vicinity of the choledochus. Likewise identification and protection of the pancreatic ducts are equally essential if these ducts are in close relationship with the diverticular sac. Acute pancreatitis, pancreatic necrosis or fistula may follow ill advised dissection and trauma to the pancreatic parenchyma.

Should direct operation on the diverticulum appear to be unduly hazardous, one or occasionally may consider some alternate procedure, such as duodenojejunostomy, low gastric resection with a Billroth II type of anastomosis and exclusion of the duodenum, or gastroenterostomy. Although complete and lasting relief of symptoms has been reported after operation for duodenal diverticulum, a favorable result is not always obtained.

Fistulae. Duodenal fistulae may be of two types: external, in which the duodenal contents drain to the outside, or internal, in which an abnormal communication is present between the duodenum and some adjacent structure.

Fortunately an external duodenal fistula which is always a serious or potentially serious lesion, is not seen as often today as it was in former years. It may result from trauma, such as a perforating wound, the unintentional application of a clamp to the duodenal wall during the process of removing an extensively diseased right kidney or other forms of trauma during the course of a difficult operation in the right upper quadrant of the abdomen. It may result from failure of a suture line in the duodenum to heal satisfactorily. An external duodenal fistula is seen most often after a gastric resection of the Billroth II type in which the closure of the duodenum does not hold and the stump does not heal properly. Many factors may contribute to leakage from a duodenal stump

after gastric resection: some of these are general in nature and others are local. A poor nutritive status, anemia, extensive neoplasm, serious inflammation and infection are systemic factors which may result in poor healing of a duodenal stump. The local factors include inadequate blood supply of the duodenal wall, insecure closure of the duodenal stump, excessive tension on the suture line or increased intraluminal pressure. It appears likely that the last-named factor is the most important one in the majority of cases. Increased pressure within the duodenal stump after gastric resection may result from some type of obstruction in the proximal jejunal limb or from obstruction where the proximal jejunal limb joins the stomach. Unrelieved gastric retention due to obstruction of the distal jejunal limb may also increase pressure within the duodenal stump. Obviously all factors, systemic as well as local, which might favor leakage from a duodenal stump should be assiduously avoided.

If leakage occurs from the duodenal stump after gastric resection, it most often takes place between the third and tenth postoperative day. If drains have not been left in place to permit ready access of duodenal contents to the exterior, severe pain develops in the right upper abdominal quadrant. This pain may become generalized over the entire abdomen and perhaps be referred to the right shoulder. Localized tenderness is pronounced and significant increase in temperature and in pulse rate is associated. Prompt treatment is indicated. Free external drainage must be established, the stomach kept empty by means of an indwelling tube and fluids and electrolytes replaced according to the losses and requirements of the individual patient. The abdominal wall must be protected from the digestive action of the duodenal contents. This last requirement may best be accomplished by insertion of an aspirating catheter that exerts constant mild suction in the drainage tract. Many different preparations have been suggested to protect the skin of the abdominal wall around a duodenal fistula. One such preparation that works well is Sebrecht's ointment, a small quantity of which may be prepared by mixing the following materials: bismuth subgallate, 4 gm., cottonseed oil, 25 cc., zinc oxide, 50 gm., starch, 50 gm., lanolin, 60 gm.

In addition to the necessary local measures in treatment of a duodenal fistula, general measures are also important. Antibiotic therapy is usually indicated. Every effort to

improve the general healing ability of the tissues must be made, including satisfactory replacement of fluids and electrolytes, blood transfusion and as high a caloric intake as possible, especially aimed toward increasing the intake of protein. Improvement in the patient's nutritive status occasionally may be best accomplished by administration of appropriate formulas through a small polythene tube which has been passed through the nose and on into the jejunum. Intravenous administration is, of course, necessary. Any attempt at early direct surgical closure of the fistula is not recommended, primarily because of the hazard and slight chance of success. Later efforts of this type may be advisable in some cases. In past years, the mortality rate associated with external duodenal fistulae after gastric resection has been high. This should not be so, as early recognition and prompt treatment, both local and general, should greatly reduce the risk of this complication. Obviously, it is most desirable to make every effort to avoid this complication.

Internal duodenal fistulae may be of various kinds. The commonest one occurs between the gallbladder and the duodenum, usually because of severe acute cholecystitis associated with stones and obstruction of the common bile duct or more often the cystic duct. Under these circumstances, the gallbladder becomes adherent to the duodenum, a zone of necrosis develops in the wall of

the gallbladder which then involves the duodenal wall in a similar process and a communication develops between these two structures with discharge of contents from the gallbladder into the duodenum. Such a fistula remains patent if biliary obstruction persists but may tend to heal if obstruction is relieved. It usually causes no direct symptoms despite the opportunity for reflux of duodenal contents into the gallbladder. It is usually discovered on plain roentgenographic examination of the abdomen when air is seen in some parts of the biliary tract. It also may become apparent on roentgenographic study after the ingestion of barium when this medium is seen to enter the gallbladder.

A somewhat less common type of fistula may occur in a similar manner between the common bile duct and the duodenum (Fig. 69). Rarely a duodenal ulcer may perforate into the common bile duct. Fistulae between the duodenum and the hepatic flexure of the colon or distal parts of the small intestine may result from a neoplastic process, usually arising in a structure other than the duodenum.

Other Duodenal Lesions. Odd and unusual lesions of the duodenum occasionally may be encountered. These include cysts, unusual adhesive bands, localized duodenitis, foreign bodies, cutaneous pancreaticoduodenal fistulae and functional disorders. Careful clinical investigation will help in the diagnosis in cases of this type and consultation among the roentgenologist, internist and surgeon should determine the course of treatment to be followed.

THE JEJUNUM AND ILEUM

Injuries of the Small Intestine. The surgeon may find himself in a dilemma concerning the possibility of injury to the small intestine, but in a large city hospital or an industrial region he may become expert in such a field. Even in a small community he occasionally will be called to deal with the problem because of today's frequent automobile accidents. Even though no penetrating or perforating wound is present, a concussion type of injury to the abdomen may result in serious trauma to the small intestine. In some cases this is obvious and the general systemic signs disclose the necessity for surgical intervention. In other cases it is only by repeated close observation that the surgeon can make his decision.

In a gunshot wound, stab wound, or wound resulting from violence with a per-



Figure 69 Roentgenogram revealing choledochoduodenal fistula, as evidenced by presence of opaque medium in the common duct after ingestion of barium by mouth.

forating type of instrument, examination may be easy and the conclusion may be reached quickly. The surgeon should not waste time by probing the wound, as he will not derive valuable information from this maneuver and merely may introduce more contamination. If the patient is seen in the early minutes after injury his general condition may be most misleading and the surgeon must not be lulled into a false feeling of security. It is impossible to single out the small intestine alone because of the possibility of injury to other neighboring viscera, with physical signs and physiologic disturbances based on these concomitant changes. In addition, perforation of the small intestine occasionally may be obscured by partial or complete protection of the point of perforation by another viscus. Injuries to the abdominal wall produced by blows with a blunt instrument, crash injuries or crushing injuries will be most difficult to evaluate and the surgeon may be hard put to distinguish between extraperitoneal and intraperitoneal changes. The important factor is that the surgeon recognize the possibility of injury to the small intestine and make the error on the side of early interference rather than too prolonged observation. It goes without saying that the general condition of the patient is to be evaluated as to shock, hemorrhage and gross sepsis.

Laboratory findings vary at times helpful findings may be numerous and at other times little or no aid is derived from laboratory studies. Determination of values for hemoglobin, erythrocytes, leukocytes and hematocrit may be of considerable help initially and of even more help if a period of observation is to be employed.

Röntgenologic findings may not be helpful at all in the early stages as the changes will not have had time to develop. The presence of perforation accompanied by free air is obvious. Distention produced by paralytic ileus may be a relatively late sign, but it should always be looked for. In general, results of roentgenologic study do not change the surgeon's decision but occasionally give him additional information.

The treatment for injury to the small intestine is surgical. The surgeon first must place the patient in the best possible condition and this may call for heroic measures as time is of such great importance. Shock can be treated efficiently today and the various types of fluids available for intravenous use all play a role. It is to be emphasized that no substitute exists for whole blood.

The passage of a gastric tube through the nose serves the dual purpose of emptying the stomach and obviating the factor of swallowed air later. In addition, the tube may be advanced manually through the upper portion of the small intestine at the time of operation should the surgeon feel this to be valuable.

The location of the incision may be dictated by local findings, but with the mobility of the small intestine it may not be as paramount as in other conditions. Small lacerations of the intestine may be sutured adequately; they must be made not only water-tight but airtight as well. Large lacerations, multiple lacerations and disturbances of the mesentery demand resection of the intestine. The surgeon first must determine the complete extent of injuries to the small intestine, as he may waste valuable time repairing or resecting one segment and then find that a nearby segment demands the same treatment, whereas repair of both segments could have been included in one procedure. In earlier years surgeons were forced to exteriorize the ends of the small intestine, but today this practice should be extremely rare indeed. Constant discharge of the highly irritating and digestive contents of the small intestine on the skin of the abdominal wall is something to be avoided if at all possible. The operation is not complete until thorough examination of all other abdominal viscera is carried out in an orderly fashion and the peritoneum is examined from within for small wounds or punctures which may have gone unnoticed.

In recent years there has been a tendency to lavage the peritoneal cavity thoroughly before closure of the wound. This is a revival of earlier teaching. Surgeons are divided in their opinion as to the effectiveness of this maneuver; some of them consider that septic material may be disseminated in this fashion. A few years ago powdered sulfonamide drugs were dusted about the abdominal cavity but this was largely discontinued because of the large dose that was administered at one time with irregular rates of absorption. It was difficult to know the exact level of drug obtained in the blood by this method, serious complications in the form of systemic reactions, plugging of the ureters with crystals of sulfonamide and other untoward phenomena led to general abandonment of the procedure. If injuries to other organs coexist and it is necessary to place drains in the abdominal cavity substantial loss of the introduced

antimicrobial substance occurs. Various antibiotic agents have been placed in the abdominal cavity; however it is now recognized that neomycin so administered may lead to respiratory failure. Thus, present opinion is that it is far better to administer properly measured doses of such agents in a parenteral fashion after operation.

When the patient is returned to bed after operation, there are many measures which the surgeon must keep in mind to bring about a satisfactory conclusion. The gastric tube should be attached to a suction device to maintain deflation of the upper part of the small intestine. This requires frequent attention, with checking of the suction and irrigation of the tube. Since nearly 70 per cent of the intestinal gas is swallowed air the prophylactic effectiveness of a properly functioning tube is obvious. The tube should be kept in place until peristalsis has resumed a normal pattern and the patient is expelling flatus. Proper attention to fluid balance, with supply of the indicated electrolytes, protein supplements and vitamins is rewarding. Blood transfusion is used as needed. Antibiotics are to be used liberally if a true indication exists. The surgeon will do well to be alert to the best possibilities for antibiotic support and above all to be alert to any possible serious sequelae of massive antibiotic therapy. With proper management, injuries to the small intestine may be controlled adequately with a generally good prognosis.

Infectious Diseases Involving the Small Intestine. Nonspecific infections. A vague and poorly defined group of symptoms, which, for lack of a better term, is often called "gastroenteritis" may cause confusion in differential diagnosis. Ordinarily a properly taken history elicits possible exciting causes of the disturbance and serves to differentiate the condition. The presence of a mild epidemic in a family, community or region may be of significance. Unusual dietary factors, travel in places where sanitation might be open to question, and similar variations from a normal routine are important information for the surgeon to seek. Surgically this poorly defined group rarely assumes much importance. The exact causal relationships of certain bacteria are open to some debate, as a variety of organisms can be recovered from the contents of the small intestine at different times. It is difficult to clarify why these organisms should be suspected of producing disease complexes at certain times and not at others.

Mesenteric adenitis is a fairly well-defined entity which has been recognized for many years. Its chief importance is its confusion with acute appendicitis. It is almost always at surgical exploration that the true nature of the condition is revealed. The symptoms are frequently those associated with acute appendicitis. The findings may be relatively mild, with tenderness over the abdomen, especially in the right lower quadrant. The presence of muscular spasm and pronounced localizing tenderness is unusual. It is difficult to explain why the findings should be more common on the right than on the left. The patient often exhibits mild fever and commonly is convalescent from an acute infection of the upper part of the respiratory tract. The patient often complains of nausea and occasionally vomiting; he is restless and may complain of "the grippe." At laparotomy the appendix appears normal, but a striking feature is the extreme enlargement and engorgement of the mesenteric lymph nodes. Occasionally these masses will be of such proportions as to suggest lymphosarcoma, but in mild forms of the disease only moderate enlargement of a few of the nodes may be noted. Biopsy of these nodes and bacteriologic studies invariably result in a report of hyperplastic lymph nodes without specific pathogenic bacteria. In early years the possibility of tuberculosis was considered and perhaps many patients were given that diagnosis without laboratory confirmation. The cause of mesenteric adenitis is unknown although many factors have been suggested.

It is considered proper to remove the appendix at the time of laparotomy as later confusion with appendicitis is thus obviated and it is considered entirely worth while to protect the patient by appendectomy. Recurrent attacks of mesenteric lymphadenitis have not been reported. It appears likely that recurrence is possible, but once the appendix has been removed the patient, or the parents, may never call the physician should a mild attack occur subsequently.

Specific infections. Earlier writings devoted considerable space to specific infections, in contrast to current articles, in which only passing mention of them is made. In a modern hospital in the United States it is extremely rare for the surgeon to be called on to assist in a problem of this type. In earlier years the reverse was true.

Typhoid fever characteristically strikes the lymphoid follicles in the small intestine, especially in the terminal portion of the

ileum Peyer's patches become the site of extreme ulceration, thus in former years surgeons occasionally observed general peritonitis on this basis. Laparotomy was necessary at times in desperately ill patients when perforation occurred, it is a matter of record that many of these patients recovered surprisingly well. At present effective prophylactic and therapeutic measures are so general that the modern surgeon almost has forgotten typhoid fever. However with travel made increasingly easy throughout the world, he would do well to keep it in mind.

Tuberculosis is another condition which was found to strike the small intestine on occasion. One suspects that at present the establishment of the diagnosis of tuberculosis is a much more scientific process than it was formerly and that many nonspecific granulomatous lesions of the intestine in past years were labeled tuberculosis. However intestinal tuberculosis is still seen from time to time and the surgeon may be called on to resect the intestine because of the great obstruction produced by the contracture of the scarred tissues. The presence of pulmonary tuberculosis in these patients complicates the nursing care and the public health problems involved. Fortunately true tuberculosis of the intestine is extremely rare today.

Systemic Diseases and Their Relation to the Small Intestine. With increasing knowledge in roentgenologic techniques for study of the small intestine the roentgenologist has become expert in detecting conditions which went unrecognized in earlier years. *Vitamin deficiencies* especially those of the vitamin B complex, may occur with wasting diseases or faulty nutrition. The roentgenologist points out that he frequently can recognize such deficiency because the normal motility of the small intestine is greatly reduced. During fluoroscopy he notices that the small intestine gathers its content of barium in irregular pools which is a characteristic change. This is of importance to the surgeon, as the findings elicited and the symptoms presented by the patient might lead him to advise surgical intervention when the correct therapy would be entirely medical. Perhaps even more serious would be the error of failing to recognize the condition should it be complicating some other surgical lesion. The surgeon would do well to pay strict attention to the nutritional needs of the patient if time permits.

It is also probable that the small intestine may be involved secondarily in any *debilitat-*

ing systemic disease of prolonged duration and also in conditions associated with complete lack of free hydrochloric acid in the gastric contents because of the great speed with which the stomach empties into the small intestine.

Parasitic diseases may produce changes in the small intestine their presence occasionally is disclosed by studies of blood smears as well as by systemic reactions. Of importance to the surgeon is the knowledge that *Ascaris lumbricoides* the common round-worm, can produce a great degree of intestinal obstruction and he may be called on to relieve this condition surgically.

Although not exactly a systemic disease and admittedly rare *gallstones* ileus should be mentioned. A previous history of cholelithiasis may be helpful, but it is not essential as "silent" gallstones may have been present. In this condition intermittent severe ileus may puzzle the surgeon and the true nature of the lesion may be disclosed only at laparotomy.

Regional Enteritis. In 1932 Crohn and associates presented the first clear description of this disease. They pointed out that isolated cases similar to theirs had been reported for many years. The condition also has become known as "terminal ileitis" or "regional ileitis" with other similar pseudonyms being applied on occasions. It is a matter of some conjecture at present whether or not the incidence of this disease actually has increased sharply since 1932. It might be that tuberculosis was the diagnosis formerly assigned in many cases but careful scrutiny of the literature suggests that an actual great increase in incidence may have occurred.

The changes are located characteristically in the terminal portion of the ileum. However any part of the small intestine may be involved and we have observed cases in which the duodenum was the zone of primary involvement. One of the most ominous developments is the intermittent involvement of intestine in so-called skip areas, which indicates that the disease is of such a fulminating character that little of curative nature can be done for the patient. In chronic disease the changes in the terminal reaches of the ileum may spread into the right portion of the colon, which probably gave rise to confusion with tuberculosis in earlier years. A mass usually is present because of the granulomatous changes. The disease starts in the mucosa and, in its early stages, may be overlooked by the surgeon.

who examines the small intestine in a casual way. Later the thickening of the intestinal wall is extreme and the mesentery becomes tremendously broadened and infiltrated. The lymph nodes are greatly hypertrophied, which leads to considerable difficulty at the time of resection. Strictures of the intestine are commonplace and almost equally common is the formation of fistulae. These fistulae frequently extend to the cutaneous surface, as an unwary surgeon previously may have performed appendectomy because of a mistaken diagnosis. Fistulae involving other loops of small intestine the colon and other organs, such as the urinary bladder are common. The disease may be limited sharply to one region of the intestine but the surgeon may have difficulty in determining just how far proximal the disease process actually extends on the mucosal side. Secondary changes are detected in the proximal part of the small intestine when constriction of the diseased segment has produced chronic obstruction, so that the proximal portion of the intestine is dilated and thick walled.

In a classic case of regional enteritis the history is one of chronic disease. An acute form probably exists, but frequently a neophyte surgeon operates with a provisional diagnosis of appendicitis, finds a normal appendix and, after prolonged search, decides that the terminal portion of the ileum is a bit edematous and indexes the patient as suffering from "acute enteritis." This probably accounts for earlier reports of spontaneous cure and complete recovery without sequelae. True regional enteritis probably is irreversible and a properly taken history reveals that the patient has had difficulty for some time. Proper care should eliminate the necessity for emergency operations on these patients and thus sharply reduce the number of appendectomies done erroneously in this disease, with their associated high incidence of fistulae. The patient frequently is of Jewish extraction and often is in the second or third decade of life. However patients of any race or age theoretically may be involved. Evidence of chronicity frequently is obvious. When the disease is in its advanced stages, the patient is somewhat emaciated and complains of diarrhea, cramps attacks of intestinal obstruction, chronic loss of weight, anemia, repeated bouts of fever and, occasionally hemorrhage. Formation of fistulae may be an important factor and, in addition to those mentioned above the experienced proctologist may recognize the presence of perineal fistulae, which for some

reason are prone to appear in regional enteritis.

The diagnosis of regional enteritis is often suspected from the findings already mentioned. Routine hematologic studies disclose anemia in a large percentage of the subjects. Studies of stools eliminate parasitic infestations from the differential consideration. The presence of a mass is frequently reported and the patient may have been able to palpate the mass in the right lower quadrant for some years. He may state that during obstructive episodes the mass has ballooned up to a point at which it is easily visible on inspection of the abdominal wall. However the ultimate diagnosis rests with the roentgenologist. The characteristic picture is that of the "string sign" caused by the pronounced constriction of a long segment of the intestine. The experienced roentgenologist can make the diagnosis with a great degree of accuracy.

The primary treatment of this condition is medical and every effort should be made to treat the patient in a conservative fashion. A bland diet free of foods high in residue should be the rule. Use of vitamin concentrates in liberal quantities helps to maintain a better nutritional state. Iron may be indicated to combat anemia. During periods of stress when diarrhea may be a large factor codeine and paregoric may be used in liberal doses. Chemical agents, such as the sulfonamides, have been employed in large doses. The results have not been striking, but some workers consider that prolonged courses of sulfonamides may be of some benefit. All antibiotics developed up to the present writing have been entirely without effect on the disease itself, but secondary infections may respond to their use. With the advent of corticotropin (ACTH) and cortisone, it was only natural that these steroids should be tried in this disease. Opinion is somewhat divided, but reliable authorities have condemned use of these hormones in regional enteritis, as perforation of the lesion has been known to occur and severe hemorrhage has been reported as a result of their administration. Beneficial effects of these hormones have not been demonstrated in regional enteritis, aside from temporary production of a sense of well-being.

The surgeon will be called on to take over these cases when medical management does not suffice. Intestinal obstruction produced by the constriction demands surgical relief. Formation of a fistula is an obvious indication for surgical correction. Although most

of the patients are somewhat anemic hemorrhage may not be a major factor if it is however operation is to be seriously considered. The presence of an abdominal mass is not in itself a clear-cut indication but it often demonstrates that a sufficient degree of obstruction exists to place the patient in a surgical category.

The type of operation is subject to some debate at present. In earlier years complete resection of all diseased intestine along with removal of long segments of normal intestine in each direction with corresponding mesentery was considered compulsory. A relatively high mortality rate soon demonstrated that a better method should be sought. In those patients in whom the lesion was considered inoperable because of extreme inflammatory changes, with attachment of the intestine to adjacent viscera and vital structures it was found that short-circuiting or diverting types of operation had surprisingly good, even if temporary, effects. It was learned that a side-to-side ileocolostomy had little or no effect, but it is now common knowledge that excellent results may be obtained if the normal ileum above the diseased segment is divided completely and ileocolostomy is then done. Formerly it was considered obligatory to reopen the abdomen at a later date when the inflammatory change had subsided, and remove the diseased intestine. This is no longer the rule as those patients who are progressing satisfactorily after a diversionary ileocolostomy may well be treated without later resection. In fact, some surgeons today consider that resection is not a good policy and leave the diseased intestine in place in most instances.

If the lesion is amenable to surgical treatment and is removable without the sacrifice of surrounding vital structures, it should be resected in a one-stage operation. If the operability is in question, we leave the diseased segment in place and short-circuit it by ileocolostomy after dividing the ileum. The patient is observed closely for several years to determine whether or not further operation is indicated.

With the widespread adoption of vagotomy for lesions of obscure cause such as duodenal ulcer it was only a matter of time before vagotomy was tried for ulcerative colitis and later for regional enteritis. The rationale for this operation in the management of enteritis remains obscure. Surgeons were reluctant to adopt it when they observed diarrhea in patients who had been submitted to vagotomy for peptic ulcer hesi-

tating to make diarrhea even worse in patients with regional enteritis. Roentgen ray therapy has been administered to a few patients especially those exhibiting recurrence after surgical resection. Not enough sound information has accumulated on the results to warrant more than its mention.

Enough patients with regional enteritis have been operated on and followed for sufficiently long periods for physicians to realize that the disease is fraught with a high rate of recurrence. Even the most radical operation has been known to fail to eliminate the disease in certain patients if a sufficiently long follow up period has been observed. This had led some surgeons at present to become more conservative in their treatment. As already indicated, instead of putting the patient to the risk of an extremely radical resection, they are more prone to carry out conservative resections or even do mere ileocolostomy without an attempt at removal of the lesion. Complete removal probably is still the best choice, however. Recurrence at the site of anastomosis may be treated by further resection if indicated, and we consider that the development of "skip areas" in proximal segments some years after resection need not condemn the operation. The surgeon is well advised to look on regional enteritis as an extremely serious situation, since all manner of treatment may fail in more than 30 per cent of cases. Van Patter and associates have demonstrated that widespread optimism after any treatment is scarcely justified.

Tumors of the Small Intestine. Tumors of the small intestine are not common. Benign tumors (Fig. 70) include lipomas, myomas, fibromas, aberrant pancreas polyps and hemangiomas. Many of them arise on the mucosal surface and it is only after prolonged growth that they give rise to symptoms. It is strange that the leiomyomas noted so frequently in the stomach occur so rarely in the small intestine.

Malignant tumors of the small intestine fortunately are rare. They usually are divided into three general groups namely adenocarcinoma, carcinoid and sarcoma. Primary adenocarcinoma has all the characteristics of this tumor seen in other glandular viscera (Fig. 71). Some of the patients with carcinoma of the small intestine are surprisingly young. The lesion is a vicious one, as diagnosis may be late, and widespread metastasis may be noted at the time the diagnosis is finally established.

Carcinoid of the small intestine is similar

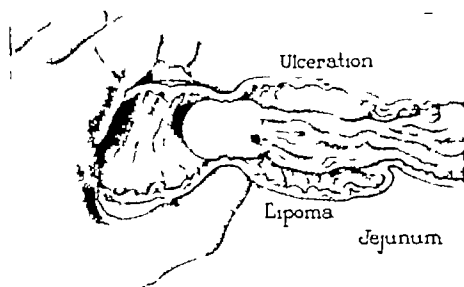


Figure 70 Benign tumor of small intestine with obstruction and bleeding.

histologically to the same type of tumor more commonly located in the vermiform appendix. Carcinoids have been reported in the rectum and at other points in the intestinal tract. Carcinoid has been likened histologically to basal cell carcinoma. Many authorities have pointed out the argentaffin relationships exhibited and considerable debate persists as to the developmental background of these tumors. Frequently they present themselves as small submucosal growths which are characteristically yellow on cut section. They may extend around the circumference of the intestine to produce symptoms of obstruction. Numerous carcinoids often are scattered along the small intestine, but more commonly they are found in the distal part of the ileum. In contrast to the carcinoid of the appendix, that of the small intestine produces metastatic lesions rather commonly not only in the regional lymph nodes but also in the liver. Hence the latter lesion is usually considered to be far more malignant than is the appendiceal tumor.

By far the commonest type of sarcoma encountered in the small intestine is lymphosarcoma. Studies of age incidence show that this tumor occurs in considerably younger patients than do other malignant tumors. Frequently it occurs as part of widespread disease so that other abdominal masses are much more striking. Occasionally other types of sarcoma may be seen and a differentiation is made only by the experienced pathologist.

The symptoms of tumors of the small intestine may be vague, so that considerable valuable time may be lost before a diagnosis is definite. As expected, symptoms of obstruc-

tion may predominate, with an intermittent character to the syndrome since intussusception plays such a great part. Once the intussusception is reduced spontaneously the patient may experience a long interval of relative comfort. The triad ascribed to obstruction of the small intestine, namely intestinal colic, vomiting and distention, applies in advanced lesions. Melena is variable, depending entirely on the type of tumor. Frequently the patient may be able to feel a mass through the abdominal wall, but this may vary considerably with peristaltic activity and the examining surgeon should not wait for such a finding if other factors are present.

The history may serve to confuse the sur-



Figure 71 Primary adenocarcinoma of small intestine.

geon but at times it may be most suggestive. Chronic intestinal obstruction often is the rule. The patient usually has lost considerable weight and may be emaciated. If definite severe obstruction is present, the usual roentgenologic findings are noted. If obstruction is not a dangerous feature, or is not imminent, the roentgenologist may employ barium and may be able to distinguish several varieties of tumor of the small intestine. This is a difficult field of roentgenologic diagnosis; however, and the surgeon should be satisfied if the roentgenologist merely verifies the presence of an indeterminate lesion of the small intestine. Anemia may be severe owing to chronic loss of blood and to serious derangement in the nutritional process. The anemia is often macrocytic so that confusion may have been part of the picture early in the course when the possibility of a primary anemia might have been considered. The final decision rests with the surgeon, however, as the true nature of the lesion is rarely disclosed before laparotomy. A carefully elicited history, combined with certain cardinal physical and laboratory findings in the presence of an indeterminate roentgenologic report, leads the surgeon to explore the abdomen with good reason.

Resection of the intestine is indicated in the majority of benign tumors of the small intestine. Enterotomy with local excision of the tumor may suffice if one is satisfied that the resulting lumen at the point of repair is adequate. Frequently the surgeon accepts the risk of resection even in such a patient because of the possibility of postoperative obstruction otherwise. Radical resection should be done in malignant tumors with removal of a large segment of mesentery. The method of anastomosis varies with the ideas of the surgeon but an end-to-end union is satisfactory in most cases. Great discrepancy between the lumina of the two loops to be connected may lead to some modification of this anastomosis.

The prognosis depends entirely on the nature of the lesion. Benign tumors are permanently eradicated, but the outlook in malignant tumors is ordinarily rather poor. Distant spread often is present at the time of operation and palliation is all the surgeon can offer. The extremely malignant nature of some of these tumors means early recurrence in spite of what appears to be a very radical approach. Postoperative roentgen ray therapy is of great help in lymphosarcoma but in few if any of the other lesions. Even in

lymphosarcoma, the long term results are disappointing.

THE APPENDIX

Appendicitis. Appendicitis is one of the commonest surgical lesions in the civilized world today. It is rather amazing to reflect on the length of time that was required before this disease was recognized fully and its seemingly simple treatment was accepted generally. It was not until 1886 that Reginald Fitz recognized the entity completely and it was through his publications and enthusiastic discussions that the medical profession at long last became aware of the true nature of appendicitis.

The gross appearance of the vermiform appendix is variable and its length appears to be of little significance. It is located at the tip of the cecum at the point of convergence of the taeniae, which at times is of convenience in locating it surgically. Much has been written about the possible positions of the appendix and even the most experienced surgeon may encounter difficulty in locating it, especially during a severe attack of appendicitis. Thus the surgeon must remember that almost any location of the appendix in the general cecal region is possible, so that he may have to search down into the pelvis, along the great vessels of the abdomen, medially toward the sacrum, posteriorly or actually among coils of small intestine. The appendix frequently is found in a retrocecal position and often appears to be retroperitoneal also. This only serves further to confuse the surgeon and at times makes proper recognition and removal a most difficult task.

Acute appendicitis may make its appearance at almost any age from the neonatal period to the period of most advanced senility. It appears to be more common, however, during the second and third decades of life. Most authorities state that it predominates in the male sex. It is most common among so-called civilized peoples. It has been suggested that dietary habits, rugged living associated with constant physical exercise, and bowel habits all have something to do with this incidence. People living in cities apparently are more commonly afflicted than are rural residents. Certain races such as those in the Far East, appear to be relatively free of this disease.

The exact cause is largely unknown. Experimental appendicitis in laboratory animals cannot be classed as closely similar to naturally occurring disease in human beings.

and the difference in dietary and living habits is so great that an abnormal situation obviously obtains. Since the appendix represents a sac with only one opening, authorities agree that the "blind loop" principle may be a factor. It appears that obstruction to the outflow of mucus and other contents of the organ is important. Indeed, in many patients with appendicitis, extremely hard fecaliths can be demonstrated in the lumen of the specimen. However other specimens may contain multiple fecaliths with no evidence of any inflammatory change. It is difficult to assess the role of the bacteria within the intestinal tract since myriads of bacteria which do not appear to have much to do with this disease are constantly present. It appears, theoretically, that an inflammatory process would ensue if the mucosa of the organ suffers a rent, allowing a direct attack on the submucosal elements by the bacteria. Recurring bouts of appendicitis would be expected to lead to more serious attacks, since the resultant gradual occlusion of the ostium should further distend the remainder of the appendix. Hunking of the appendix by postoperative or postinflammatory adhesions should have much the same effect.

As already indicated, bacteriologic culture of the contents of appendices has yielded a multitude of organisms. One would expect to find *Escherichia coli* because of its natural habitat in the vicinity. Cultures in patients having appendicitis with perforation usually reveal several strains of streptococcus also. At present, routine culture in any patient in whom fluid or pus is found is considered valuable because of the more or less specific antibacterial agents now available. Many patients who have severe acute appendicitis exhibit cloudy fluid in the right lower quadrant which yields no organisms on culture. The surgeon can estimate the severity of the changes by observation of the fluid, especially by noting its odor. The familiar smell of an infection produced by the gram-negative bacilli commonly found in the colon is an ominous sign. Mixed infection is the rule in a gangrenous process with rupture.

The location of the appendix is important in determining what changes are exhibited during an inflammatory episode. It is convenient to think of appendicitis as starting as a simple inflammatory change of the so-called catarrhal variety involving the mucosa. This spreads to include all layers of the appendix. If allowed to go unchecked, the inflammatory change proceeds to gangrene and still further progression results in

perforation. Often the perforation is walled off by surrounding structures such as the small intestine mesoappendix, large intestine and omentum. If the process of walling off has not been effective perforation leads early to generalized peritonitis. A word of caution should be sounded in that use of modern antibiotics may alter the inflammatory process considerably so that it may not follow any typical pattern.

The clinical picture in appendicitis adheres to no hard and fast pattern and even the most experienced surgeon must remember that he may be misled from time to time. Because the location of the appendix is so variable, appendicitis may simulate many other intra abdominal lesions. The various stages of inflammatory change may confuse the picture, so that the only important consideration is to remember that the appendix may be inflamed and, on the basis of possibility alone, this diagnosis should be kept uppermost in mind.

The patient may note certain *prodromal features* which are gone by the time a surgeon is consulted. A feeling of malaise followed by nausea, vomiting and general restlessness, often initiates the syndrome. These are such common symptoms that "gastroenteritis" may be the diagnosis and the patient and his family try to recall some dietary indiscretion leading to the temporary derangement. Abdominal pain may be relatively severe early in the course of the disease but may be so generalized that nobody thinks of the appendix. Frequently the patient states that the pain later centered about the umbilicus this occurs so constantly that it bears repetition. Often at this time the symptoms appear to decrease in severity so that the surgeon may be caught off guard. Frequently however the pain shifts downward and to the right and although the nausea and vomiting, which may have been severe, now may tend to abate the pain becomes the outstanding symptom. It may be colicky at first, but soon it becomes more constant in nature so that even the most uninitiated now thinks about the appendix. The patient assumes a position which gives maximal comfort, characteristically lying with the right leg drawn up and the knee flexed, and may refuse to straighten the leg.

At this point examination of the abdomen often is suggestive, as *tenderness* is present over the entire right lower quadrant, perhaps most pronounced at McBurney's point. However in view of the variable location of

the appendix, this finding may not be at all reliable. The contact of the inflamed appendix with peritoneum is the exciting factor as far as tenderness and muscular spasm are concerned, but should the appendix be buried in other viscera these signs may be absent. One never must neglect to carry out rectal examination and also pelvic examination in the female as tenderness occasionally may be detected only by these maneuvers. The retrocecal appendix may lie directly on the right ureter and it is possible to have urinary symptoms secondary to this.

Fever is an unreliable sign. Gastroenteritis, or any similar upset, may be accompanied by fever especially if vomiting is a factor and if dehydration is present. An increased pulse rate is too general a phenomenon to be ascribed to this disease. The restless patient who is experiencing more and more intense pain naturally has a fast pulse. However a sustained rapid pulse especially with an increasing rate, serves to stimulate the observer to reach a conclusion more quickly. *Leukocytosis* is always to be looked for routinely in attempting to make the diagnosis but its importance may have been overstressed in the past. Intercurrent disease may be present which tends to increase the leukocyte count, but at times this determination is helpful. Many surgeons have seen severe appendicitis without much increase in the number of leukocytes. However it is consistent to observe not only an increase in the total leukocyte count but a pronounced shift in their distribution, so that polymorphonuclear leukocytes predominate and evidence of an acute and overwhelming infection is clear cut. A blood count may be superfluous in a classic case of acute appendicitis. However the clinical syndrome of appendicitis under present conditions may be confusing, with the widespread use of antibiotics, the process may be so thoroughly masked that the surgeon is hard put to make a decision.

It is obvious that the use of any form of catharsis during an illness which could have any possibility of being appendicitis is the grossest possible error. Rupture of the appendix has been observed far too frequently after the use of catharsis in such instances. Likewise, the administration of narcotics is looked on as poor practice as the masking of the syndrome may make most difficult the decision regarding definitive care.

Several diseases may present symptoms which resemble those of appendicitis so

closely that differential diagnosis may be difficult.

Acute gastroenteritis is a poorly defined process and the term is used to include poorly understood syndromes. Dietary indiscretions mild epidemic forms of diarrhea and vomiting and undue reactions to noxious substances are frequently classed in this manner. This is especially true if the patient is traveling in tropical or other countries which are radically different from his own with regard to food and drink. The prodromal symptoms of acute appendicitis may be practically identical to those of severe gastroenteritis and it is only by frequently repeated observation that the true nature of the condition will be disclosed. The development of definite localizing tenderness over the appendiceal region is the most important single sign and takes precedence over almost any other feature. Occasionally the two conditions may coexist, at which time it requires considerable courage for the perplexed surgeon to advise appendectomy in the face of what appears to be a severe medical problem. Ordinarily it is not too difficult to make the differentiation over a period of hours.

The differential diagnosis between appendicitis and stones in the urinary tract may be most difficult. Even though the stone may be lodged in the renal pelvis the referred pain may be in the appendiceal region, so that considerable confusion arises. The wise surgeon obtains scout films of the region of the kidney ureter and bladder as well as excretory urograms if necessary. These studies occasionally save a patient from undergoing an unnecessary operation.

It is not common to mistake appendicitis for *pyelitis* or *cystitis* but it has occurred. The general reaction associated with these infections may produce a somewhat similar picture. Fever, chills, leukocytosis, abdominal pain and tenderness may confuse the issue. Ordinarily the differentiation can be made on physical examination, coupled with careful analysis of the urine. An inflamed appendix lying directly on the right ureter can produce not only urinary symptoms but also erythrocytes and leukocytes in the urine.

Many surgeons have made the mistake of operating for appendicitis only to discover peritonitis due to a perforated duodenal ulcer. This confusion probably results from the course described by the duodenal contents after leaving the perforated duodenum and passing along the right lateral abdominal

fossa. Usually a past history of peptic ulcer helps the surgeon to decide and if the signs of appendicitis are present the one who considers the possibility of a perforated ulcer obtains scout films of the abdomen to search for free air under the diaphragm. Since the treatment of both conditions is surgical confusion of these two entities may not be too serious a mistake but the location of the incision, of course is different.

Difficulty occasionally occurs in distinguishing between acute inflammation of the gallbladder and of the appendix. If the gall bladder lies low in the abdomen this differentiation may be almost impossible. Conversely an acutely inflamed appendix high in the abdomen may be misleading. Past history of disease of the gallbladder is most helpful. Frequently acute gallbladder colic subsides in a relatively short period. The extension of cholecystic pain to the back and the scapula may be of great importance in making a diagnosis.

Pelvic conditions do not play the prominent role they did in the days before the availability of complete antibiotic and chemotherapeutic programs. For a time acute salpingitis defied even the most alert surgeon when it came to distinguishing it from appendicitis. The history is usually unreliable and the common factors of the two diseases, such as fever leukocytosis and gastrointestinal symptoms, merely serve to cloud the issue. Pelvic examination often helps since motion of the uterus produces extreme tenderness in pelvic infection and the tenderness usually is bilateral. The presence of gonococci in vaginal smears is suggestive but not conclusive evidence in attempting to rule out appendicitis. It is now uncommon for surgeons to explore the abdomen for appendicitis and find salpingitis to be the true diagnosis. The so-called *twisted ovarian cyst* remains a relatively common phenomenon and gives rise to many local signs suggesting appendicitis. Often the history and pelvic findings serve in the differential diagnosis, but occasionally laparotomy is the deciding factor. Since operative treatment is indicated in either event and the location of the incision is similar no great mistake is made. More rarely a pedunculated uterine fibroid may become twisted on its pedicle and produce a similar clinical picture. Another gynecologic condition which might suggest appendicitis is rupture of a graafian follicle. This normally appears about midway in the menstrual cycle and the pain associated with it is commonly referred to as

"mittelschmerz." Pain fever and leukocytosis identical with the findings in appendicitis may be present. A similar picture is occasionally observed when a corpus luteum ruptures at about the time of onset of the menses.

Many other abdominal conditions may produce syndromes suggesting appendicitis. These are almost too numerous to list but the chief factor is to keep the possibility of appendicitis in mind and to be alert for variations. *Mesenteric adenitis* may be the underlying cause of a syndrome similar to that of appendicitis. The patient's symptoms, physical findings and laboratory data may be identical with those of the patient having appendicitis. Frequently a recent respiratory infection may be helpful in suggesting to the surgeon that lymphadenitis is the true diagnosis. However it is only at operation that the distinction can be made and as already pointed out, it is considered entirely proper to remove what appears to be a normal appendix in such a condition, thus ridding the patient of the likelihood of further difficulty. For the sake of completeness, one must include acute inflammation of *Meckel's diverticulum*. The picture can be exactly that of appendicitis; in fact, if the surgeon is removing what appears to be a relatively innocuous appendix, he should trace the small intestine for some distance searching for Meckel's diverticulum inflammation of which may have given rise to the clinical signs. Diverticulitis of the sigmoid, ruptured ectopic pregnancy and acute hemorrhagic pancreatitis are all conditions which can be mistaken for appendicitis.

Abscess formation may be a complication of appendicitis. The abscess may depend on the location of the appendix, but occasionally one sees an abscess of alarming proportions whose walls are made up of viscera at some distance from the appendix. It may be impossible to state where a given abdominal abscess arose since the fallopian tubes, the sigmoid and other organs within the abdomen may be the original source of the abscess and the findings may be identical.

With escape of noxious intestinal contents into the peritoneal cavity *peritonitis* is the inevitable result. If Nature is successful, the peritonitis is localized so that a small abscess may eventuate. A dreaded complication, however is one of widespread abdominal contamination. In this situation, the patient is in extremely serious straits with generalized abdominal tenderness and rigidity. The drawn, anxious appearance of the face

the pronounced abdominal distention and the complete lack of normal peristalsis make the dire situation evident to all examiners. This complication was common in earlier years and accounted for the high mortality rate.

One of the commonest causes of subphrenic abscess is gangrenous appendicitis with rupture. If the appendix is in a retrocecal position, the pus can burrow up behind the right portion of the colon to contaminate the entire subphrenic space. The possibility of a subphrenic abscess should be considered in any patient who is not recovering properly after prompt management of a ruptured appendix. If one waits until the roentgenologic findings are definite valuable time will have been lost. If subphrenic abscess is kept in mind and is sought surgically at an early date, dire consequences can be averted.

In earlier years *pylephlebitis* was almost uniformly fatal. Its presence should be suspected when a patient with appendicitis especially of the gangrenous variety later experiences shaking chills and fever of the septic type. When suppurative thrombosis develops within the portal vein the liver enlarges and the chills and fever continue unabated, frequently intensifying as time goes on. Jaundice becomes evident within a matter of days. Fortunately the modern therapeutic adjuncts to surgical care have served to reduce sharply the incidence of *pylephlebitis*.

The surgeon is frequently surprised when operating at some interval after rupture of an appendix to note the paucity of adhesions within the abdomen. However it is always possible that extensive adhesive bands will be the sequel after such a septic change, especially if several operations have been necessary. Many months or even years after operation, such patients may exhibit signs of definite obstruction requiring surgical relief.

The only treatment for acute appendicitis is surgical intervention. This disease presents a true surgical emergency and nothing but disaster will accompany delay. In general, several varieties of appendicitis may be encountered. The surgeon must decide for himself which type is present. Experience is the greatest aid in such a decision. In acute appendicitis without gangrene and rupture immediate surgical removal of the appendix is the established practice and produces uniformly excellent results. A second type of appendiceal disease is encountered when a definite abscess has formed and surgical con-

sultation has been sought late. A difference of opinion has existed in the past as to the proper management of such patients. For some years it was considered good practice to hospitalize the patient and keep him in bed until the abscess drained itself spontaneously, which it did in a great number of patients by bursting into the colon. Occasionally it pointed through the skin of the abdominal wall and ruptured externally. A change has occurred in surgical thinking which appears to be much better from the patient's standpoint. If the patient can be put into reasonable physical condition by use of the adjuncts now available, surgical drainage of the abscess is indicated. Often the appendix may be removed at the same time but should this entail considerable dissection, with the threat of injury to surrounding structures, such removal should be postponed. Two months should be allowed to elapse and, if the patient is in good condition at the end of that time, a second operation is in order to remove the appendix. It may be surprising, indeed, to note what an extreme local change has been brought about during that time and the appendix usually can be removed with ease. It is considered compulsory eventually to remove the appendix of these patients because repeated rupture is not at all rare.

The third type of appendiceal disease is that in which *general peritonitis* already is present. It is in such cases that the ingenuity and judgment of the surgeon may be taxed to the limit. He will do well to pool his knowledge with that of other observers so that the proper time for operation may be selected. Evidence of spreading peritonitis demands immediate intervention. In former years, evidence that the infection was being localized usually meant that the surgeon should allow more time to elapse. With the present help from all possible sides the surgeon has considerably more latitude and can operate under more nearly ideal conditions so that the prognosis for the patient is considerably better than it was previously. He may remove the appendix of such patients and adhere to those general criteria which guarantee the safest possible course.

In simple acute appendicitis, no elaborate preparation will be necessary and early operation will be the only feature. When the disease is in a more advanced stage, the surgeon can take a few steps to assure more nearly optimal conditions. Dehydration may be overcome quickly by intravenous administration of properly selected.

cal studies of the blood indicate what elements should be replaced. Lavage of the stomach is easily accomplished and may obviate the sequelae of tracheal aspiration of gastric contents which otherwise might occur should the patient vomit during or after anesthesia. The gastric tube may be left in place so that suction applied to it will be of considerable help in the removal of swallowed air and gastric secretion early in the postoperative period.

It was formerly common to insert anti-microbial substances into the peritoneal cavity at the time of operation. This procedure largely has been abandoned, as the dose is uncertain and rapid absorption might ensue with dire consequences. It now appears better to avoid intraperitoneal medication and give proper postoperative doses of the anti-microbial substances parenterally. The question arises concerning establishment of drainage in these subjects. Obviously no reason exists for drainage in acute, uncomplicated appendicitis. When a well recognized abscess is present, with definite collection of pus, adequate drainage must be established. Several types of ingenious drains are available to which suction may be attached, their use has facilitated the care of these patients. The real question arises in general peritonitis. In such patients, it early became the rule in many medical centers to close the wound tightly without drainage. Occasionally peritonitis went on to fatal issue because drainage had not been established. Many authorities point out that in general peritonitis it appears rather futile merely to place several drains in the right lower quadrant, as they hardly could be expected to accomplish a great deal. The use of several soft rubber tubes for several days would appear to be harmless, but considerable question is now raised as to their effectiveness. More recently some authorities have argued for tight closure of the peritoneum and fascia, a drain being placed just at the level of the fascia and brought out of the wound.

In patients with simple, uncomplicated cases, postoperative care may be no problem whatsoever. The patient is allowed to get out of bed as soon as he wishes, peristalsis soon resumes its normal action so that alimentation returns to normal at an early date and the patient is dismissed within a matter of several days. In the presence of abscess, however, considerable attention must be directed to the wound and the proper care of the abdominal wall. Ju-

dicious use of properly selected antibiotics based on sensitivity tests carried out on organisms obtained by culture of material taken at the time of operation speeds the healing process. However when the abscess is well walled off and has been drained properly use of antibiotics actually may be superfluous. In such patients the establishment of normal alimentation depends entirely on the resumption of normal peristalsis. The condition of the wound dictates the activities of the patient and the proper course to follow.

Postoperative care in general peritonitis serves as a challenge to the surgeon. The importance of fluid balance already has been stressed and this must be maintained in detailed and conscientious fashion. Repeated transfusion of blood may spell the difference between success and failure. The maintenance of proper suction on the indwelling gastric tube is most important. In the face of severe paralytic ileus, it may be difficult to pass a Miller Abbott tube through the pylorus and into the small intestine, but if this can be done it serves to combat the distention better than any other single factor. Certain surgeons now use catheter gastrostomy in preference to the employment of indwelling nasal tubes. Use of antibiotic therapy is dependent on scientific rather than wishful thinking. Tests of sensitivity performed on the organisms found to be present at the time of operation dictate which antibiotics to use. A word of caution must be sounded with regard to antibiotics lest their indiscriminate use produce complications just as serious as is generalized peritonitis. It is only after peristalsis begins to resume its normal activity and flatus is being expelled by rectum that the gastric tube may be clamped intermittently and, if the patient tolerates this well, later removed. A liquid diet is given cautiously and increased until a full diet may be taken.

Definite progress has been made so that at present the prognosis in almost any case of appendicitis is excellent. The concentration in recent years has been on the complications of appendicitis and such strides have been made that the surgeon today faces the problem with considerably more confidence than he did in earlier years. However in spite of all the recent advances, there is no substitute for sound clinical judgment tempered with reason and based on frequent, accurate and consistent observations made at the bedside, with correlation of all the clinical phenomena as they are revealed.

Chronic Appendicitis. Perhaps little should be said about the so-called entity of chronic appendicitis. No one has been able to prove whether or not such a condition exists. It is obvious that repeated attacks of acute inflammation in the appendix may leave local sequelae and certainly the surgeon is well justified in such a patient in removing the appendix during a so-called quiet interval. It is always better to make the error on the side of surgical aggressiveness because prevention in such instances is worth far more than a pound of cure. However the patient who presents himself with a collection of *vague symptoms* and a great lack of signs complaining of fatigue, nervousness, excessive gas, constipation and indigestion probably should be looked on as a constitutionally inferior person and it is better not to draw attention to the appendix without some definite reason. It is well known that appendectomy in such persons is fraught with a high degree of failure when judged in the light of relief of presenting symptoms.

Tumors of the Appendix. True tumors of the vermiform appendix are rare. They may be either benign or malignant. The commonest form is not a neoplasm but a condition known as *mucocoele* in which the ostium of the appendix becomes occluded and the continued secretion of mucus by the glands within the appendix results in a characteristic mass that is easily recognized grossly. Removal of the appendix cures the condition. In greatly advanced lesions the mucocoele may rupture, spilling its contents and the secreting cells into the free peritoneal cavity. As the process subsequently continues, the condition known as *pseudomyxoma peritonei* (jelly belly) results. Malignant mucocoeles have been reported.

Carcinoid tumors of the appendix are not

common but are rather characteristic when found. A firm mass is noted which on microscopic study shows a glandular picture. Many aggregates of tumor cells are buried in the submucosal layer as already noted, the appearance has been likened to that of basal cell carcinoma, hence the origin of the term *carcinoid*. It is possible that these cells are of chromaffin origin. As has been mentioned previously the small intestine can give rise to a similar tumor where it is prone to characteristics of malignancy and is capable of metastasis. Carcinoids of the appendix remain localized and once appendectomy has been performed further trouble usually does not occur. A correct preoperative diagnosis is almost unheard of the lesion being discovered incidentally.

True *adenocarcinoma* of the appendix is extremely rare. The surgeon may not know for some days after appendectomy that a malignant lesion was present. Since these are true cancers they should be treated as such. With the low risk to the patient of right hemicolectomy at present, it is well to submit the patient to this procedure once the diagnosis is established.

READING REFERENCES

- Carhale, J. C., and Judd, E. S. Jr. Regional Enteritis Involving the Duodenum: Report of Case. *Proc. Staff Meet. Mayo Clin.* 27:560, 1932.
 Crohn, B. B., Ginzburg, L., and Oppenheimer, G. D. Regional Ileitis. A Pathologic and Clinical Entity. *J.A.M.A.* 99:1323, 1932.
 Ewing, J. *Neoplastic Diseases. A Treatise on Tumors*, 4th ed. Philadelphia, W. B. Saunders Company, 1940. 1160 pp.
 Hilsabeck, J. R., Judd, E. S. Jr., and Woolner, L. B. Carcinoma of the Vermiform Appendix. *S. Clin. North America* 31:965, 1931.
 Van Patten, W. N. and others. Regional Enteritis. *Gastroenterology* 25:347, 1954.

The Colon

By CLAUDE E. WELCH, M.D.

CLAUDE EMMERSON WELCH is a Nebraskan whose father spent most of his early life teaching school. This fact may have influenced his son, a graduate of Doane College, a small college in Nebraska, to teach chemistry and obtain a graduate degree in organic chemistry at the University of Missouri. He received his medical education and surgical training at Harvard University and Massachusetts General Hospital. His surgical interests have been centered upon various phases of gastroenterology.

SURGICAL ANATOMY

The colon is defined anatomically as that section of the large intestine which extends from the ileocecal valve to the rectum. The lower level is difficult to identify accurately and the indefinite term "rectosigmoid" often is used to describe this junctional area. It is more accurate to employ Gilchrist and David's terminology. They divide the rectum into an extraperitoneal portion located entirely below the peritoneal floor and an intraperitoneal division which extends from the lower portion of the peritoneal reflection to the level of the third sacral vertebra. Above that level the bowel is no longer fixed and the true colon begins at the spot where this mobile mesentery is noted. Since the entire rectum is approximately 15 cm in length, about 10 cm of the terminal colon can be visualized through the ordinary 25-cm. sigmoidoscope.

The colon is subdivided loosely into the right, transverse and left portions. It is frequently better to employ more specific terms, so that from the proximal end these divisions become the cecum, ascending colon, hepatic flexure, proximal, transverse and distal transverse colon, splenic flexure, descending colon and sigmoid colon.

The arterial blood supply is furnished by the superior and inferior mesenteric arteries. Usually anastomosis between these two systems is maintained by the marginal artery which skirts the inner margin of the colon. However, short sections of this artery may be absent, particularly in the sigmoid and ileocecal areas, so that caution is necessary at the time of any operative procedure. Usually

ally the right colon is supplied by the ileocecal and right colic branches and the transverse by the midcolic, which arises from the superior mesenteric. The left colon is supplied by the left colic and sigmoid branches of the inferior mesenteric. The most common variations of this pattern include absence or an abnormal origin of the right colic branch. The left colic artery also may arise from the superior sigmoidal artery.

Venous drainage corresponds to the arterial supply and runs by way of the superior and inferior mesenteric veins through the portal system. Hence, blood borne metastases from cancer nearly always appear first in the liver though, later, collateral channels may involve the systemic circulation. The lymphatic drainage follows the arterial supply closely.

PHYSIOLOGY

While the colon is primarily an excretory tube, it also has absorptive powers. Water and electrolytes are reabsorbed, particularly by the cecum, so that the consistency of the fecal discharge is changed completely in this portion of the bowel. Glucose and sucrose are absorbed by the rectum and colon. Normally the colon secretes only mucus, although small amounts of some metals such as bismuth and arsenic, may be excreted directly by the colonic mucosa.

HISTORICAL ASPECTS

Modern surgery of the colon has developed within the course of the last sixty years. Prior to 1800 sporadic cures of intestinal obstruction had been obtained by vari-

ous means such as the forcible injection of enemas or the administration of mercury by mouth. Multiple percutaneous punctures of the distended bowel occasionally prolonged life.

In 1710 Littré, a French surgeon, first suggested that a colostomy might be useful to relieve obstruction of the colon. However it was not until 1776 that Pillore first successfully performed a cecostomy; the patient died a month later. In 1793 Duret first had a patient recover from a sigmoidostomy. After anatomic studies in 1839 Amussat found that a colostomy in the left lumbar area could be made without entering the peritoneal cavity and advised its routine use for cancer of the rectum.

The next stage in the development of colonic surgery involved the actual extirpation of a cancer of the rectum. Early surgeons resected the rectum, leaving an uncontrollable perineal colostomy. Lisfranc carried out the first successful operation in 1828 while Kraske added an excision of the coccyx and part of the sacrum in 1885. In 1833 a patient of Reybard survived after a resection and anastomosis for cancer of the colon. In 1879 Billroth resected the sigmoid colon and brought out the proximal end of the bowel as a colostomy opening. Thereafter surgeons followed two separate modes of attack on cancer of the colon. The first involved resection and primary anastomosis. The mortality rate for this operation was high and was not reduced significantly by the introduction of Murphy's button type of anastomosis in 1902. It was not until 1906, when Parker and Kerr introduced the principle of aseptic anastomosis that this method again became safe. However the operation of resection and immediate anastomosis was not accepted widely until recently but now has been adopted generally. That this procedure is unwise in the presence of acute obstruction was appreciated at an early date and Gibson's method of cecostomy (1902) was used widely to decompress the distended colon before resection was undertaken.

Meanwhile, the second trend in surgery of the colon involved the exteriorization of the obstructing lesion and its resection outside the abdominal wall. Thus, the death rate from peritonitis was practically eliminated. Bloch (1892) and Paul (1895) independently developed this method that was popularized by Mikulicz (1902). The same technique was modified and extended by Rankin (1930) and was for many years the

most widely accepted and by far the safest of the resections. Now however it has been nearly entirely replaced by resection and primary anastomosis, except for traumatic wounds of the colon, for which exteriorization is usually the treatment of choice, or for some instances of perforation secondary to diverticulitis or cancer.

WOUNDS AND INJURIES

The colon is particularly likely to be injured by missiles that penetrate the abdominal wall since this viscus occupies a relatively large space within the peritoneal cavity. Knife wounds will cause simple lacerations, while high velocity missiles usually produce jagged holes that are surrounded by contused bowel that will not prove satisfactory for suture. On the other hand, blunt trauma does not often lead to rupture of the colon since its relative mobility and elasticity protect it.

Perforation of the colon may also occur from foreign bodies that are swallowed, or are introduced through the rectum. Usually all such objects that pass through the ileocecal valve will go through the colon easily though, in the presence of disease such as diverticulosis, a sharp-pointed object may be caught and will penetrate the bowel. Chicken or fish bones, toothpicks and needles are the usual causes of such perforations. The sigmoid also may perforate from the accumulation of large fecaliths in this part of the bowel. Fecaliths are particularly likely to form when a large diagnostic barium meal has been followed immediately by operation, so that ileus and dehydration combine to produce this mass. A great variety of foreign bodies have been introduced into the rectum from below and then lost above the contracting sphincter. If they are long enough they may perforate the sigmoid. In our hospital such a perforation has been observed from a giant-sized carrot used ostensibly for anal dilation to relieve constipation.

Perforation of the sigmoid is always possible when an inept sigmoidoscopy is done. For this reason utmost care must be used in every case. Sigmoidoscopes over 25 cm. in length should be employed only by experts and in very co-operative patients, since an inadvertent sudden movement by the patient may be disastrous. Usually in these circumstances the perforation can be recognized by the sudden observation of the small intestine through the sigmoidoscope. However occasionally it will not be suspected until the patient later con-

enema tubes and enemas themselves may produce perforation. It is doubtful that the normal colon could be perforated by an ordinary enema, but a bowel wall weakened by inflammation or cancer may tear apart.

Workers about compressed air nozzles have occasionally either by accident or design, received a blast within the rectum. This is an extremely dangerous accident which is likely to blow out the colon. The region of the splenic flexure is most likely to be involved. A somewhat similar disruption of the colon may occur rarely when the intestinal gases are exploded by a spark during electrodesiccation of a polyp through the sigmoidoscope.

The symptoms of perforation of the colon are pain and tenderness appearing first at the site of injury and spreading rapidly to involve the entire peritoneal cavity.

Nausea and vomiting usually follow and then are superseded by the signs of vasomotor collapse. A shocklike syndrome is particularly likely to occur after perforation of the right colon has filled the abdomen with liquid feces and virulent peritonitis is established. Occasionally the passage of blood by rectum will indicate an injury to the colon.

Treatment will depend upon the cause and the location of the injury and the amount of bacterial contamination. In general, it is safe to suture fresh wounds of the colon which have not vitiated the blood supply of the segment. In the presence of well-developed peritonitis, intraperitoneal sutures are much less likely to hold and, when there has been additional trauma to the bowel beyond the laceration, or damage to the blood supply the exteriorization procedures are safer.

The earlier the operation is carried out, the better is the chance of success. Every attempt should be made to operate on the patient within six hours of injury. Furthermore, wounds of the right colon are more dangerous than those of the left because of the rapid dissemination of liquid feces when the cecum is lacerated.

The standard operation for injuries of the colon—exteriorization of the damaged segment—involves a wide mobilization of the colon to observe all areas of injury and withdrawal of the injured portion of the colon from the peritoneal cavity as a colostomy segment. In certain instances, when the perforation is in the low sigmoid or rectum, where the damaged area cannot be exteriorized, the perforation must be closed by suture and a completely defunctioning colostomy opening made, preferably in the

transverse colon. The colostomy stoma is closed by resection and intraperitoneal anastomosis four to six weeks later.

VOLVULUS

Volvulus is an important cause of obstruction of the colon. The term should be restricted to those cases which show a rotation of the bowel about its mesenteric axis sufficient to produce obstruction. While it may occur in any mobile portion of the intestine, in the colon it by far is found most commonly in the sigmoid. The cecum is involved less frequently and the transverse colon rarely. Megacolon and a consequent history of constipation usually antedate an acute volvulus by many years.

The disease is much more common in persons of advanced age than in younger individuals, and in males than in females. There is a great variation in incidence in different countries, suggesting that congenital causes are important. For example, it is much more common in Russia than in America. In our hospital it causes about 4 per cent of all acute obstructions of the colon.

Pathologic studies show that the volvulus may be either clockwise or counterclockwise. Such a twist rapidly produces a closed loop obstruction that may be fatal shortly unless the situation is remedied. Increased tension within the loop leads to obliteration of the blood supply, gangrene, perforation, peritonitis and death. A rapid progression is most likely to occur in the cecum because of its thin wall that easily becomes avascular.

The symptoms are those of large bowel obstruction. The lower abdominal cramps are followed by obstipation and distention. In early cases, the distention is limited to the obstructed loop though later it becomes generalized. Absence of peristalsis or tenderness is a serious sign indicating strangulation. An abdominal x ray examination usually shows an enormous loop of colon filled with gas and fluid. Since the twisted loop may appear in an unusual position, a barium enema should always be employed to confirm the diagnosis and locate the volvulus anatomically. Thus a twisted cecum is not infrequently palpable in the left upper quadrant and, without a barium enema, an unfortunate incision may be made for its correction. Characteristically the barium enema will outline the lower end of the volvulus showing a cone shaped deformity which may resemble a bird's beak, at the point of obstruction.

The treatment of volvulus of the colon is

surgical. However it must be recognized that spontaneous reduction of the volvulus may occur if the twisted loop can be deflated. When the sigmoid is involved and there is no evidence of strangulation of the blood supply deflation often can be accomplished by the careful introduction of a rectal tube through a sigmoidoscope. Need less to say such a maneuver must be done with great care. When the cecum is involved, resection and primary anastomosis may be carried out in good risk patients. Resection is necessary if the blood supply is questionable. Otherwise an alternative procedure is detorsion fixation of the cecum is then accomplished by means of cecostomy. When the sigmoid is involved and the patient in good condition, resection and anastomosis likewise are feasible sometimes, simple detorsion, which will be followed by resection at a second stage, or detorsion plus an obstructive resection, will be wise. In all patients, unless the offending loop has been resected, there is a great tendency to recurrence. Careful attention to the bowels, with the avoidance of constipation or distention, is essential.

INFLAMMATORY DISEASES

The important inflammatory diseases of the colon include ulcerative colitis, bacillary dysentery, amebiasis, tuberculosis and actinomycosis. Diverticulitis since it depends on the presence of diverticulosis will be discussed as a separate entity. Recently an acute enterocolitis usually due to the staphylococcus has emerged as an important post-operative complication. It is particularly apt to follow resections of the colon for cancer. Lymphogranuloma primarily affects the rectum but also may involve the sigmoid colon.

Bacillary Dysentery. This disease is of minor importance to the surgeon. Occasionally in the subacute or chronic form, it may be mistaken for chronic ulcerative colitis since, on sigmoidoscopic examination, ulcers may be seen along the transverse ridges of the lower colon. Later these ulcerations tend to coalesce and to encircle the bowel. Bacteriologic study of the organisms isolated from these ulcers should reveal one of the dysentery bacilli.

Amebiasis. Amebas are ingested as cysts, the walls of which are dissolved in the small bowel. Free, motile amebas then enter the mucosa of the large bowel. Initially widespread ulcerations are produced, involving particularly the cecum, ascending colon and upper rectum. Superficial, ragged ulcers are

produced that penetrate below the upper layers of the submucosa. Motile amebas are recovered regularly from these fresh ulcerations and usually in warm stool preparation. The diagnosis of acute amebic dysentery can be made by this method, since the ulcers are nearly always observable through the sigmoidoscope at this stage of the disease.

Later in the course of the disease, chronic colitis is produced which may progress to the formation of large granulomas or amebomas, as they sometimes are called. Amebomas are often confused with cancer since the other signs of amebiasis may be absent. These granulomas have been encountered in all parts of the colon but are most common in the cecum and rectum.

Amebiasis of the colon is of surgical significance chiefly because it mimics other diseases. The differentiation of amebic colitis and ulcerative colitis is often different in the acute and chronic stages, while the granulomas are likely to be resected in the belief that they are cancer. Specific complications referable to the disease are perforation and hemorrhage. Perforation usually occurs in the cecum or in the appendix, which is involved in about 40 per cent of the subjects of amebic dysentery. The most important complication of amebiasis is liver abscess; it should be suspected in the presence of increased fever or marked leukocytosis.

Tuberculosis. Tuberculous enterocolitis is encountered rarely except in sanatoria since it is usually secondary to pulmonary tuberculosis. This secondary tuberculous enterocolitis was found by Rubin in two-thirds of a series of 569 patients who died of pulmonary tuberculosis. Primary ulcerative enterocolic tuberculosis is a very rare disease in the United States at this time because of milk pasteurization. Either of these lesions may lead to a granulomatous form known as hypertrophic intestinal tuberculosis. In this type of disease, a large inflammatory tumor is produced somewhere in the intestinal tract and is followed by the observation of a mass or by subacute intestinal obstruction. The cecum is the usual site of disease. Some of these cases actually are manifestations of regional enterocolitis. The disease is rare, but, when it occurs surgical resection is required.

Regional Enterocolitis. Regional enteritis always involves the small intestine but, in certain instances a similar disease may occur that is confined to the terminal ileum and ascending colon. This lesion is more closely related to regional enteritis than it is to

ulcerative colitis. It tends to remain localized in this area and therefore the results following resection and anastomosis are good.

Chronic Ulcerative Colitis. This is a disease of unknown cause manifested by chronic colitis with ulceration. While it follows a typical clinical course the diagnosis is usually established by the elimination of other etiologic agents. Many causes of the disease have been suggested, such as specific bacteria, viruses, fungi, allergies, digestion of protective mucous membrane by the enzyme, lysozyme and abnormal colon function due to disordered nerve reflexes.

Clinically the disease usually starts abruptly most commonly in the individual's third decade of life. Diarrhea, which usually is bloody, fever and loss of weight occur. Unless the first acute attack is fulminant, leading to surgery or death, the patient improves, but the disease tends to become chronic with repeated exacerbations.

The diagnosis is usually made by sigmoidoscopy, since in about 95 per cent of the subjects the disease begins in the upper rectum and spreads back to the cecum often for a short distance in the small bowel. Occasionally the disease may start in the right colon. The barium enema will demonstrate saw-tooth serrations along the margin of the bowel in patients in the early phase of the disease, but, as the disease progresses, the colon becomes shortened and tubular with loss of haustrations.

Sigmoidoscopic examination in an early stage shows an edematous, boggy mucous membrane which bleeds on wiping. Later irregular superficial ulcerations appear. The ulcers deepen and may penetrate the bowel wall. Meanwhile, normal mucosa disappears. Attempts at regeneration lead to pseudopolyp formation. Cancer is likely to develop later in any portion of the involved colon.

Medical measures used for therapy include a bland diet, sedatives, antispasmodics, elimination of all known psychic problems and, in numerous cases, cortisone or ACTH. They are usually successful but will fail in at least a quarter of the patients. Surgery becomes necessary for those patients who cannot be controlled otherwise. The indications for surgery are numerous. In the first attack, debilitating diarrhea, high fever and toxemia may make surgery necessary within the first two weeks of symptoms. Fortunately cortisone or ACTH therapy is most helpful here. More often these same symptoms are alleviated to some extent, but surgery is necessary in the chronic phase of the

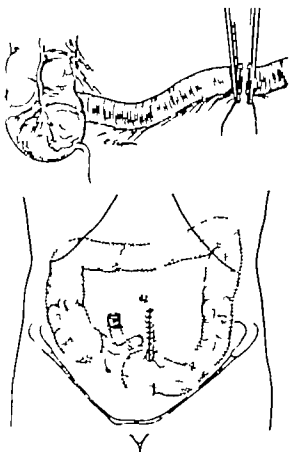


Figure 72. Ileostomy. This type of ileostomy will defunction the entire colon. It is used particularly in the surgery of ulcerative colitis and multiple polyps involving the entire colon and rectum. The ileum is divided between clamps and the stoma made in the right lower quadrant.

disease. Complications referable to the colon requiring surgery are subacute perforation, abscess or fistula formation, obstruction, persistent or severe hemorrhage and the development of pseudopolyps. Other complications that can be improved only by ileostomy and colectomy include infectious arthritis, uveitis and, occasionally, wide spread cutaneous infections.

Surgical therapy includes ileostomy (Fig. 72). At the present time this is nearly always combined with subtotal colectomy or with total colectomy and proctectomy. Ileostomy alone usually will relieve symptoms, and the appearance of the colon as visualized through the sigmoidoscope will improve. However, despite this improvement, it is not wise to consider closure of the ileostomy stoma at a later date, since this nearly always leads to an exacerbation of the disease. Thus, in a series of 149 patients with ulcerative colitis treated surgically in the Massachusetts General Hospital, reported by McKittrick and Moore, closure was thought advisable in only five instances and was a success in only

one. Furthermore, many complications can follow ileostomy. They include stenosis of the stoma, prolapse of the distal ileum through the stoma, intestinal obstruction due to prolapse of the ileum about the terminal ileum and fistula formation about the stoma. One of the most serious of these complications—the so-called ileostomy dysfunction—actually is a type of obstruction due to a stoma which is blocked by edema or granulation tissue. Suture of mucosa to skin has essentially eliminated this problem. These complications are most common within the first few months after the establishment of the ileostomy opening. When this period is over the patient can lead a relatively normal life thanks to the development of bags that can be cemented to the skin.

Later in life however if the colon has not been removed, cancer of the colon is likely to develop. Statistical studies by Dennis and Karlson indicate that the chances of cancer are almost nil within the first ten years after ileostomy but increase regularly after that time at the rate of about 1 per cent a year. It therefore seems wise, in the light of our present knowledge, to advise total removal of the colon and rectum after an ileostomy opening has been made.

Recently an increasing number of patients particularly in older age groups have appeared with segmental ulcerative colitis which is amenable to segmental colectomy. Whether or not this is the same disease as that observed in younger patients is not clear.

DIVERTICULOSIS AND DIVERTICULITIS

Diverticula of the colon are abnormal herniations of mucosa through the muscular coats of the bowel which produce small saclike projections. Inflammation of these diverticula sufficient to produce symptoms or visible evidence of localized spasm of the colon on x-ray study or sigmoidoscopy is known as diverticulitis.

The etiologic background of the disease is not known except for solitary cecal diverticula which are believed to be congenital in origin. Other diverticula appear near to blood vessels that run from the mesentery through the wall of the bowel. It is presumed that this intrinsic weakness of the bowel is followed by increased intracolonic pressure with the production of diverticula. The predilection of the disease for the sigmoid is accounted for by the fact that feces in this area are much firmer than elsewhere. The anatomic curvature of the sigmoid may

contribute. Furthermore, spasm in the sigmoid is common, especially during the expulsion of feces, while it is not observed in the cecum and ascending colon. At any rate, it is clear that diverticulosis is one of the degenerative diseases appearing rather uncommonly in persons below the age of fifty years and becoming more common thereafter as age advances.

Autopsy studies have shown that about 7 per cent of all colons have diverticula. Careful barium enema examinations should show a much higher incidence since demonstration is more exact by this technique. In a check of 2000 consecutive persons in the Massachusetts General Hospital who had been administered barium enemas, essentially neither diverticulosis nor diverticulitis was found in persons below the age of thirty-five years. From that point onward the disease increased rapidly in frequency so that diverticula could be demonstrated in two-thirds of the eighty-five-year-old patients and the individual diverticula increased in size with age. The incidence of diverticulitis also showed a steady rise.

Diverticulosis is rare in the rectum and in the cecum. According to Malters' autopsy studies, it was confined to the sigmoid (Fig. 73) in 40 per cent of the subjects and scattered throughout the entire colon in 30 per cent. Interestingly enough, the clinical manifestations of diverticulitis, on the other hand, are practically confined to the sigmoid. In the Massachusetts General Hospital, of the patients requiring surgery for diverticulitis, 92 per cent had involvement of the sigmoid or lower descending colon, 3 per cent of the cecum, 3 per cent of the rectum and 2 per cent of the transverse colon.

It is often impossible to tell the exact time diverticulosis undergoes transition to diverticulitis. Certainly many or all patients having pure diverticulosis are entirely asymptomatic. The symptoms of diverticulitis are alteration of bowel habit, pain, tenderness and bleeding.

Alterations in bowel habit include constipation, diarrhea, gas and distention. They are difficult to evaluate in many instances. However distinct episodes of diarrhea, particularly when they are accompanied by left lower quadrant pain and tenderness, are very suggestive of diverticulitis. Constipation is common and becomes more severe if inflammatory narrowing of the bowel occurs.

Pain is of two types. One consists of cramplike attacks that are relieved by t.

passage of gas or feces. The other consists of localized pain and tenderness due to actual inflammation about the diverticula. The latter of course may progress to varying degrees of peritonitis.

Bleeding is not an infrequent symptom. Theoretically it should not occur with diverticulosis per se. Probably it occurs in 15 to 20 per cent of all patients with diverticulitis. The source of the bleeding may be coincidental hemorrhoids, cancer polyps, pseudopolyps, acute nonspecific ulcer occurring in an area of diverticulitis or the diverticulitis itself. Persistent daily bleeding is more characteristic of cancer.

The indications for surgery include perforation, obstruction, hemorrhage and persistent discomfort unrelieved by medical care.

Perforations vary greatly in significance. A perforation into the mesenteric fat may produce only a minimal amount of pain and tenderness. In other instances a large inflammatory mass may be produced. If perforation occurs into the general peritoneal cavity a localized abscess or general peritonitis will occur. The clinical signs will parallel these manifestations. Fever and leukocytosis of varying degrees are found. With free perforation, gas may be demonstrated below the diaphragm by x-ray examination. The pain from acute penetrating diverticulitis of the sigmoid may be felt chiefly in the right lower quadrant, so that acute diverticulitis may be confused with appendicitis.

One of the clinical features of diverticulitis is that of the progression of local perforations to form fistulae, which usually extend into the bladder but the small intestine, other portions of the colon, vagina or skin all may be involved. Sigmoidovesical fistulae are much more common in the male than in the female because the uterus often forms a protecting barrier between the colon and bladder.

Though diverticulitis produces nearly 15 per cent of all the acute colonic obstructions observed in our hospital, it characteristically produces a chronic, slowly progressive obstruction, so that acute complete obstruction without any previous symptoms is not common. It is surprising to note how severe a degree of stenosis may be observed radiologically and the patient still have fairly adequate colonic function. Abdominal cramps, relieved by the passage of gas or feces, are early symptoms suggesting mild obstruction. They are followed later by

gradual distention and, in rare instances, by complete obstipation.

Bleeding is an indication for surgery in a few instances because it is so massive that control of blood loss is necessary. In other cases, continued bleeding and a questionable radiologic picture that could be either diverticulitis or cancer will make surgery wise.

The acute phases of diverticulitis usually can be treated medically with relief. If these acute episodes occur rarely after long intervals of freedom from discomfort, the patient may prefer to continue with this therapy. Attacks recurring in close succession, particularly if they occur while on adequate dietary regulation, make surgery necessary.

The differentiation of cancer and diverticulitis is often very difficult since the symptoms may be the same. Sigmoidoscopy may give no clue. The radiologist usually is able to distinguish them, though occasionally he may be wrong in his diagnosis or the diseases may coexist.

Schatzki has noted the important radiologic features of diverticulitis and cancer of the sigmoid. Diverticula are typically present with diverticulitis and absent with cancer. A long segment of the colon tends to be involved by diverticulitis and a short one by cancer. The mucosa in the involved segment is normal with diverticulitis and destroyed by cancer. The adjacent colon is spastic with diverticulitis and normal with cancer. The end of the defect is cone shaped with diverticulitis and shelflike with cancer. On



Figure 73 Diverticulosis of the sigmoid. Barium enema, postevacuation film. The diverticula are located entirely in the sigmoid. A single gallstone is seen.

repeated examinations, an enlarging lesion is very suggestive of cancer. Obviously, many cases are atypical, so that differentiation of the two diseases is impossible. Thus in nearly 25 per cent of the patients operated upon for diverticulitis, cancer is suspected preoperatively at the time of operation by the surgeon, and is only ruled out when the pathologist opens the specimen. This implies that the utmost vigilance must be used whenever a diagnosis of diverticulitis is made.

Experience has shown that several other groups of patients will eventually require surgery for diverticulitis. Those in whom the diagnosis is made before the age of fifty years have a particularly virulent form of the disease. Those who have severe persistent deformity of the sigmoid on radiologic examination have organic changes rather than spasm. Those who develop urinary symptoms in the presence of diverticulitis, particularly if they are males, must be suspected of developing a sigmoidovesical fistula. Resection of the involved segment of colon is advised for these groups of patients.

The medical therapy of diverticulitis consists of a low residue diet, daily doses of mineral oil or a demulcent such as Mucamucil, antispasmodics and sedation. The more acute phase will require sharper restriction of the diet and antibiotics if there is evidence of perforation.

Surgical therapy has as its objective the wide excision of the involved section of the colon and re-establishment of intestinal continuity. This can be accomplished in one stage in many instances. Perforation or complete obstruction will make a colectomy desirable as a first stage procedure to be followed later by resection and anastomosis and, at a third stage closure of the original colectomy opening. As an alternative procedure in the presence of perforation or obstruction some surgeons prefer an obstructive resection.

Nearly three-quarters of all the patients will respond to medical therapy. Those treated surgically by a generous resection nearly always remain free of symptoms even though diverticula develop later in remaining portions of the colon.

BENIGN TUMORS

The benign tumors which arise in the colon or rectum are important because they often are precursors of malignant lesions and many produce symptoms which require relief. They are most easily classified by

their histologic appearance. They include the common adenomas and the rarer tumors such as lipomas, fibromas, angeliomas, leiomyomas, carcinoids, endometriomas and pseudopolyps. These lesions nearly always protrude into the lumen of the bowel and therefore may be described as polyps. Polyp, by definition is a nonspecific term insofar as the histologic pattern is concerned but commonly is used to designate an adenoma, since the adenomas are far more common than are all other types of benign tumors combined.

Adenomatous Polyps. Helwig found a 9.5 per cent incidence of polyps of the colon and rectum in 1400 autopsies and noted an increase in number with age. Long series of sigmoidoscopies on asymptomatic patients have shown a very similar incidence.

Wilson, Dale and Brines did sigmoidoscopic examinations on 20,847 patients, all of whom presumably were asymptomatic. They found the incidence of polyps to rise steadily with age. In males, it was 2.6 per cent in those below the age of forty increasing to approximately 11 per cent at the age of seventy or over. In females the curve is distinctly lower rising from 1.7 per cent under age forty to less than 5 per cent at age seventy. Thus Hauch, Buie and Smith found adenomas in 8.1 per cent of 1919 patients. Approximately two-thirds of all adenomatous polyps are found in the rectum. Most of the others are found in the sigmoid and the remainder are scattered throughout the colon.

Adenomatous polyps may be classified in several ways. Depending upon their number they fall into three distinct classes. In the first, a single or a few polyps are located in one segment of the bowel. In the second (scattered polyposis) a few polyps are found in different segments of the colon and, in the third, multiple polyposis, the polyps are both numerous and widely disseminated (Fig. 74). Grossly the individual polyp may be described as pedunculated or sessile, but this is of much less importance than the microscopic picture. Histologically these lesions are either benign adenomatous polyps, benign polyps with malignant degeneration of a portion of the polyp or papillary adenomas.

The cause of polyposis is not known. A definite familial susceptibility has been demonstrated in persons having multiple polyposis and may well be present in others.

It is generally believed that adenomatous polyps eventually develop into clinical can-

cer This transition has been observed rarely but was seen by Bule and Brust in a period of two to ten years in four patients who refused treatment. Other arguments favoring this conclusion are that the distribution of polyps and cancer is similar throughout the colon, that the incidence by age and sex is approximately the same for the two lesions, that polyps are found in about 25 per cent of all colons resected for cancer and usually in close proximity, and that frequently multiple lesions can be demonstrated in the colon which show all changes of transition from benign polyp to invasive cancer. Patients with multiple polyposis nearly always die of cancer before the age of forty years unless a colectomy has been done.

Recently Spratt, Ackerman and Moyer have introduced evidence to indicate that benign polyps do not degenerate into cancer. This concept, however, should not influence the clinical surgeon who must realize that the only method of differentiation between benign polyp and polypoid cancer is by microscopic analysis of the resected tumor.

Polyps often are entirely asymptomatic. If symptoms occur they comprise bleeding, change in bowel habit, abdominal discomfort or the discharge of mucus by rectum. Bleeding is the most common symptom. While it usually consists of blood streaking on the surface or in the substance of the stool, it may occasionally be very profuse. Diarrhea is particularly common with multiple polyposis, but constipation also may occur. Intussusception of the polyp may lead to bleeding and cramps.

The diagnosis is made by sigmoidoscopy or barium enema. Sigmoidoscopy must always be done as the first step. Later a barium enema is given. Preferably this is followed by an air contrast enema. Demonstration of polyps by barium enema is often difficult and usually impossible until the polyp is over 1 cm. in diameter. The bowel must be completely prepared or fecal lumps will be mistaken for polyps. If a defect is found that is consistent with a polyp the barium enema must be repeated to confirm the diagnosis before surgery is undertaken.

Unless there are serious contraindications, polyps should be removed. By no other way than microscopic examination can they be differentiated from cancer. When the lesion is located in the colon, this generally means that a laparotomy is necessary though some small polyps of the sigmoid can be removed safely through the sigmoidoscope.

At the time of laparotomy the surgeon

must find the polyp, determine whether it is benign or malignant, discover whether other polyps are present or not and then carry out the appropriate operation. In the individual patient this is often a difficult problem and it is not surprising that surgeons do not always agree on the most satisfactory procedure. Examination of the colon by the sigmoidoscope at the time of laparotomy through one or more colotomy incisions has been proved to be the best method to demonstrate small polyps.

From a practical point of view it may be said that two operations are possible. In the first—colotomy and polypectomy—the colon is opened and the polyp removed locally. A frozen section is made whenever possible to confirm the diagnosis of benignancy. This operation should be avoided whenever the polyp is hard or has a broad base since cancer is likely to be present.

The second operation which may be done is segmental resection of the colon. The author among others, prefers this procedure as a routine on several bases. Cancer is relatively common in these polyps, as shown by a series of 102 colonic polyps in our hospital in which cancer was found in 24 per cent. Also recurrence of supposedly benign polyps that have been removed locally is not rare. Judd and Carlisle followed 248 patients from the Mayo Clinic five years or more after transcolonic polypectomy had been done. They found definite clinical cancer of the colon had developed in fifteen of these. Local recurrence was the rule if there was



Figure 74. Diffuse polyposis of the colon. Resected specimen. The terminal ileum and colon, except for the terminal sigmoid, are shown. Polyps are present in all portions of the colon. The terminal ileum is in the lower portion of the outer circle.

any microscopic evidence of invasive cancer. In these patients, in contrast to those with lesions of the rectum which can be kept under observation with a sigmoidoscope, local recurrence cannot be detected until a very sizable tumor is present.

In a series of cases in the Massachusetts General Hospital, thirty four patients had local excision of a malignant polyp of the colon or rectum and nine were later found to have metastases or to have died of cancer. Microscopic examination determines the prognosis since all patients with recurrence had adenocarcinoma, grade II or III. Those with adenocarcinoma, grade I, did well after local resection.

Multiple Polyposis. In the typical patient with multiple polyposis there are hundreds of polyps scattered throughout the colon and rectum. The most serious variant is "polyposis en nappe" when the whole colon is carpeted with polyps. They usually develop in childhood and may be associated with polyps of other portions of the gastrointestinal tract. Jeghers noted that these patients often have melanin spots on the lips. The disease is hereditary, is present in male and female and may be transmitted either as a dominant or recessive characteristic. Whenever the disease is found, other relatives of the patient ought to be examined.

Treatment depends upon the location of the polyps. If the rectal mucosa is completely covered with polyps the entire colon and rectum will have to be sacrificed and an ileostomy done. If the rectum is comparatively normal, a subtotal colectomy can be done and the ileum anastomosed to the upper rectum. Later the rectal polyps will be destroyed by electrocoagulation. Needless to say, careful follow-up observation of the rectum must be made for many years.

Papillary Adenoma. This is a special type of polyp which tends to be large and soft, with elevated papillary folds. It tends to spread laterally and is prone to develop malignant degeneration. Microscopically these tumors present a distinctive picture of large villi covered with proliferating epithelium.

While nearly 10 per cent of polyps are of this type, the great majority of them occur in the rectum. Occasionally however they may be found in any other portion of the colon. In these areas even if the surgeon knows where they are located, it may be totally impossible to feel them through the wall of the bowel.

In the colon these lesions must be treated

by segmental resection including a wide portion of the mesentery.

Other Tumors. Lipomas may develop in the submucosa of the colon and, by enlarging within the lumen of the bowel, produce a large submucosal tumor. Often they initiate an intussusception. They are with the exception of polyps the most common benign tumors of the bowel. The preoperative diagnosis is often possible on the basis of a long history and an intussuscepting tumor. Occasionally the radiologist can demonstrate the typical radiolucent lipoma.

Endometrial implants are often found on the sigmoid and cecum. In the sigmoid colon, endometriosis may produce a constricting deformity which produces symptoms of colonic obstruction. Since the disease practically never involves the mucosa, radiologic examination should suggest the diagnosis. While symptoms are most apt to occur during the menstrual life postmenopausal obstruction may occur. Pseudopolyps are actually inflammatory granulomas and not true tumors. They occur particularly with ulcerative colitis and occasionally with other inflammatory lesions.

The other benign tumors are rare and are usually diagnosed only by microscopic diagnosis of the resected specimen. They include angiomas, fibromas, leiomyomas and carcinoids.

MALIGNANT TUMORS

Malignant tumors of the colon essentially are restricted to carcinoma. The various forms of malignant lymphomas are encountered rarely. Liposarcomas, fibrosarcomas, malignant carcinoids with metastases, malignant melanomas and angiosarcomas occur rarely while metastatic tumors from other sites such as the stomach or breast, may produce colonic obstruction. An indication of the relative frequency of these tumors may be shown by the fact that, during a period of eleven years 1300 cases of cancer of the colon were observed in the Massachusetts General Hospital while only nine colonic lymphomas, the next most common tumor were encountered in forty years. Since the diagnosis and treatment of all malignant tumors are essentially the same, regardless of type, carcinoma of the colon will now be considered as an entity.

Cancer of the Colon. This is one of the most common types of cancer. According to the United States Public Health Service statistics over 28,000 deaths occurred from this disease in 1956 and if _____ of the

colon and rectum are grouped together this is the most common of the fatal cancers. The disease is slightly more common in females than in males in the colon while in the rectum this frequency is reversed, according to Ottenheimer and Oughterson who conducted an exhaustive statistical study of cancer of these viscera in Connecticut.

The cause of cancer of the colon is unknown. It is particularly common, however, in colons involved by multiple polyps or by ulcerative colitis. In the latter disease it appears to originate from areas of mucosal scarring and abnormal regeneration rather than from the pseudopolyps which often occur with ulcerative colitis. If cancer is encountered before the fourth decade, it usually is associated with one of these diseases. As noted above, many cancers are believed to originate from adenomatous polyps. Swinton and Warren believe they have found sound microscopic evidence of this origin in about 20 per cent of all cancers of the large bowel.

Symptoms are quite variable, depending upon the primary location of the tumor. It is best to consider lesions of the right and left colon separately.

In the right colon early symptoms are usually vague and consist of indefinite flatulence or distention that gradually progresses to true cramplike pains. The picture often mimics gallbladder disease so that careful exploration of the colon is essential whenever a gallbladder is removed. Secondary anemia is very common since these lesions usually present a large infected surface that is easily digested by the ileal contents. This anemia often is the first symptom so that anemic patients, particularly when they have many reticulocytes in the smear must be investigated for cancer of the right colon. This anemia also may be encountered with lesions of the transverse colon but is rare with cancer of the sigmoid. The anemia is often manifested by fatigue and loss of weight. The stools, while positive for occult blood, rarely show gross blood. At times the first indication of the cancer is a large palpable mass, perhaps first detected by the patient. A change in bowel habit is common and usually is manifested by diarrhea.

In the left colon, symptoms differ because of the solid nature of the feces and the relatively small diameter of the bowel. Slowly increasing constipation is most common, though there may be short episodes of diarrhea. Bleeding with bowel movements is

common bright or dark blood being mixed with the feces.

Occasionally the first symptom of colonic cancer is complete acute intestinal obstruction. This is less common when the cancer is in the right colon but occurs in perhaps 10 per cent of patients having cancer of the left colon being most common when the tumor is in the splenic or sigmoid flexures. Other less common symptoms are those of perforation or intussusception.

The diagnosis suspected from the history must be confirmed by sigmoidoscopy and barium enema. Sigmoidoscopy is important, not alone to observe cancers of the lower sigmoid, but also to visualize any polyps which might be present in the rectum. Benign polyps coexist with cancer of the colon in about 25 per cent of all the patients and while they are most common near the malignant tumor may be found anywhere throughout the colon.

The barium enema study is extremely important and must be made with great care (Fig. 75). Even then some cancers of the colon may be overlooked. Cancer of the cecum and ascending colon is notoriously difficult to demonstrate, but it is also possible to miss cancer of other portions of the colon. Hence if the examiner suspects cancer of the colon strongly an exploratory lapar-



Figure 75 Carcinoma of the colon. In this barium enema study a typical napkin-ring type of deformity is seen in the upper end of the descending colon. The splenic flexure is low since it had been resected previously for carcinoma.

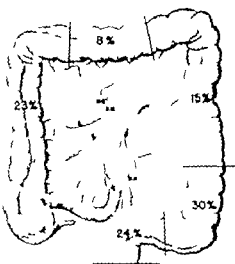


Figure 76 Distribution of carcinoma of the colon. The relative percentage of carcinoma arising in various segments is shown. The arterial blood supply of the colon is inserted. S.M. = superior mesenteric; I.M. = inferior mesenteric; L.C. = ileocolic; R.C. = right colic; M.C. = middle colic; L.C. = left colic; S. = sigmoidal; S.H. = superior hemorrhoidal.

rotomy may be necessary at any rate, very careful follow up will be essential.

Cancer of the colon is most common in the sigmoid flexure where about 50 per cent of all colonic cancers are found, and occurs in order of descending frequency in the ascending, descending and transverse colon (Fig. 76). The lesion is nearly always an adenocarcinoma, though squamous metaplasia rarely may produce an epidermoid carcinoma. Malignant carcinoids are found rarely. The adenocarcinomas may be subdivided into several grades and, in many cases, the tumors produce large amounts of mucoid material forming the colloid carcinomas.

Spread of this cancer occurs in several ways by direct infiltration of the wall of the bowel, by intramural extension along the wall, by lymphatic or blood-borne metastases or by direct desquamation of living cancer cells within the lumen of the bowel (Fig. 77). These factors are all of importance and will be demonstrated in varying degrees, depending on the type of the tumor and the length of time it has existed. Direct infiltration through the bowel wall will produce peritoneal carcinomatosis or in more favorable cases direct infiltration into an other viscus or the abdominal wall. Intramural extension along the wall of the bowel is frequent but, in the absence of lymphatic metastases, only rarely is found more than 2 cm. from the primary lesion. Lymphatic metastases occur in about a third of our re-

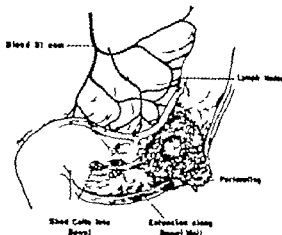


Figure 77 Methods of spread of cancer of the colon. The five ways by which cancer of the colon is disseminated are shown. Extension occurs into the blood stream, into the lymphatics, by perforation through the bowel wall, by intramural growth, and by the shedding and reimplantation of viable cells in the lumen of the colon.

sected specimens. Four sets of lymph nodes are involved—the epicolic, the pericolic, the intermediate and the deep nodes. They follow the arterial blood supply. Blood-borne metastases follow invasion of veins by cancer. Microscopic incidence of this invasion was present in over 40 per cent of all patients having cancer of the rectum investigated carefully by Madison, Dockerty and Waugh, so that it is not surprising that hepatic metastases are frequent and early. That operative manipulation may contribute to tumor embolization has been proved by Fisher and Turnbull.

Nitrogen mustard, administered at the time of operation, theoretically should reduce the danger due to manipulation of these metastases. Cole has contributed important experimental data in proof of this concept.

Direct desquamation of cancer cells can often be observed by cytologic studies of the rectal discharge following a colostomy proximal to the tumor so that it is reasonable to suppose that this occurs regularly. This factor is not of much importance except to the surgeon, who after resection of the tumor and primary anastomosis of the colon, may implant cells in the fresh suture line and later observe recurrence at this site.

An important characteristic of cancer of the colon is its tendency to multiplicity. Two separate primary tumors are encountered not infrequently when the patient is first admitted to the hospital. Also, the development of new colonic cancer in later years is many times more common than in the nor-

mal colon. In approximately 5 per cent of patients having colonic cancer multiple primary cancers develop. One of our patients who did not have multiple polyposis, finally had seven primary lesions.

This is one of the important reasons that Wangensten and co-workers have urged subtotal colectomy as the therapy of choice for cancer of the colon, though most surgeons still elect less extensive operations for single tumors.

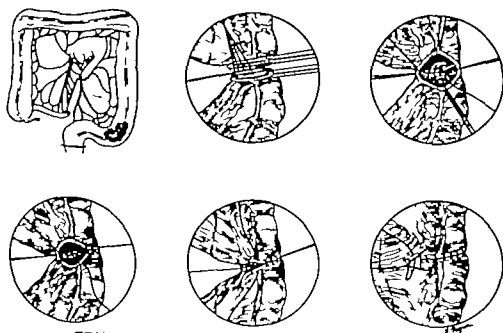
Cancer of the colon is amenable to surgical extirpation since it usually grows slowly and remains localized for a long time. Wide block dissection can be accomplished with removal of involved adjacent viscera and usually a permanent colostomy can be avoided. The operation selected will depend primarily upon the segment of colon involved, the presence or absence of such complications of cancer as obstruction or perforation and the condition of the patient.

Generally speaking, operations involving exteriorization of the involved segment of the bowel have been discarded. Preferably a one-stage resection of the colon with a wide section of the mesentery is accompanied by a primary end-to-end anastomosis (Fig. 78).

To attain this result with safety the bowel must be prepared carefully and no important degree of obstruction can be present. Mechanical cleansing by mild cathartics or ene-

mas is most important. In recent years great significance has been attached to the reduction of the bacterial flora of the colon by various antibiotics. There is no question but that the bacterial count of the stool can be reduced significantly by many drugs particularly by the nonabsorbable sulfa derivatives such as Sulfathalidine or Sulfasuxidine and even more dramatically by many of the broad spectrum antibiotics such as Terramycin or neomycin. Unfortunately nearly total abolition of coliform bacilli not infrequently has led to a compensatory proliferation of virulent organisms not responsive to antibiotics. Thus pseudomembranous enterocolitis due to the *Staphylococcus pyogenes* var *aureus* has emerged as an extremely serious complication of antibiotic therapy. Since this complication is very uncommon after the use of Sulfathalidine or Sulfasuxidine, this method of preparation is preferred at the Massachusetts General Hospital. Neomycin also appears to be relatively safe, while Terramycin has been dangerous.

Assuming that no element of obstruction is present and a one-stage operation is to be done a wide segmental resection of the colon is carried out. Before manipulation of the tumor clamps or ties are placed about the colon near the growth to retain viable tumor cells released by manipulation of the growth. Preferably at least 10 cm. of normal colon should be resected on either side of the



TECHNIQUE OF OPEN END-TO-END ANASTOMOSIS

Figure 78. Resection and anastomosis for cancer of the colon. The section of colon and mesentery to be resected is shown in the stippled area. After resection an outer posterior layer of interrupted cotton sutures is placed, followed by an inner one of fine continuous catgut. An outer anterior row of interrupted cotton is followed, when possible, by careful closure of the mesenteric defect.

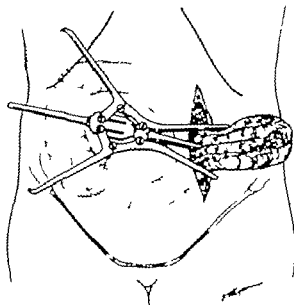


Figure 79 Obstructive resection of the colon. A Rankin clamp is applied, after which the tumor bearing portion of the colon will be excised with the cautery. At a later date the colostomy stoma will be closed. A limited portion of colon and mesentery is obtained by this method.

tumor Preliminary ligation of the tributary blood vessels will reduce the chances of tumor emboli. The mesentery, lymph nodes and tributary blood vessels are excised down to the superior mesenteric artery on the right side. On the left side the terminal portion of the inferior mesenteric artery must be removed with carcinomas of the lower sigmoid, while some surgeons prefer to remove

the entire vessel whenever the carcinoma arises in the left colon.

Certain technical details are of importance in every intestinal anastomosis. The anastomosis must lie without any tension. There must be an adequate blood supply and, preferably pulsating arteries should be demonstrated over the cut edge of the bowel. The stoma should be made in such fashion that there is an adequate lumen. Two layers of sutures are preferred to assure a tight closure—the outer of interrupted cotton or silk and the inner of continuous fine chromic catgut. Finally the rent in the mesentery must be closed to avoid any chance of postoperative herniation of a loop of small bowel. The anastomosis preferably is made in an end-to-end fashion, though, in certain instances the surgeon may feel that after resection of the right colon, the lumen of the terminal ileum is so small that he prefers side-to-side ileocolostomy. Postoperatively the proximal intestine must be kept decompressed for several days until the patency of the anastomosis is established.

When the carcinoma has produced complete intestinal obstruction, a much more dangerous situation has been produced. In about half of the patients, the ileocecal valve is so competent that the distended colon cannot decompress itself by evacuation into the ileum. Hence the Levin type or any of the long intestinal tubes cannot be relied upon to relieve large bowel obstruction. Furthermore, the distended cecum ruptures

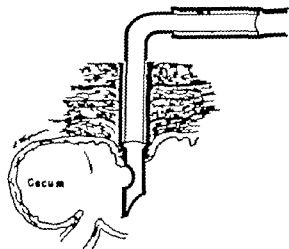
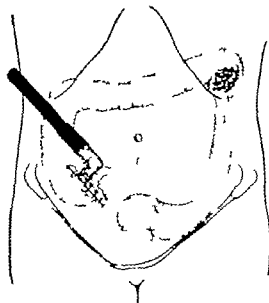


Figure 80 Cecostomy. A cecostomy decompresses the colon, but does not sidetrack the entire fecal stream. Here, by a modification of the Gibson technique, a large rubber tube, supported by a right-angle glass tube, has been sutured in the cecum.

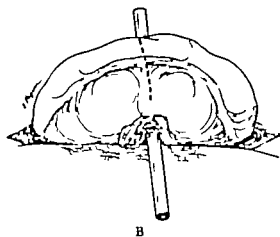
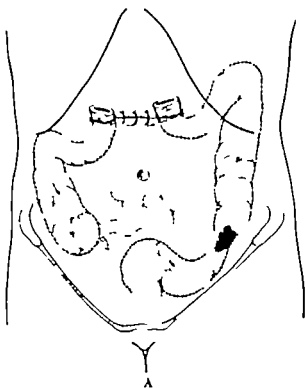


Figure 61 Transverse colostomy. A transverse colostomy can defunction all the colon distal to it. Two types are shown here. A, The colon has been completely divided and the ends brought out at the margins of the skin incision. B, In this loop colostomy a section of the transverse colon has been delivered and is held in place by a glass tube passed through the mesentery. The colon is opened along the dotted line as soon as the peritoneal cavity has sealed off.

at a comparatively low pressure so it is essential that colonic obstruction be relieved by operation as soon as possible after the patient arrives in the hospital.

When the obstruction is in the right colon, primary right colectomy is the operation of choice, though a side-to-side ileocolostomy will relieve obstruction if the ileocecal valve is incompetent. When the obstruction is in the left colon, a one-stage resection and anastomosis is hazardous, particularly in the presence of distention so that the obstruction must be relieved first, either by cecostomy or transverse colostomy (Figs. 60 and 61).

The operative results vary considerably in different reports, depending upon material observed and the attitude of the surgeon. In a series of patients studied by Allen, Donaldson and me 323 persons with cancer of the colon were observed from 1943 to 1954. Although no hope of cure could be entertained in many because of distal metastases 96.6 per cent had resections of the tumor. The operative mortality of patients who had resections with the hope of cure was 2.3 per cent. The results showed that considering all patients seen, 47 per cent were alive five years after operation. The most important feature was that 66 per cent of all patients

who had resections for cure were still alive five years later.

READING REFERENCES

- Adams, R., and Parsons, L. J.: Tuberculosis of the Cecum. *New England J. Med.* 224: 315, 1941.
- Allen, A. W.: The Development of Surgery for Cancer of the Colon. *Ann. Surg.* 134: 785, 1951.
- Allen, A. W., Donaldson, G., Sniffen, R. C., and Goodale, F., Jr.: Primary Malignant Lymphoma of the Gastro-Intestinal Tract. *Ann. Surg.* 140: 428, 1954.
- Bacon, H. E.: *Anus, Rectum, Sigmoid, Colon*. Philadelphia, J. B. Lippincott Company 1949.
- Colcock, B. P.: Colostomy: Historical Role in the Surgery of the Colon and Rectum. *Surgery* 31: 704, 1952.
- Cole, W. H.: Recurrence in Carcinoma of the Colon and Proximal Rectum Following Resection for Carcinoma. *Arch. Surg.* 65: 264, 1952.
- Cole, W. H.: Data presented at American College of Surgeons Meeting, Stockholm, 1953.
- Dennis, C., and Karlson, K. E.: Surgical Measures as Supplements to Management of Idiopathic Ulcerative Colitis, Cancer, Cirrhosis and Arthritis as Frequent Complications. *Surgery* 32: 892, 1952.
- Fisher, E. R., and Turnbull, R. B.: The Cytologic Demonstration and Significance of Tumor Cells in the Mesenteric Venous Blood in Patients with Colorectal Carcinoma. *Surg. Gynec. & Obst.* 100: 102, 1955.
- Gilchrist, R. A., and David, V. C.: Consideration of Pathological Factors Influencing Five-Year Sur-

- vival in Radical Resection of Large Bowel and Rectum for Carcinoma, *Ann. Surg.* 126:421, 1947.
- Gross, R. E. *The Surgery of Infancy and Childhood.* Philadelphia, W. B. Saunders Company, 1953.
- Jeghers, H., McKusick, V. A., and Katz, K. H. Generalized Intestinal Polyps and Melanin Spots of the Oral Mucosa, Lips and Digits. *New England J. Med.* 241:993, 1949.
- Judd, E. S. Jr. and Carlisle, J. C. Polyps of the Colon. *Arch. Surg.* 67:353, 1953.
- Lillehei, R. C. and Wangenstein, O. H. Bowel Function after Colectomy for Cancer Polyps and Diverticulitis. *J.A.M.A.* 159:183, 1955.
- Madsen, S. M., Dockerty, M. D. and Waugh, J. M. Venous Invasion in Carcinoma of the Rectum as Evidenced by Venous Radiography. *Surg. Gynec. & Obst.* 99:170, 1954.
- McKuttrick, L. S. and Moore, F. D. Ulcerative Colitis. Ileostomy Problem or Solution? *J.A.M.A.* 139:201, 1949.
- Ottensmeyer, E. J. and Oughterson, A. W. Observations on Cancer of the Colon and Rectum in Connecticut, Analysis Based on 5572 Proved Cases. *New England J. Med.* 252:561, 1955.
- Ripstein, C. B., Miller, G. G. and Gardner, C. M. Results of Surgical Treatment of Ulcerative Colitis. *Ann. Surg.* 135:14, 1952.
- Schatzki, R. The Roentgenologic Differential Diagnosis between Cancer and Diverticulitis of the Colon. *Radiology* 34:651, 1940.
- Singleton, A. O. The Blood Supply of the Large Bowel with Reference to Resection. *Surgery* 14:328, 1943.
- Spratt, J. S., Jr., Ackerman, L. V. and Moyer, C. A. Relationship of Polyps of the Colon to Colonic Cancer. *Ann. Surg.* 148:682, 1958.
- Svensson, O., Rheinlander, H. F. and Diamond, L. Hirschsprung's Disease: New Concept of Etiology. Operative Results in Thirty-four Patients. *New England J. Med.* 241:551, 1949.
- Swinton, V. W. and Doane, W. A. The Significance and Treatment of Polyps of the Colon and Rectum. *New England J. Med.* 240:673, 1953.
- Turell, R., Krakauer, J. S. and Maynard, A. de L. Colonic and Anorectal Function and Disease. *Internat. Abstr. Surg.* 96:313, 1953.
- Welch, C. E. *Intestinal Obstruction.* Chicago, Year Book Publishers, 1958.
- Welch, C. E., Allen, A. W. and Donaldson, G. E. An Appraisal of Resection of the Colon for Diverticulitis of the Sigmoid. *Ann. Surg.* 133:332, 1953.
- Welch, C. E., McKuttrick, J. B., and Behringer, G. Polyps of the Rectum and Colon and Their Relation to Cancer. *New England J. Med.* 247:959, 1952.
- Wilson, G. S., Dale, E. H., and Brice, O. A. An Evaluation of Polyps Detected in 20,847 Routine Sigmoidoscopic Examinations. *Am. J. Surg.* 90:534, 1955.

The Anal Canal and Rectum

By J. PEERMAN NESSELROD, M.D.

JERROLD PEERMAN NESSELROD, a North Dakotan, had his college and medical education at Northwestern University. With an excellent basic education in anatomy preceding his surgical training, he has devoted himself to proctology and the development of proctoscopic cinematography.

The all too common mismanagement of anorectal problems reflects a lack of understanding of the fundamental principles involved in this field of surgery. To appreciate fully the diagnostic pitfalls and the proper treatment of anorectal deformities, diseases and injuries, a review of anorectal anatomy and neurophysiology is helpful.

A common error on the part of both patient and physician is to consider all trouble pertaining to the outlet of the intestinal canal as "rectal trouble." Paradoxically, the neurophysiology of the terminal gut is such

that the rectum is frequently the site of serious disease which is asymptomatic early in its course and is therefore discovered late—often too late—unless a thorough examination is made. The neurophysiology of the anal canal is such that involvement of these tissues is promptly called to the patient's attention and, thus, to the attention of the physician. In fact, anal disease is much more common than rectal disease.

Anal canal (anus) is the terminal segment of the intestinal tract. It varies in length from 1.5 cm. (in t) anus f

male) to 3 cm. It is bordered proximally by the dentate margin and distally by the anal verge (Fig. 82).

Rectum is the terminal segment of the colon proper. Its length is about 15 cm. The rectum is bounded inferiorly by the dentate margin. Superiorly, it joins the sigmoid colon at approximately the level of the second or third piece of the sacrum. This juncture, by no means distinct, is called the rectosigmoid.

Anorectum is a term which has crept into recent surgical literature. It pertains, of course, to both anal canal and rectum. The practical value of this term is to emphasize the importance of the anal canal (anus) as well as of the rectum.

Developmental Anatomy. The tissues forming the anal canal are derived from the embryonic proctodeum and are thus ectodermal in origin. The rectum developed from the hindgut, is endodermal in origin. The anal plate, which early occludes the anorectal outlet, disintegrates in the seventh week of fetal life. Partial or complete failure of any or all of these complex processes results in congenital deformities such as partially imperforate anus (congenital anal stenosis), completely imperforate anus, abnormal location of the anus, arrested descent of the rectum, atresia of the rectum and communication of the rectum with the bladder, urethra, uterus, vagina or perineum.

Gross Anatomy. **Anal canal (anus).** The anal canal is lined by thin anal skin, not mucous membrane, and is surrounded by the external and internal sphincteric muscles

which hold the anal walls in apposition and thus occlude the canal during its normal state of rest. The fibers of the anal levator muscles pass between the external and internal sphincteric muscles to find attachment to the lateral anal walls (Fig. 82). The long axis of the anal canal extends antero-superiorly toward the umbilicus.

The serrate dentate margin (Fig. 82) forming the superior boundary of the anal canal comprises the anal papillae and the intervening anal valves. Immediately external to and slightly inferior to the dentate margin are the anal crypts, eight to twelve in number. Anal skin invests the component parts of the dentate margin and partially lines the crypts. Vestigial anal glands communicate with the crypts via the anal ducts.

Support to the anal canal is provided by the sphincteric musculature, the anococcygeal body, the perineal body and the pelvic diaphragm (the anal levator and coccygeus muscles and their investing fasciae).

Rectum. The mucosal lining of the distended rectum (i.e. from one atmosphere of pressure) is smooth except for the three prominent reduplications of mucous membrane which form the rectal valves of Houston.

The lower (distal) half of the rectum is retroperitoneal. The upper (proximal) half of the rectum is invested by peritoneum on its anterior and lateral walls (Fig. 82). Although the entire posterior rectal wall follows closely the sacrococcygeal curve, the

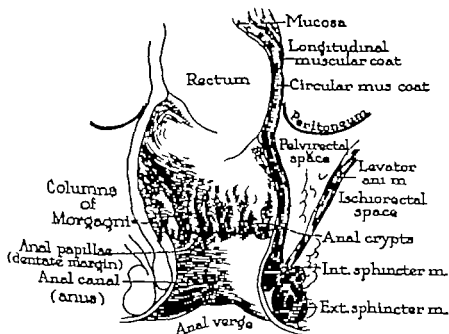


Figure 82. Diagrammatic representation of rectum and anal canal (coronal section)

Neurophysiology The practical importance of the nervous physiology of the ano rectum is twofold. Anal lesions promptly cause sufficient sensory disturbance usually pain, to bring the patient to his physician without delay. Rectal lesions on the contrary do not cause symptoms early resulting in late discovery or failure in discovery of serious rectal disease unless the lesion is adjacent to the dentate margin. Thus the absence of the sensation of pain in rectal mucosa provides a treacherous pitfall in the discovery of precancerous lesions and of early asymptomatic cancerous lesions. This same absence of pain sensation allows the surgeon to remove tissue from a rectal lesion for microscopic study and to apply electro-surgical methods in the removal or destruction of polyps without anesthesia.

These facts are pertinent to the establishment of four important rules:

1. The principal indication for proctoscopy is a patient.
2. Every patient whose history provides the slightest indication of disease of the terminal gut is a candidate for proctoscopy.
3. No physical examination is complete without proctoscopy.
4. Every candidate for proctoscopy harbors a serious lesion until the contrary is proved.

Surgical Spaces. A surgical space is potential only but can be established as a reality by abscess formation or by dissection. There are five such spaces about the anorectum. The two ischio-rectal spaces lie beneath the pelvic diaphragm and are therefore infra-diaphragmatic. The two pelvirectal spaces and the single retrorectal space lie above the pelvic diaphragm and are thus supra-diaphragmatic. The surgical anatomy and relationships of the anorectal surgical spaces are shown in Figure 84.

Relations. The relations of the rectum to its neighboring structures are of practical

importance first with regard to physical diagnosis and second concerning surgical procedures.

Anterior to the distal half of the rectum in the male lie the prostate, the seminal vesicles and ducts and the urinary bladder. On palpation through the anterior rectal wall, the examiner can readily feel the prostate. He can also palpate the seminal vesicles if they are distended.

Anteriorly in the female are the vagina, the uterus and anterolaterally uterine adnexa. The accessibility of the uterine cervix to palpation through the rectovaginal septum enables the physician more safely to follow the course of cervical effacement and dilatation during the first stage of labor.

Extrarectal masses in the cul-de-sac in either sex are accessible to palpation through the anterior rectal wall. Nodular masses in the cul-de-sac may be "drop" metastases from an abdominal malignant growth. This anatomic relationship is of further practical importance in that the anterior wall of the proximal half of the rectum, above the level of the peritoneal reflection, provides a proper site for drainage of an abscess in the cul-de-sac. In the female patient such an abscess can be drained vaginally through the posterior fornix.

The important structures lying lateral to the rectum are the potential pelvirectal space, the pelvic diaphragm, the sacrospinous and sacrotuberous ligaments, the ischial spine, the sacral plexus and the piriformis muscle.

An abscess in the pelvirectal space will thus encroach on the rectal lumen from the involved side.

Of the anatomic landmarks lying lateral to the rectum the ischial spine is the most prominent. It serves to guide the examiner's finger to the immediately adjacent region of the greater sciatic foramen where the sciatic plexus and the piriformis muscle lie.

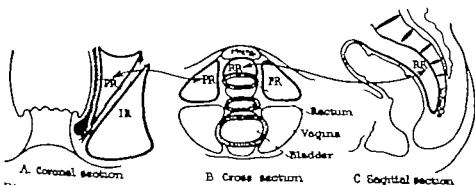


Figure 84 Diagrammatic representation of anorectal surgical spaces. PR, Pelvirectal space. RR, Retrorectal space. IR, Ischio-rectal space or fossa (modified from Nesselrod, J. P.: *Clinical Proctology*)

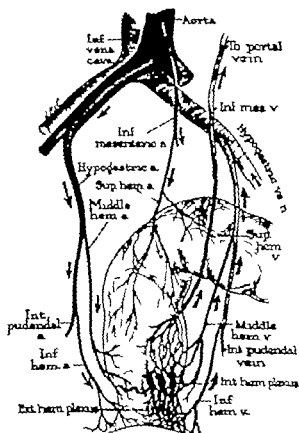


Figure 83 Diagrammatic representation of blood supply and venous drainage of anorectum

long axis of the retroperitoneal portion of the rectum lies at a right angle to the long axis of both the anal canal and the proximal portion of the rectum. These anatomic facts are of practical importance to the student in his appreciation of both digital and endoscopic study of the terminal gut.

The principal supports of the rectum are the pelvic diaphragm, the rectal stalks and the mesorectum.

Blood Supply The single superior hemorrhoidal artery and the paired middle and inferior hemorrhoidal arteries provide the principal blood supply of the anorectum (Fig. 83). The superior hemorrhoidal artery is the terminal branch of the inferior mesenteric artery. The middle hemorrhoidal artery arises from the hypogastric artery and the inferior hemorrhoidal artery is internal pudendal in origin. Since the internal pudendal artery too is hypogastric in origin, the surgical significance of the ligation of both hypogastric arteries, in addition to ligation of the inferior mesenteric artery in the present-day performance of combined abdominoperineal proctosigmoidectomy can be readily appreciated.

Venous Drainage. Venous drainage is provided by a single superior hemorrhoidal vein

and by paired middle and inferior hemorrhoidal veins (Fig. 83). The internal portion of the hemorrhoidal venous plexus lies submucosally immediately proximal to the dentate margin, while the external part lies subcutaneously immediately distal to the dentate margin. From this plexus the hemorrhoidal veins take origin. The single superior hemorrhoidal vein is tributary to the portal system via the inferior mesenteric and splenic veins. The paired middle and inferior hemorrhoidal veins are tributary to the hypogastric veins and thus to the caval system. This anatomic arrangement is another point of practical importance with reference to the spread of infection and of malignant disease.

Lymphatics. The collecting vessels arising from the anal and perianal lymphatic plexus of origin are among the afferent vessels of the inguinal lymph nodes. These constitute an inferior pedicle or "downward zone of spread" (Viles).

The submucosal lymphatic plexus of origin is poorly developed in the lower rectum but constitutes a rich network above the rectal columns. The rectal collecting vessels and nodes accompanying the superior hemorrhoidal inferior mesenteric artery comprise the superior pedicle of lymphatic collectors and correspond to the "upward zone of spread" (Viles). This pedicle is the principal lymphatic drainage of the rectum.

The middle pedicle, or "lateral zone of spread" (Viles) accompanying the middle hemorrhoidal artery is thought to be the least important of the three.

Nerve Supply The integument lining the anal canal and investing the structures of the dentate margin, together with the perianal skin receives a somatic sensory nerve supply from the primary posterior divisions of the sacral and coccygeal nerves and from the primary anterior divisions of the second, third and fourth sacral nerves by way of the inferior hemorrhoidal branches of the pudendal nerve.

A somatic motor supply is provided for the external anal sphincteric muscle via the inferior hemorrhoidal nerves and for the anal levator muscles via branches from the sacral plexus. The fibers of these muscles are striated. The internal anal sphincter and the remainder of the musculature of the terminal gut, being composed of smooth muscle fibers, receive a visceral motor supply through the autonomic nervous system.

The mucous membrane of the rectum is believed to receive a visceral sensory supply by way of autonomic nerves.

mon to all the ordinary forms of anorectal inflammatory disease *Stage III* the final stage, will be discussed separately under each of the entities presented. Thus each of the common disorders which have been listed is in reality a manifestation of anal infection. It is a common occurrence for patients to present any combination of these disorders as the result of anal infection.

HEMORRHOIDAL DISEASE

Hemorrhoidal disease pertains to any and all manifestations of hemorrhoids. Hemorrhoids, in turn, are vascular tumors made up of infected varices involving a part or all of the hemorrhoidal venous plexus (Fig. 86 A and B).

Mentioned frequently as causes of hemorrhoids are such factors as heredity, occupation, erect posture of man, lack of valves in the veins of man, portal obstruction, straining at stool, pregnancy and parturition. These factors, whatever their importance may be, serve only as contributing causes of hemorrhoids.

The principal etiologic agent in hemorrhoidal disease is anal infection.

The third stage of anal infection follows upon invasion of perianal and perirectal tissues by infectious material from the bowel. The normally thin walled hemorrhoidal

venous plexuses are thus subjected to periphlebitis, endophlebitis and further thinning of their walls with resultant dilatation in varying degrees.

Hemorrhoidal disease may vary from an asymptomatic, chronic inflammatory reaction seen only on microscopic study of the tissues to an acute disabling episode characterized by severe pain, edema of anal tissues, multiple thromboses, gangrene, sloughing and ulceration easily recognized clinically.

Pain is a prominent symptom of an external thrombosed hemorrhoid, as one might expect in view of the somatic sensory nerve supply of anal tissues, and is directly proportional to the amount of tension within the tissues. The "sore lump" of which the patient complains is due mostly to the clotted blood beneath the anal skin.

Bleeding occurs from a thrombosed external hemorrhoid only when the overlying skin has undergone erosion with resultant partial or complete extrusion of the clotted blood.

The symptoms suggestive, but by no means pathognomonic, of internal hemorrhoids are bleeding and protrusion. The patient may observe fresh blood on the toilet tissue in the water in the toilet bowl or on his clothing. This, however, is of academic

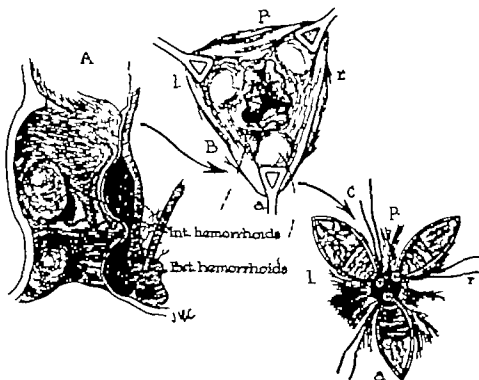


Figure 86 Hemorrhoids. A, Coronal section showing both surface view and section of hemorrhoidal tissue. B, Hemorrhoids as seen at beginning of hemorrhoidectomy. C, Hemorrhoidectomy complete; ligature-excision technique.

The sacrum and coccyx lie posterior to the rectum. Between the sacrum and rectum is a potential space known as either the retrorectal space or the presacral space (Fig. 84 B and C). Any mass in this space inflammatory or neoplastic, if it be of sufficient size will tend to obliterate the hollow of the sacrum and will encroach on the rectal lumen from behind.

The examiner can easily palpate and manipulate the coccyx by means of the examining index finger and the thumb. He can then sweep the index finger laterally and anteriorly to follow the sacrospinous ligament to the ischial spine and the sacrotuberous ligament to the ischial tuberosity. The findings may be of significance pertaining to coccygodynia and/or to otherwise unexplained pain which the patient thinks is "rectal" in origin.

ETIOLOGY AND PATHOGENESIS OF ANORECTAL INFLAMMATORY DISEASE

The principal underlying cause of the common anorectal inflammatory diseases,

such as hemorrhoids, anal fissure, anal abscess, anal fistula and anal stenosis which is spontaneous in origin, is anal infection. Anal infection pertains to the orderly sequence of events occurring in the development of these entities. Anal crypts, anal ducts, anal glands and adjacent blood vessels and lymphatics are the anatomic structures involved in this process.

Anal infection can be divided into stages (Fig. 85).

In *stage I* infectious material from the bowel content invades one or more anal crypts and the tiny vestigial anal ducts and glands attached thereto. Thus, Nature provides portals of entry into the perianal and perirectal tissues. Neither cryptitis nor trauma plays an essential role.

In *stage II* infection is carried into the perianal and perirectal tissues indirectly by way of the lymphatics or directly through breaks in the continuity of the gland-duct structure resulting possibly from overdistention.

Stages I and II of anal infection are com-

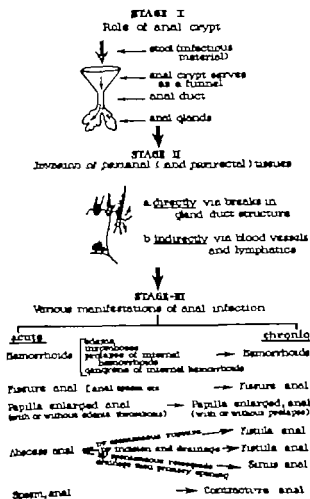


Figure 85 Diagrammatic representation of anal infection (Nusslehd, J. P.: C)

ment of hemorrhoids is threefold the destruction of all external hemorrhoidal tissue, all internal hemorrhoidal tissue and all anal crypts, in view of the etiologic importance of anal infection.

There are three indications for adequate surgical treatment of hemorrhoidal disease: repeated attacks of "painful hemorrhoids," bleeding which may be responsible for hypochromic anemia, and annoying prolapse of internal hemorrhoids. Any one of these alone is a sufficient reason for surgical treatment.

The great majority of hemorrhoidal problems requiring surgical management are amenable to a ligature-excision type of hemorrhoidectomy (Fig. 86 C) which in the hands of the experienced surgeon is a remarkably flexible procedure. For the patient whose hemorrhoidal deformities involve the entire circumference of the anorectal outlet, an amputative-plastic procedure is indicated.

ANAL FISSURE

Although an anal fissure appears to be a crack in the anal skin, it is in reality an ulcer involving the entire thickness of the skin of the anal wall usually the posterior anal wall, and lying in the long axis of the anal canal (Fig. 87 A).

The chief etiologic agent in anal fissure is anal infection, the first two stages of which have been described. Infectious material having gained entrance to anal tissues, infection can localize superficially in the subcutaneous tissues, inviting dissolution of the overlying anal skin (stage III of anal infection). The resulting defect in the anal skin is an anal ulcer. Failure of the ulcer to heal spontaneously or to respond to palliative treatment is due to the continuous mechanism of anal infection.

The characteristic symptom of anal fissure is cyclic pain of varying degrees of severity, initiated by defecation and lasting from several minutes to several hours. The pain is due to spasm of the anal sphincteric musculature and endures until the muscle is completely fatigued. Since muscle fatigue is accompanied by a subsidence of anal spasm, the patient gains welcome relief from his pain until the next occurrence of defecation.

Additional symptoms are slight bleeding at stool and a gradual trend toward constipation as a result of fear of painful defecation. In some instances of prolonged suffering from anal fissure, there may be abdominal discomfort, headache, irritability and nervousness.

An anal fissure may become sufficiently quiescent to cause little or no discomfort, giving rise to the erroneous conception of spontaneous healing.

The diagnosis is based upon the almost pathognomonic history of cyclic anal pain related to defecation and upon demonstration of the lesion (Fig. 87 A).

Gentleness in the handling of anal tissues in the presence of an acutely painful lesion is mandatory. Careful separation of the margins of the anus will disclose at least the distal part of the lesion. Exposure of the entire fissure is often difficult because of the accompanying anal spasm.

When the outlet is relaxed as the result of regional anesthesia, the margins of the fissure can be retracted easily, revealing the presence of a definite ulcer. Figure 87 A calls attention to an enlarged anal papilla proximal to the fissure. Immediately beyond the distal border of the fissure there may be a piling up of skin-covered tissue beneath which is frequently found a shallow pocket. This structure constitutes a sentinel tag, so-called because it seems to stand guard, as it were, at the distal border of the lesion.

Differential diagnosis necessitates consideration of a simple anal abrasion, specific venereal lesions such as chancre and chancreoid, a tuberculous ulcer, the ulcer of agran-

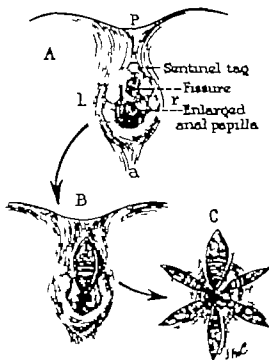


Figure 87 Anal fissure. A, Posterior anal fissure. B, Anal fissure excised. C, Multiple cryptotomy completed.

interest, because a serious lesion must always be considered the source of the blood until the contrary is proved. The loss of blood from internal hemorrhoids at any one time is of little significance compared with the summation of such losses of blood over a period of time, leading to hypochromic anemia.

Protrusion or prolapse of internal hemorrhoids occurs initially at defecation and recedes spontaneously. In time the prolapse may occur with other forms of abdominal straining, such as coughing and lifting. Digital replacement of the prolapsed hemorrhoids becomes necessary. Ultimately, barring corrective treatment, the internal hemorrhoidal tissue remains prolapsed constantly, resulting in a discharge of blood and mucus which is irritating to the anal skin and which stains the clothing.

Diagnosis in any anorectal problem depends on careful and meticulous performance of inspection, digital palpation and proctoscopy. In the presence of an exquisitely painful anal lesion, proctoscopy is deferred until suitable anesthesia is provided, usually at the time of surgical intervention.

A thrombosed external hemorrhoid is found at the anal margin as a tender bluish-colored swelling. The overlying skin is tense.

Differential diagnosis of external hemorrhoids requires consideration of anal epithelioma, condyloma latum, condyloma acuminatum and sentinel tag. The slightest doubt calls for biopsy.

Internal hemorrhoids are not visible on external inspection unless they protrude or unless the anal margins can be easily retracted. They are not palpable except when induration is present as the result of a recent inflammatory process. Internal hemorrhoids can be visualized readily by means of an anoscope or a proctoscope and are seen to be covered by a velvety rectal mucosa. Hemorrhoidal tissue, both external and internal, is commonly arranged in three groups: one on the left, one on the right posterior and one on the right anterior (Fig. 86 B). There may, however, be such extensive involvement of the hemorrhoidal plexus that hemorrhoidal tissue is arranged around the entire circumference of the anorectal outlet.

Acute, disabling, hemorrhoidal disease affects all parts of the hemorrhoidal plexus. A "collar" of edematous anal tissue containing multiple thromboses surrounds the anal outlet. The involvement of anal tissues is responsible for the disabling pain. In the center of the ring of external hemorrhoids are seen three masses of prolapsed internal

hemorrhoids. The internal hemorrhoidal tissue is edematous and may present discoloration, ulceration and gangrene in varying degrees and combinations. The term "strangulated hemorrhoids," often applied here, may lead to the belief that the strangulation is due to spasm of the sphincteric musculature. Actually, the examining finger can be inserted easily into the patient's anal canal, showing the absence of anal spasm. The strangulation is due to blockage of the internal hemorrhoidal varices by numerous thromboses.

Internal hemorrhoids should be differentiated from enlarged anal papillae, sessile and pediculate polyps and carcinoma. Anal papillae are invested by skin rather than by rectal mucosa, can be shown to arise from the dentate margin and are sensitive to pain stimuli. The microscopic study provides the final word with regard to neoplasm.

Protruding internal hemorrhoids require differentiation from an enlarged anal papilla with prolapse, a pediculate polyp with prolapse and rectal prolapse, either partial or complete.

The simplest management of a thrombosed external hemorrhoid seen within twenty-four or forty-eight hours is incision of the overlying anal skin, permitting extrusion of the clotted blood. Relief from pain is usually prompt because of the release of tension within the sensitive anal tissues. Such incision, however, is commonly inadequate, favoring the formation and retention of another clot.

Excision of the thrombosed hemorrhoid, employing infiltration of procaine to produce local anesthesia, is a more satisfactory method. It entails, of course, postoperative care.

In the instance of an acute disabling attack of hemorrhoidal disease, conservative management, i.e., bed rest, ice packs and anodynes is preferred. The risk of surgical intervention, when the tissues are edematous and are host to numerous small, infected thrombi, is great; chemotherapeutic agents and antibiotics notwithstanding.

Since sclerotherapy is applicable only to internal hemorrhoidal tissue, it cannot be considered a curative measure. Too this method entails some risk. For these reasons the use of injection treatment should be limited to the patient whose problem is persistent bleeding from internal hemorrhoids and for whom adequate surgical treatment is contraindicated.

The objective in adequate surgical treat-

Throbbing pain and the classical signs of acute inflammation render the diagnosis of a superficial abscess rather easy.

The discovery of a deep (supradia-phragmatic) abscess depends upon a careful digital examination of the patient's rectum. A "doughy" swelling is found encroaching on the rectal lumen. The swelling is posterior in the instance of a retrorectal abscess and lateral in the instance of a pelvirectal abscess.

Any draining sinus lying in anal para anal or perineal skin must be considered to be a secondary opening of an anal fistula until proved otherwise. The opening may be bordered by granulation tissue and the surrounding skin is discolored. The opening may be found in scar tissue. Too it may appear to be healed. Multiple openings are not uncommon.

Careful bidigital examination reveals a firm, subcutaneous "cord" leading from the sinus toward the anal wall. In reality the cord leads from its primary source in the dentate margin to the opening in the skin. The cord of tissue is the fistulous tract or tube (Fig. 88 H). The exact origin in the dentate margin can be palpated if there is sufficient scarring. The digital examination may reveal also a submucosal cord arising at the level of the dentate margin and ending in a puckered mucosal scar. This process represents the internal limb of an anal fistula (Fig. 88 D).

Direct visualization of the dentate margin may disclose pus oozing from the involved crypt or sufficient deformity from scarring to identify the guilty crypt.

The use of probes in exploring the fistulous tract should be postponed until the time of surgical treatment.

A superficial abscess arising posterior to the anus must be differentiated from an acutely inflamed pilonidal cyst. The presence of one or more pilonidal openings may facilitate the diagnosis.

Draining sinuses which are pilonidal in origin have been found lateral to and anterior to the anus.

Perineal sinuses require consideration of disease and/or deformity of the genitourinary tract.

Multiple draining sinuses, especially when no anal origin is found suggest the possibility of hidradenitis suppurativa involving the perianal apocrine glands.

Osteomyelitis of the pelvic bones may occasionally explain a draining sinus in the anal or para anal tissues.

Either a true sinus or a fistula can result from a penetrating injury. The history of course is helpful in such instances.

Sacrococcygeal sinuses originating in pre sacral tumors and cysts are rare but must be considered.

An abscess requires drainage. The choice of a point for incision is important. If fluctuation is present the incision should be made at that point, but, in addition, it should be carried as near the anal verge as possible, without damage to sphincteric muscle in order to shorten the resulting fistulous tract. Overhanging skin should be excised to provide adequate drainage.

In the case of a pelvirectal abscess, with or without involvement of the ischioanal

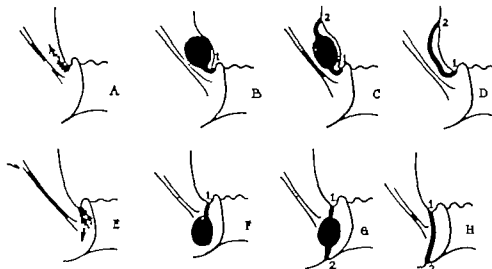


Figure 88. Diagrammatic representation of pathogenesis of anal fistula. A, B, C and D pertain to a deep or supradia-phragmatic abscess and its resulting fistula. E, F, G and H pertain to a superficial or infra-phragmatic abscess and its resulting fistula. 1, Primary opening, 2, secondary opening.

ulocytic angina, ectropion and anal epithelioma.

Some relief can be had by means of such palliative measures as hot packs sitz baths anal hygiene and anesthetic ointments. Cure however depends on adequate surgical treatment and proper after-care. The surgical procedure comprises wide excision of the anal ulcer including the underlying anal crypts the enlarged anal papilla and the sentinel tag (Fig. 87 B).

The anal crypts in the remaining quadrants should be destroyed by means of exteriorization (multiple cryptotomy) as a prophylactic measure (Fig. 87 C). Adequate surgical treatment is aimed not only at destruction of the anal fissure but also at avoidance of further anal infection.

ANAL ABSCESS AND ANAL FISTULA

Anal abscess and anal fistula are not separate entities. The former is a stage in the development of the latter. Thus there is ample logic in Buie's presentation of these subjects under the same heading.

Anal fistula is a tube inflammatory in origin, having its primary opening in an anal crypt. The secondary opening, or openings may lie in anal, para-anal or perineal skin or in rectal mucous membrane (Fig. 88 H and D).

Anal sinus is a tube inflammatory in origin, having its primary opening in an anal crypt and ending blindly in para anal or pararectal tissue. Obviously a sinus has no secondary opening.

Proper understanding of the accepted definitions of "fistula" and "sinus" should obviate further usage of such misleading terms as "blind internal fistula," "blind external fistula" and "incomplete fistula."

The chief cause of anal abscess and of the resultant anal fistula is anal infection, the earlier stages of which have been described. Thus, infectious material gains entrance to para-anal and pararectal tissues (Fig. 88 E and A). Although the anal ducts and glands lie superficially in anal subcutaneous tissue they are occasionally sufficiently complex in structural arrangement to extend into the superficial fasciculi of the sphincteric musculature. Also on occasion, they are seen to communicate with a crypt of Lieberkühn immediately proximal to the mucocutaneous juncture. Hence, infection can localize in the tissues of the ischioanal fossa on one or both sides (infradiaphragmatic spaces) and, less commonly within the rectal wall or in

the tissues of any or all of the potential pararectal spaces above the pelvic diaphragm (supradiaphragmatic spaces).

Such contributing factors as anatomic variations virulence of the infecting organisms and resistance of the tissues of the host no doubt play a role in determining the clinical course and manifestations of the inflammatory process.

The acute phase of stage III of anal infection pertains here to abscess formation (Fig. 88 B and F). This phase soon passes into a chronic phase by one of three methods: spontaneous rupture of the abscess, incision for drainage of the abscess or spontaneous retrograde drainage of the abscess through its crypt of origin (Figs. 85 and 88 C and G). Either of the first two methods results in the establishment of an anal fistula (Fig. 88 D and H). The third method determines an anal sinus.

The abscess can involve any or all of the anorectal surgical spaces. Likewise the resulting fistulous process can have one or several limbs and one or several secondary openings. An internal limb has its secondary opening, or openings in rectal mucosa (Fig. 88 D). The secondary opening, or openings of an external limb lie in skin (Fig. 88 H).

Throbbing pain is the outstanding complaint when the abscess involves anal, para-anal or perineal tissues since these tissues have a somatic sensory nerve supply. Such an abscess can involve a part or all of the ischioanal fossa, and is considered to be superficial because it is below or inferior to the pelvic diaphragm.

In the instance of a deep abscess involving a supradiaphragmatic space or of a submucosal abscess pain is entirely absent early in the course of the trouble or is present only as a vague discomfort in the region of the rectum. Pain is not prominent until the process involves infradiaphragmatic tissues possessing a somatic sensory nerve supply.

The patient harboring a deep abscess is acutely ill. He presents chills fever and a generalized malaise. Until he develops localized pain "in the rectum" the diagnosis is likely to remain undetermined or in error.

The chief complaint of the patient who harbors an anal fistula is periodic, spontaneous drainage by way of the secondary opening, with prompt relief of any discomfort which exists immediately prior to the drainage. This safety valve mechanism often diverts a recurrence of the initial acute abscess.

the little finger and finally the index finger can be passed.

The treatment of acquired contracture is preventive whenever possible. In routine postoperative care and in the care of injuries to anal tissues, the surgeon must learn to be constantly aware of the probability of contracture and must therefore make every effort to prevent such a complication. This includes separation of wound margins, digital dilation, irrigation of wounds and sitz baths, daily during the first week and at frequent intervals thereafter until healing is complete and satisfactory.

The corrective treatment of acquired contracture is surgical. Anal skin and subcutaneous scar tissue are incised radially at one or more points until ample room is provided in the anal canal. All anal crypts present are destroyed by means of exteriorization (cryptotomy) (Fig. 87 C). Thus circumferentially arranged scar tissue is broken up by multiple radial incisions. The correction established here surgically must be maintained during healing by means of meticulous postoperative care.

BENIGN RECTAL STRICTURE

Benign strictures of the rectum may be either congenital or acquired, the latter in turn, may be extrinsic, intramural or intrinsic in origin. Any extrarectal process, whether neoplastic or inflammatory can encroach on the rectum with resultant narrowing of its lumen. In these instances, however the rectal mucosa is usually intact, being involved only secondarily when there is malignant invasion of the rectal wall. Benign strictures which are intrinsic in origin may be the result of inflammatory disease, of trauma, of surgical procedures or of radiation therapy.

In the light of recently acquired knowledge, the most common cause of inflammatory rectal stricture is the virus of venereal lymphogranuloma (lymphogranuloma venereum). As the name implies, this entity is venereal in origin. Since the lymphatic drainage of the external genitalia is inguinal in either sex, a primary lesion of the external genitalia in the male or the female is followed shortly by inguinal adenitis. In the female however the primary lesion is likely to be established in the vagina or on the cervix. As a result the virus can invade the perirectal and rectal lymphatics either by way of the plexus of the rectovaginal septum or through the uterosacral ligaments (rectal stalks). In the male it is possible that the

infection may travel along the urethra into the lymphatics of the prostate and thence to the rectal absorbents by way of the rectal stalks. In either sex infection may spread cephalad from an anal lesion, through lymphatic vessels which connect the anal absorbent plexus with that of the rectum. Lymphatic stasis in the presence of infection favors the growth of fibroblasts. The fibrous tissue thus formed undergoes contraction. The resultant inflammatory stricture occurs usually in the lower half of the rectum. The Frei test and a complement fixation test are established diagnostic procedures.

Surgical procedures are often necessary but the results are disappointing. The newer treatment of venereal lymphogranuloma includes such antibiotics as Aureomycin and chloramphenicol.

However drug therapy has thus far proved useless once deforming stricture has occurred. For advanced rectal stricture the best plan appears to be to establish a permanent colostomy and then, if necessary to remove the rectum by means of a combined abdominoperineal operation.

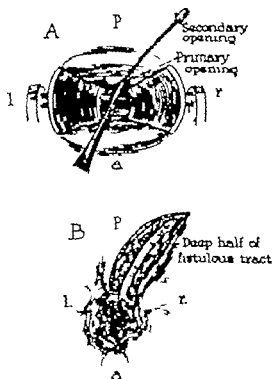
RECTAL PROLAPSE

The terms prolapsus and procidentia—meaning a falling down—are synonymous.

Bule's classification of rectal prolapse is both simple and logical. He divides the types of prolapse into those which are visible and that which is concealed. The visible prolapse may be partial, consisting only of rectal mucosa (Fig. 90 A) or complete, involving all the layers of the rectal wall. In the first degree of complete rectal prolapse the wall of the anal canal is everted too so that there is no groove or sulcus surrounding the protruding mass of tissue (Fig. 90 B). In complete prolapse of the second degree the anal structures retain their normal positions accordingly there exists a sulcus entirely surrounding the prolapsed gut (Fig. 90 C). Concealed rectal prolapse (complete prolapse of the third degree) is an intussusception of the upper portion of the rectum and rectosigmoid into the lower portion of the rectum (Fig. 90 D).

The chief underlying factor in the etiologic background of rectal prolapse is an alteration or defect in the supporting structures of the anorectum. The exciting causes are mechanical strain, disease and injury.

Partial prolapse must be differentiated from hemorrhoidal prolapse: in many instances the two conditions are associated.



Figures 89 Anal fistula. A, Probe lying in fistulous tract. B, Fistulotomy completed deep half of fistulous tract lying in base of wound.

fossa, drainage should be established through the ischiorectal fossa and the pelvic diaphragm on the involved side rather than through the rectal wall. In the instance of a retrorectal abscess drainage should be established on either side of the anococcygeal ligament and as near the anus as possible. Fistulotomy should follow in four to seven days.

The treatment of anal fistula is fistulotomy which entails laying open the entire tract from its primary source to its secondary opening and excision of overhanging margins (Fig. 89). An essential requirement is adequate relaxation of the anal outlet, best provided by means of sacral anesthesia.

The preference for fistulotomy rather than for fistulectomy is based upon the following considerations:

1. Fistulotomy follows logically upon the conception of the pathogenesis of anal fistula as has been presented. The same can be said of fistulectomy but

2. Fistulotomy is a much less extensive surgical procedure than is fistulectomy and yet is adequate. In the performance of fistulotomy the tissues deep to the fistulous tract are not disturbed.

3. Fistulotomy allows conservation of tissue which is useful in the healing process. The membranous lining of the remaining deep half of the fistulous tract quickly ac-

quires a covering of granulation tissue. Scarring following this procedure is much less than that following fistulectomy.

4. Fistulotomy necessitates the sectioning of only those fibers of sphincteric musculature which are superficial to the fistulous tract.

Here, perhaps more than in any other problem of anorectal surgery meticulous postoperative care is mandatory.

ANAL CONTRACTURE (STENOSIS, STRICTURE)

The terms anal contracture, anal stenosis and anal stricture pertain to narrowing of the anal outlet (anus).

Anal narrowing can be congenital in origin or acquired.

Congenital anal contracture or stenosis results from partial failure of disintegration of the anal plate in the seventh week of fetal life.

Acquired contracture can occur as a manifestation of anal infection, either alone or in conjunction with other clinical forms of anal infection such as hemorrhoids, anal fissure or anal fistula. It can develop as a result of a penetrating injury and can occur as a complication following anorectal surgery.

The mechanism in acquired contracture involves the well known tendency of scar tissue to contract. When scar tissue is arranged circumferentially rather than radially about the anus cicatricial contracture will cause narrowing of the anal outlet.

The chief complaint is difficulty in varying degree in passing stool. Pain and bleeding are common, especially if the anal skin is fissured.

In the infant an attempt at careful digital exploration, using the little finger will usually determine the diagnosis. It may be impossible at first to pass the little finger in which instance the careful use of a small Hegar dilator will aid in establishing the diagnosis and will serve to initiate therapy.

In the adult, too digital examination may be difficult, depending upon the degree of narrowing. The small-caliber endoscope is especially useful when digital exploration is unsatisfactory.

Examination with the patient under regional anesthesia is indicated when the anal outlet is rigidly scarred and markedly narrowed. Surgical correction should be undertaken at the same time.

The treatment of congenital anal stenosis in the infant entails careful dilation Hegar dilators (sizes 5 through 10) are preferred

manipulation. They were apparently able to create negative intra abdominal pressure and thereby to draw in the prolapsed gut.

In acute prolapse resulting from sudden strain or from a crushing injury the engorgement of the tissues may become so great that gangrene threatens. Reduction in such instances is a difficult problem. If the rectal wall is so friable that perforation is likely to occur the safest procedure is an amputative operation with every precaution being taken to avoid opening the peritoneal cavity and injuring the small bowel. If only the mucosa is damaged, gentle but firm attempts at reduction should be undertaken with the patient in the inverted position. If necessary, relaxation of the anal canal can be accomplished by the use of regional anesthesia. After successful replacement of the prolapsed gut it is well to confine the patient to bed until the supports of the anorectum have had an opportunity to resume a normal state.

When partial prolapse fails to respond to conservative management an amputative operation such as that described by Bule is indicated. If this procedure is carried out properly and if diligent postoperative care is provided, the outcome should be satisfactory.

The treatment of complete prolapse is usually surgical, necessitating obliteration of the cul-de-sac via an abdominal approach.

ANAL PRURITUS

Anal itching is a common disorder involving anal, perianal and perineal skin.

The many causes of anal itching can be grouped under three headings: systemic, local and psychogenic. Not uncommonly a definite causative factor remains undetermined. Local causes include such factors as improper anal hygiene, fungal infections, infestation with pinworms, irritation from overmedication (dermatitis venenata) irritation from ingestion of certain antibiotics, acute dermatitis, specific anal dermatoses such as psoriasis and seborrheic dermatitis, anal warts and venereal lesions.

Excess moisture resulting from a draining anal sinus, from an anal fissure or from mucous secretions accompanying prolapsed internal hemorrhoids or rectal prolapse, may be a factor in anal pruritus. Anal itching is common during the healing of anal wounds owing probably to secretions and to the perianal stubble of hair.

Needless to say the chief complaint is itching, which may be periodic or constant

and may vary in degree from mild to such severity that it brings the sufferer to demoralizing exhaustion.

The usual findings in pruritic anal and perianal skin are thickening, maceration and multiple excoriations. In the presence of superimposed acute dermatitis the involved skin is reddened, there is increased local heat and there may be considerable swelling. The appearance of pruritic skin is often altered by the presence of a secondary infection resulting from the trauma of scratching. Further confusion may be added by the presence of dermatitis venenata. The physical evidence of anal pruritus is limited proximally by the dentate margin.

The diagnosis of fungal and monilial infections is confirmed by microscopic study of scrapings mounted in sodium or potassium hydroxide.

Although the examiner may occasionally see the adult pinworm on the anal skin or within the rectum on proctoscopic examination he will usually establish a diagnosis of parasitic infestation by means of proper stool tests for ova.

A history of diarrhea and of persistent anal itching following the ingestion of an antibiotic is strongly indicative of the antibiotic as the causative agent. A bacterial imbalance of the intestinal flora is involved.

When the cause of the itching is known, the treatment is usually clearly indicated.

The difficult problem is the patient for whom no etiologic factor has been determined and whose anal itching has failed to respond to any of the conservative therapeutic measures, including fractional roentgen ray therapy.

A temporary interruption of sensory nerves can be accomplished by the injection of an oil soluble, long acting anesthetic agent or by an undercutting operation.

Radical treatment of anal pruritus pertains to methods by which the sensory nerve endings can be destroyed more or less permanently. These methods are alcohol injection and tattooing. Either of these methods can be used in conjunction with subcutaneous neurotomy.

PILONIDAL DISEASE

The adjective pilonidal is derived from the Latin terms pilus, meaning hair and nidus meaning nest. Pilonidal disease pertains here to the clinical manifestations, usually inflammatory of pilonidal cysts occurring in the sacrococcygeal region (Fig. 91 A).

The simplest conception of the develop-

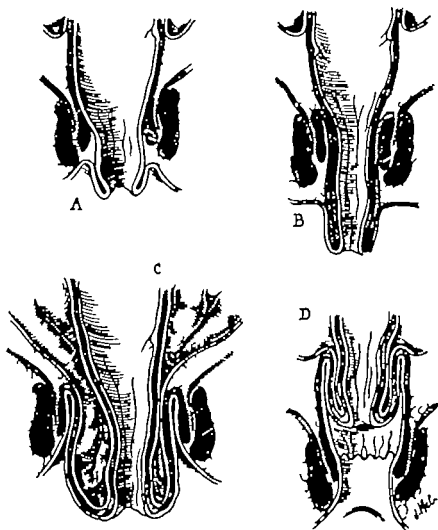


Figure 90 Diagrammatic coronal sections, modified from Tuttle, representing rectal prolapse. A, Incomplete or partial prolapse. B, Complete prolapse, first degree. C, Complete prolapse, second degree. D, Complete prolapse, third degree (Nesselrod, J. P. *Clinical Proctology*)

The concentrically arranged ridges on the surface of a completely prolapsed rectum are characteristic features; there is a striking difference between these "rings" and the radially arranged grooves or sulci seen in hemorrhoidal prolapse. Whenever there is a history of prolapse and examination fails to disclose the deformity the patient should be asked to strain on the toilet in order to demonstrate his trouble. The nature of the condition is usually rendered quite obvious. The chief diagnostic problem is that presented by concealed prolapse. Examination of the patient in the usual inverted position fails to disclose the trouble, because the intussusception is usually reduced by such a position. It may be necessary to examine the patient while he is standing upright in order to visualize the deformity.

In infants and young children presenting partial prolapse, conservative methods are

often successful. The patient should lie down to defecate and the buttocks should be firmly strapped together during intervals between defecations. In older patients with partial prolapse it may be possible to accomplish satisfactory results with linear cauterization or with injection treatment, preferably with quinine and urea hydrochloride 5 per cent solution.

The reduction of a chronically recurrent prolapse, either partial or complete, is usually accomplished easily by the patient himself who has long since become adept at manual replacement of the prolapsed tissue. In these instances the anal sphincteric musculature lacks its usual tone and offers little or no resistance. Oddly enough this atony facilitates both the prolapse and its reduction. Several patients with complete rectal prolapse have been observed who could reduce the prolapsed tissue without local

2. Simplicity and ease of performance
3. Simplicity of postoperative management.
4. Correction of an overlooked limb or sinus easily accomplished
5. Reasonable compromise in over-all time of healing.

CANCER OF THE RECTUM

Consideration of cancer of the rectum is incomplete without brief mention of pre cancerous tumors (adenomas) and their relation to rectal carcinoma.

A more common term for rectal adenoma is polyp; polypoid disease pertains to the entire subject of polyps or adenomas of the large bowel. It should be emphasized that any polypoid lesion of the gut must be considered potential cancer. Proof that an adenoma is benign is no security whatsoever against ultimate malignant degeneration of that lesion.

The majority of malignant lesions of the rectum are carcinomas and, of these, most are adenocarcinomas. The less common forms of carcinoma are carcinoma simplex and epithelioma.

Pathology Adenocarcinoma is a glandular neoplasm in which acinar structures are present in varying degrees of differentiation.

In carcinoma simplex there is little or no differentiation of the glandular elements. Absence of any differentiation amounts to complete anaplasia and signifies a highly malignant process. In the scirrhous form of carcinoma simplex, fibrous tissue predominates, the tissue is hard, and there is a tendency to contracture of the involved segment of gut. In the medullary form of carcinoma simplex there is a preponderance of glandular tissue and the growth is soft.

Epithelioma (i.e., anal epithelioma) pertains to cancer of anal and/or perianal skin. Anal epitheliomas are not common. The majority are of the squamous cell type, whereas basal cell epithelioma is a rare finding.

Sarcoma is a cancer arising from connective tissue elements. Histologically such growths can be lymphosarcoma, fibrosarcoma, myosarcoma, leiomyosarcoma and melanosisarcoma.

Spread of cancer occurs via lymphatics, blood stream, continuity of surface contiguity of surfaces and, in light of recent studies, desquamation of living cancer cells.

The role of lymphatics in the spread of cancer is of great importance in the selection, planning and execution of surgical procedures for the eradication of cancer.

The newer knowledge of spread of cancer by desquamation of cancer cells stresses the need for gentleness on the part of the surgeon in handling cancer laden tissue.

Invasion of blood vessels by rectal cancer results in dissemination via the portal venous system leading to metastatic involvement of the liver.

Symptoms and Diagnosis. Ideally cancer of the rectum should be discovered when it is "presymptomatic." The neurophysiology of the rectum is such that rectal lesions early in their course do not give rise to symptoms. Accordingly the earliest possible symptoms of malignant disease of the rectum assume great importance. These are bleeding and a change in bowel habit.

Bleeding from the rectum must always be considered to be the result of ulceration of a malignant lesion until the contrary is proved.

A steadily progressive change in bowel habit is significant as an early symptom of rectal or colonic cancer. There may be slowly increasing constipation, a feeling of unsatisfactory defecation, frequency of stool and/or urgency of stool. Diarrhea may alternate with constipation because of perverted colonic physiology, laxatives or a combination of both factors.

Pain is likely to be a late manifestation of rectal carcinoma, unless the lesion should occur near the dentate margin with early involvement of anal tissues which, it will be remembered, possess a somatic sensory nerve supply.

Obstruction can occur rather early when a growth involves a narrow portion of the gut. The two narrowest segments of the colon are the splenic flexure and the rectosigmoid. Another factor in early obstruction by a lesion in the left colon is the firmer consistency of the content of the left colon as compared with other portions of the gut. Not uncommonly therefore, the earliest symptoms of carcinoma of the rectosigmoid or the splenic flexure are those of obstruction.

The diagnosis of carcinoma of the rectum early in its course requires, first a constant awareness of the possibility of such a lesion and, secondly a thorough examination to determine the presence or absence of such a lesion.

The majority of rectal growths are within reach of the examining finger. The lesion may be a soft, polypoid mass which encroaches on the lumen of the gut but which causes little or no obstruction. This type of lesion, especially a small one, is easily missed.

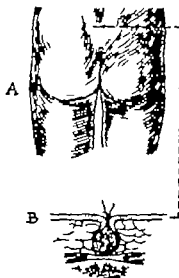


Figure 91 Pilonidal cyst. A, Pilonidal openings in skin overlying sacrococcygeal region. B, Cross section through cyst and pilonidal opening.

ment of a pilonidal cyst pertains to its congenital origin as an ectodermal rest resulting from an error in developmental anatomy. The epithelial lining of the cyst and the frequent presence of hair within the cyst suggest that ectodermal tissue destined to form skin was lost in subcutaneous tissue in or near the midline.

The cavity of the cyst communicates with the surface of the overlying skin by means of one or more short channels, each opening on the skin in the midline. Thus the lining of the cyst is in continuity at these points with skin. These are pilonidal openings, and tufts of hair are often found protruding through them.

Through the pilonidal openings organisms commonly found on skin can easily gain entrance to the cyst. Thus are provided an infecting organism and a portal of entry.

Trauma, in all probability, is the final agent involved in pilonidal disease. This is borne out by the experience of the military surgeon who sees much more pilonidal disease than does the civilian surgeon. In both military and civilian practice the majority of patients are in their late teens or early twenties, a time when the incidence of trauma is greatest. The incidence of pilonidal cyst is greater in males than in females.

Pain and swelling at "the base of the spine" are the chief complaints of the patient with acute pilonidal disease. The findings are those of acute inflammation (abscess) involving soft tissues dorsal to the sacrococcygeal juncture. One or more pilonidal openings are present.

In the patient whose abscess has ruptured

and drained spontaneously or has been incised for drainage there is a draining sinus at the point of rupture or of incision. The draining sinuses are commonly to one side of the midline. The underlying tissues are indurated and only slightly tender as compared with the exquisitely tender tissues associated with an acute abscess. This patient presents pilonidal disease in its chronic stage.

An acute inflammatory process involving the soft tissues in the sacrococcygeal region, especially when pilonidal openings are found, is most likely an acutely inflamed pilonidal cyst.

Since pilonidal cysts are more commonly found in individuals who have abundant hair in the sacrococcygeal region, it behooves the surgeon to be diligent in his search for pilonidal openings in performing routine physical examinations of hirsute persons.

In the presence of chronic pilonidal disease draining sinuses are usually found to one side of the midline and can occur lateral to the anus and even anterior to it. This may lead to some difficulty in differentiation between pilonidal disease and anal fistula. Such differentiation may have to await exploration.

The possibility of draining sinuses in this region arising from presacral tumors and cysts has been mentioned in a foregoing section.

The treatment of symptomatic pilonidal disease is surgical and comprises three methods:

1. Bloc or enucleation excision of the cyst, and primary closure.
2. Excision of the cyst, leaving the wound open to heal by second intention.
3. Compromise procedures such as excision with partial closure, and the exteriorization operation of Bule.

The exteriorization operation is preferred because nearly every pilonidal cyst is infected. This procedure consists of determining the extent of the cyst cavity and of all its ramifications by means of a probe or grooved director and then laying open the cavity and all its channels. Overhanging margins of skin are excised to provide a flat, saucerized wound, the floor of which is formed by the deep half of the lining of the cyst or channel. Thus healing is facilitated by the conservation of tissue which was destined to form skin. The advantages of the exteriorization operation are:

1. Wide applicability regardless of the extent of the process.

2. Simplicity and ease of performance
3. Simplicity of postoperative management
4. Correction of an overlooked limb or sinus easily accomplished.
5. Reasonable compromise in over all time of healing.

CANCER OF THE RECTUM

Consideration of cancer of the rectum is incomplete without brief mention of precancerous tumors (adenomas) and their relation to rectal carcinoma.

A more common term for rectal adenoma is polyp polypoid disease pertains to the entire subject of polyps or adenomas of the large bowel. It should be emphasized that any polypoid lesion of the gut must be considered potential cancer. Proof that an adenoma is benign is no security whatsoever against ultimate malignant degeneration of that lesion.

The majority of malignant lesions of the rectum are carcinomas and, of these, most are adenocarcinomas. The less common forms of carcinoma are carcinoma simplex and epithelioma.

Pathology Adenocarcinoma is a glandular neoplasm in which acinar structures are present in varying degrees of differentiation.

In carcinoma simplex there is little or no differentiation of the glandular elements. Absence of any differentiation amounts to complete anaplasia and signifies a highly malignant process. In the scirrhous form of carcinoma simplex, fibrous tissue predominates, the tissue is hard, and there is a tendency to contracture of the involved segment of gut. In the medullary form of carcinoma simplex there is a preponderance of glandular tissue and the growth is soft.

Epithelioma (i.e., anal epithelioma) pertains to cancer of anal and/or perianal skin. Anal epitheliomas are not common. The majority are of the squamous cell type, whereas basal cell epithelioma is a rare finding.

Sarcoma is a cancer arising from connective tissue elements. Histologically such growths can be lymphosarcoma, fibrosarcoma, myosarcoma, leiomyosarcoma and melanomasarcoma.

Spread of cancer occurs via lymphatics, blood stream, continuity of surface, contiguity of surfaces and, in light of recent studies, desquamation of living cancer cells.

The role of lymphatics in the spread of cancer is of great importance in the selection, planning and execution of surgical procedures for the eradication of cancer.

The newer knowledge of spread of cancer by desquamation of cancer cells stresses the need for gentleness on the part of the surgeon in handling cancer laden tissue.

Invasion of blood vessels by rectal cancer results in dissemination via the portal venous system leading to metastatic involvement of the liver.

Symptoms and Diagnosis. Ideally cancer of the rectum should be discovered when it is "presymptomatic." The neurophysiology of the rectum is such that rectal lesions early in their course do not give rise to symptoms. Accordingly the earliest possible symptoms of malignant disease of the rectum assume great importance. These are bleeding and a change in bowel habit.

Bleeding from the rectum must always be considered to be the result of ulceration of a malignant lesion until the contrary is proved.

A steadily progressive change in bowel habit is significant as an early symptom of rectal or colonic cancer. There may be slowly increasing constipation, a feeling of unsatisfactory defecation, frequency of stool and/or urgency of stool. Diarrhea may alternate with constipation because of perverted colonic physiology, laxatives or a combination of both factors.

Pain is likely to be a late manifestation of rectal carcinoma, unless the lesion should occur near the dentate margin with early involvement of anal tissues which, it will be remembered, possess a somatic sensory nerve supply.

Obstruction can occur rather early when a growth involves a narrow portion of the gut. The two narrowest segments of the colon are the splenic flexure and the rectosigmoid. Another factor in early obstruction by a lesion in the left colon is the firmer consistency of the content of the left colon as compared with other portions of the gut. Not uncommonly therefore, the earliest symptoms of carcinoma of the rectosigmoid or the splenic flexure are those of obstruction.

The diagnosis of carcinoma of the rectum early in its course requires first, a constant awareness of the possibility of such a lesion and, secondly a thorough examination to determine the presence or absence of such a lesion.

The majority of rectal growths are within reach of the examining finger. The lesion may be a soft, polypoid mass which encroaches on the lumen of the gut but which causes little or no obstruction. This type of lesion, especially a small one, is easily missed

on digital examination by the less experienced examiner

When the carcinoma has become an ulcerated, crateriform growth with firm, elevated, rolled edges and is accompanied by fixation of the involved rectal wall and by infiltration of adjacent tissues it is almost impossible to overlook such a lesion on digital examination provided the lesion involves the lower half of the rectum. By gradual encirclement of the gut, the growth becomes annular. Annular carcinoma of the rectum which is within reach of the examining finger feels somewhat like a nodular lacerated cervix.

Proctoscopic examination enables the physician to verify or to qualify the findings obtained on digital exploration of the patient's rectum and to visualize those lesions which lie beyond the reach of the finger and yet within the range of the 25-cm. proctosigmoidoscope. Should obstruction preclude the passage of the ordinary proctoscope beyond the lesion an instrument of smaller caliber can be used to complete the study and to determine the proximal level of the lesion. The distance between the anus and the distal level of the lesion or, in other words, the amount of normal bowel below or distal to the growth, is of importance in determining the type of surgical procedure best suited to the problem. As the instrument is withdrawn, the examiner can observe destruction of mucous membrane by an ulcerating carcinoma, and gangrenous tissue resulting from cancerous invasion and occlusion of the smaller blood vessels. The differentiation between a cauliflower-like polypoid lesion and an ulcerating, crateriform growth is not difficult.

Finally proctoscopic examination facilitates removal of tissue for microscopic study. In evaluating the results of a biopsy it must be remembered that the pathologist can base his opinion on only the sample of tissue provided him. Should cancer cells be present and should invasion beneath the muscularis mucosae be demonstrated, such positive information is invaluable. Should, however, the specimen for biopsy study fail to include cancer which may be present in closely adjacent tissues the resulting negative report could lead to a false sense of security and thus to a dangerous pitfall.

In most instances of rectal carcinoma, proctoscopic diagnosis should offer no difficulty. It is well, nevertheless, to bear in mind these possibilities for differential study: endometriosis in the female during her child-

bearing period, carcinoma of extrinsic origin (e.g., carcinoma of the ovary) diverticulosis and amebic granuloma.

Treatment. The anti-cancer campaign, with reference to rectal cancer should begin with careful attention to the problem of rectal adenomas or polyps which are precancerous lesions. Theoretically at least, cancer of the rectum could be prevented if all rectal polyps were found and destroyed prior to the onset of malignant degeneration. Steadily increasing interest in proctoscopy on the part of general practitioner, internist and surgeon is a step in the right direction.

It is generally agreed that malignant lesions involving the distal 6 cm. of the rectum must be removed by abdominoperineal resection which is based on a consideration of three pedicles or zones of lymphatic spread. At levels from 6 to 12 cm. above the anus, some skilled surgeons have been able satisfactorily to perform "pull through" operations which permit preservation of the anal sphincters and yet provide radical extirpation of the tumor with adequate lengths of bowel above and below. The studies of Waugh, in a sufficiently large number of patients, have demonstrated that five year survival rates are at least equal to those obtained by abdominoperineal resection. In the hands of most good surgeons the latter procedure can be applied more effectively should circumstances permit, by two teams, an abdominal team and a perineal team, working simultaneously.

Whereas the advantages of preserving the anal sphincters should not be underestimated, abdominal colostomy established properly can be managed and tolerated by the most fastidious patient.

Cancer above the levels mentioned can usually be resected widely and primary end-to-end (anterior) anastomosis performed. In some instances proximal decompression (temporary colostomy) may be necessary. At least 2.5 cm. of normal bowel distal to the lowest edge of the growth should be included in any resection. The amount of normal gut included proximal to the lesion should obviously be much greater in order to accomplish wide limits of resection for removal of lymphatic-bearing tissue.

For anal epithelioma the abdominoperineal procedure is indicated, along with dissection of both groins if inguinal adenopathy is felt to be advancing.

Palliative treatment includes colostomy for the relief of obstruction and radiotherapy for the relief of pain caused by metastatic

involvement. Chordotomy should be considered for the patient who is suffering intractable pain

READING REFERENCES

- Bale, L. A.: Practical Proctology Philadelphia W B Saunders Company 1937
- Daland, E. M., and Mitchell, L. G.: Cancer of the Rectum Results of Treatment of All Cases Admitted to Poodville Hospital, June 1927 through December 1946. Surg. Gynec. & Obst. 87 105 1953.
- David, V. C., and Gilchrist, R. K. Abdominoperineal Removal of Low Lying Cancer of the Rectum; Five Year Cures and Local Recurrences. Surg. Gynec. & Obst. 89:31 1949.
- Glover R. P., and Waugh, J. M. The Retrograde Lymphatic Spread of Carcinoma of the Rectosigmoid Region; Its Influence on Surgical Procedures. Surg. Gynec. & Obst. 82:434 1946.
- Goensey D E., Waugh, J. M., and Dockerty M. B. Carcinoma of the Rectum; Prognosis Based on the Distance of Lesion from the Levator Ani Muscle and Involvement of the Anal Sphincters. Surg. Gynec. & Obst. 92:529, 1951

- Vesselrol, J. P.: Clinical Proctology 2nd ed. Philadelphia, W B Saunders Company 1957
- Pfeiffer D B., and Miller D B. Cancer of the Rectum Five and Ten Year Follow-up Study of Cases of Cancer Below the Peritoneal Reflection. Surg. Gynec. & Obst., 91 310 1950
- Turrell, R., Krakauer J. S., and Maynard, A. deL. Colonic and Anorectal Function and Disease. Internat. Abstr. Surg. 96 313, 1953.
- Wangensteen, O. H. Primary Resection (Closed Anastomosis) of Rectal Ampulla for Malignancy with Preservation of Sphincteric Function Together with a Further Account of Primary Resection of the Colon and Rectosigmoid and a Note on Excision of Hepatic Metastases. Surg. Gynec. & Obst. 81 1 1945.
- Wangensteen, O. H. and others. An Interim Report upon the "Second Look" Procedure for Cancer of the Stomach, Colon and Rectum and for Limited Intra-peritoneal Carcinoma. Surg. Gynec. & Obst. 99:257 1954.
- Wekh, C. S., and Rhinelanders H. F. Radical Abdominal Proctosigmoidectomy with Preservation of the Anal Sphincter. Surg. Gynec. & Obst., 84 550 1952.

Intestinal Obstruction

By ROBERT M MOORE M D

ROBERT MILD MOORE grew up in a small town in Kansas, the son of a doctor. Educated at the University of Chicago the University of Kansas and the medical school of Washington University he has his surgical background in physiology. A former fellow of the National Research Council in this subject he spent several years in the experimental surgical laboratory before he became Professor of Surgery at the University of Texas.

Intestinal obstruction remains one of the commonest and most dangerous conditions in abdominal surgery. Any serious interference with the passage of content along the lumen of the intestinal tract leads to symptoms of major severity. If the obstruction is complete, the secondary effects set in motion are intense and may result fatally in a few days time. Only through prompt diagnosis and immediate institution of proper therapy can relief be afforded and a disastrous outcome avoided.

GENERAL CONSIDERATIONS

Since any interference with the normal downward passage of intestinal content is in reality an intestinal obstruction, a variety

of situations and mechanisms may be included under the term. At one time the word *ileus* was used to denote any form of obstruction mechanical or functional. It has since become customary to restrict its use to those interferences which arise from abnormalities in the propulsive force of the musculature of the intestinal wall, *paralytic ileus* (also called *inhibition ileus* or *adynamic ileus*) and *spastic ileus* (*dynamic ileus*). Aside from such conditions obstruction arises so preponderantly from mechanical lesions which interrupt the continuity of the intestinal lumen that in everyday usage the term intestinal obstruction ordinarily denotes *mechanical obstruction*.

An obstruction may be referred to as high

or low. Probably it is best to differentiate between *small bowel obstruction* and *large bowel obstruction* by using those terms and to reserve the adjectives high and low to indicate differing clinical pictures in obstructions at contrasting levels of the small intestine.

Many mechanical obstructions lead to interruption of the circulation to the intestinal loop involved, as well as to obstruction of its lumen. Such circulatory obstruction is called *strangulation* and the obstruction producing it is termed a *strangulating obstruction* as opposed to a *simple* or *nonstrangulating obstruction*. Unless relieved, strangulation leads to gross gangrene of the affected intestinal segment. It is seen when the mesentery is twisted, as in sigmoid volvulus or is constricted and compressed as at the entrance to an irreducible hernia. It also occurs not infrequently in obstructions caused by adhesion bands where a secondary torsion of the obstructed loop is not uncommon. Consequently the surgeon must keep constantly mindful of the risk of strangulation in every patient with mechanical obstruction of the small intestine.

A danger of necrosis and perforation, moreover is not limited to instances of mesenteric strangulation. A number of other mechanisms which impair the circulation may develop. Necrosis from external pressure upon the wall of the bowel is sometimes noted in nonstrangulating obstructions. For example, when the intestine is trapped by an unyielding adhesion, the edematous swelling of the bowel wall at the point of encirclement may result in local gangrene. A similar necrosis is considered to be an imminent threat in the Richter type of femoral hernia. Harmful effects arising from increased internal pressure in turn, constitute the mechanism in other cases. As distention advances the progressive increase in the tension within the bowel lumen finally obliterates the circulation through the overlying intestinal wall. In the ordinary case of simple obstruction, anemic necrosis of the bowel wall from such excessive distention is encountered only as a late effect and in neglected patients. In any obstruction which partakes of the "closed loop" character however a necrotizing intraluminal

pressure may develop relatively early. The designation *closed loop obstruction* refers to a situation in which the lumen of the intestinal segment is occluded at both ends so that there can be neither downward passage nor regurgitation. Thus, in the loop intervening between two separate mechanical obstructions no means is provided for escape of contents in either direction. The majority of strangulated loops fall into this category. For example, in volvulus there is occlusion of the lumen of both afferent and efferent limbs and the same situation or dinarily exists in strangulated hernia. However the closed loop concept has its foremost clinical application in relation to colon obstructions.

Other terms used in differentiating types of intestinal obstruction are *acute* and *chronic obstruction* and *complete* and *partial obstruction*. An obstruction is termed *chronic* when it has been partial or intermittent or when there have been recurring episodes or threats of obstruction. Since any obstructive episode is an immediate menace to the individual, it is safest to consider all obstructions or threats of obstruction as *acute* until relieved. Too often the term chronic obstruction lulls the surgeon into a false sense of security in dealing with obstructions having an insidious onset, as do, for example, certain obstructions of the large bowel.

A broad classification of intestinal obstruction might include three groupings: *mechanical obstruction*, both simple and strangulating, *neurogenic obstruction* or *ileus*, and *vascular obstruction* this last referring to the relatively infrequent obstructions arising from embolism or thrombosis of a main mesenteric vessel such as the superior mesenteric artery. As has been noted, the vascular occlusion seen in the local mesenteric sector which has been twisted by a volvulus or compressed in a hernial ring or in an intussusception, is secondary to a mechanical force which has obliterated both the intestinal lumen and the local vessels. *Vascular obstruction*, in distinction, refers to an intestinal obstruction which has resulted from a primary vascular disease, or vascular accident. The principal effect upon the intestine is a complete or nearly complete denial of blood supply ordinarily to a loop of great length. In occasional patients, the arterial occlusion is partial so that the affected bowel regains sufficient circulation to escape gangrene. Even in such patients, however there is an interim which

In describing irreducible hernias, the term *incarceration* is sometimes used interchangeably with strangulation. However, an incarcerated loop is not necessarily strangulated. Sometimes, adherent intestinal coils are imprisoned or incarcerated in a wide-necked hernial sac for a long time before strangulation or even complete obstruction of the lumen develops.

Table 10. Clinical and Pathologic Classification of Types of Intestinal Obstruction*

CLINICAL CLASSIFICATION	PATHOLOGIC CLASSIFICATION
<p>I. Mechanical obstruction</p> <p>A. Narrowing of lumen</p> <p>1. Structures of intestinal wall</p> <p>a. Congenital atresia, stenosis, imperforate anus</p> <p>b. Acquired inflammatory traumatic vascular neoplastic</p> <p>2. Obstruction</p> <p>3. Compression from without (especially in pelvis and at retroperitoneal duodenum)</p>	<p>Simple (except in complete colon obstruction when distention may have necrotizing effect on wall of bowel)</p>
<p>B. Intestinal obstruction due to adhesions and bands</p> <p>congenital inflammatory traumatic neoplastic</p>	<p>Either simple or strangulating</p>
<p>C. Hernia, external or internal</p> <p>D. Volvulus</p> <p>E. Intussusception</p> <p>F. Developmental errors giving rise to obstruction (in addition to congenital atresia and stenosis)</p>	<p>Strangulating</p>
<p>II. Intestinal obstruction due to nervous imbalance</p> <p>A. Inhibition ileus (paralytic or adynamic)</p> <p>B. Spastic ileus (dynamic)</p>	<p>Simple</p>
<p>III. Vascular obstruction</p> <p>A. Obstruction due to mesenteric thrombosis or embolism</p>	<p>Strangulating</p>

Modified from Wangenstein in Christopher: A Textbook of Surgery 5th ed.

the intestine is unable to perform its muscular functions. Forward propulsion ceases as a result and the loop becomes dilated. At this stage the situation is one of ileus from ischemic paralysis of the intestinal wall. It amounts actually to a truer paralysis than do most cases of so-called paralytic ileus since the latter generally are due to excessive nervous inhibition of the intestinal muscle rather than to a real loss of muscle power.

A detailed classification based on the clinical and pathologic types of intestinal obstruction is given in Table 10. While in practice the type of obstruction is well defined clinically in many cases in others an accurate differentiation may be difficult or impossible. The clinical picture results from a combination of primary and secondary elements. Furthermore, the characteristics of an obstruction often are changing because of the factor of *elapsed time since onset of obstruction*. An obstruction which begins as a simple obstruction of the lumen of the intestine may become a strangulating obstruction as distention develops and added

mechanical factors come into play. Therefore, despite the usefulness of a detailed classification, it is important to remember that an obstruction may change its character. Treatment must be governed accordingly and whenever operative measures are deferred on the basis that an obstruction is of the simple or nonstrangulating variety the possibility of strangulation having intervened must be reassessed at frequent intervals.

MECHANICAL OBSTRUCTION

Some of the mechanisms by which the intestine may become obstructed are illustrated in Figures 92 and 93. Data upon the relative incidence of the various causes of obstruction vary greatly depending upon the age and sex of the clientele of the hospital or surgeon reporting. In a children's hospital, many newborn babies and infants are treated for intestinal obstruction arising from such congenital anomalies as atresia, stenosis, intestinal malrotation and congenital hernia. Of these congenital causes so fre-

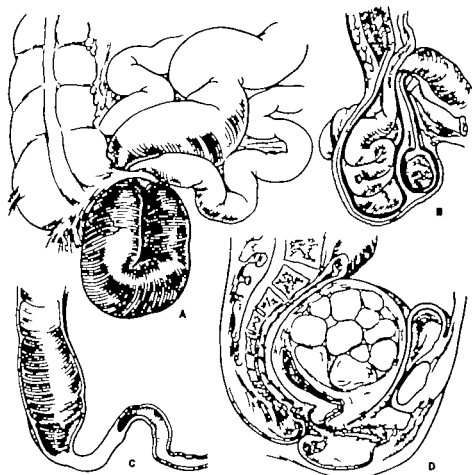


Figure 92 Common mechanisms of intestinal obstruction. A, Constriction by *adhesion bands*. Such bands may be congenital (as in the duodenal obstruction of malrotation) or acquired (inflammatory adhesions and postoperative adhesions). The constriction deformity often is aggravated by angulation of the bowel. B, Constriction at the neck or ring of a *hernia*. When an intestinal loop protrudes through a narrow rigid opening it tends to become irreducible, obstructed and strangulated. When the hernial opening is broad, however, adherent or "incarcerated" loops may occupy the sac for some time before acute complications arise. C, Developmental failure in formation of a continuous intestinal lumen. *Atresia* of the intestine in the newborn is found most commonly in the ileum or in the duodenum. Discontinuity of the lumen of the large intestine occurs chiefly at the rectum (*imperforate anus*). D, Compression by an *extrinsic tumor*. The pelvic colon may be compressed, for example, between a tumor and the rigid bony wall of the pelvis. Such obstruction usually is incomplete.

quent in babies only strangulated inguinal hernia also serves as a common mechanism to cause obstruction in adults. Intussusception, which is not related to any congenital anomaly is another frequently occurring type of obstruction during the earliest years of life.

Approximately two-thirds of all obstructions arise either from strangulation in hernias or from acquired or congenital adhesion bands, these types of obstruction being common at all ages. In men, strangulation of inguinal hernia is most frequent as opposed to strangulation of femoral or of umbilical hernia in women.

As individuals pass middle age, the incidence of obstruction caused by cancer in creases greatly. Cancer of the intestine, except for secondary lesions, is almost always

situated in the colon. This is borne out by the observation that while carcinoma accounts for only about 10 per cent of all intestinal obstructions it causes approximately 70 per cent of the obstructions of the large bowel. Next in frequency to carcinoma in causing colon obstruction is volvulus of the sigmoid, which accounts for perhaps one out of six of the acute obstructions of the large bowel. Aside from ileocolic intussusception in small children and the occasional case of imperforate anus in the newborn, large bowel obstruction is relatively rare in the younger age groups.

Physiopathology Any mechanical obstruction to the normal downward passage of content within the intestinal lumen leads to developments of an emergency nature. Since the ingestion of food can be interrupted for

weeks before life is threatened the pressing dangers of intestinal obstruction obviously are not related to starvation. In strangulating obstructions the immediate threat is readily apparent if the strangulated loop is left *in situ* until necrosis and dissolution occur overwhelming peritonitis almost certainly will follow. In simple or nonstrangulating obstructions in contrast, dangers which are very real are at first glance much less obvious.

Whenever the forward propulsion of content is blocked by occlusion of the intestinal lumen, a sequence of local reactions is set in motion. These local changes and their systemic sequelae account for the critical symptoms and the rapid death attending neglected simple obstruction. The primary lethal processes have been identified by frequent clinical demonstration that when strangulation is absent the patient's condition usually can be maintained for several days without great deterioration provided two requirements can be met. First, distention of the proximal intestine must be prevented and, second, dehydration and electrolyte loss must be

corrected effectively by parenteral replacement. Therefore the immediate dangers in nonstrangulating obstruction stem from *distention and water and electrolyte loss*. The triad of primary lethal effects of mechanical obstruction will be completed should *strangulation* enter the picture.

The initial local response to obstruction of the intestinal lumen is a marked *increase in peristaltic activity* in the loop just proximal to the obstruction. As this intestinal segment attempts to drive its contents past the obstructive barrier the waves of peristalsis become progressively more forceful and violent. They are the source of the sharp intermittent or cramping pain—the “colic” which leads the clinician to suspect that an obstruction is present.*

Colic, or intermittent pain synchronous with recurrent waves of abnormally violent peristalsis, is a characteristic symptom of obstruction of any tubular passage having walls of smooth muscle. Painful contraction waves corresponding to those in the obstructed intestine occur regularly when a stone blocks the ureter (ureteral colic) or a bile duct (biliary colic) or even, as occasionally happens, the appendix (appendiceal colic).

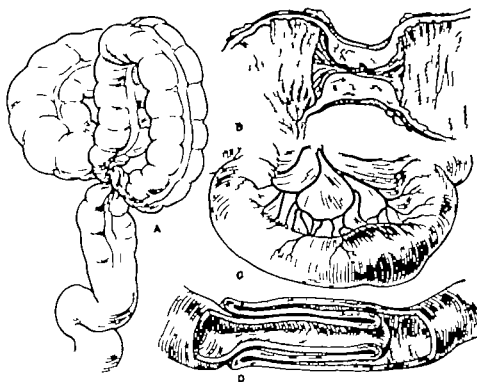


Figure 93 Common mechanisms of intestinal obstruction. A. Volvulus, or twisting of an intestinal loop, may result from congenital anomalies (an example is the midgut volvulus of an incompletely anchored mesentery) or may develop later in life (volvulus of a redundant sigmoid colon in the adult). B. Narrowing from causes intrinsic in the intestinal wall. There may be congenital stenotic areas in the newborn. Acquired strictures may be inflammatory as in sigmoid diverticulitis, or neoplastic, as in carcinoma of the colon. C. Obstruction. Rarely the intestinal lumen becomes blocked by a foreign body by an enterolith or by a mammoth gallstone which has ulcerated its way from the gallbladder into the duodenum. D. Intussusception, or the telescoping of a proximal loop of intestine into a distal loop. In infants, over 90 per cent of intussusceptions are of unknown origin or idiopathic. At any age a polyp or other intestinal tumor may serve as the “leading point” to initiate intussusception.

By this abnormally increased peristaltic activity the intestine actually traumatizes itself and the edema of the intestinal wall which follows is to some degree a result of this injury. Proximal to the point of obstruction, edema may be marked by the thinning effect of distention but at the point engaged in the obstruction there is actual swelling. Since any thickening of the wall at this point must be at the expense of the narrowed lumen the edema often serves to render complete an obstruction which was only partial at its onset. Furthermore it frequently contributes significantly to the irreducibility of the obstruction.

Distally the intestine lies empty, contracted and inactive while proximal to the obstruction there is a rapid accumulation of bowel content. For the resulting distention to be marked, it is not necessary for oral intake to continue. Under ordinary circumstances the gastrointestinal tract secretes daily into its lumen as much as 8 liters of digestive fluids and this secretion continues in part even when nothing is ingested. Normally reabsorption is at such a rate that only a small volume of water (100 to 150 cc.) is lost in the stool. Obstruction upsets this balance however. Secretion into the intestinal lumen may actually be increased whereas the normal absorptive power is significantly lacking. This combination of increased secretion and defective absorption is typical of overdistended intestine.

The degree of the distention of the proximal intestine depends, of course upon the completeness of the obstruction and upon the time which has elapsed since its onset. Although dilatation is greatest immediately above the obstruction the entire proximal tract eventually becomes involved. Except in obstructions located high in the jejunum, the aggregate volume of the distended coils is sufficient to create a serious problem in abdominal distention, the tightly expanded abdomen interfering mechanically with such vital functions as respiration and venous return.

Often the obstruction represents an abnormal point of fixation in the small intestine and the weight of the distended proximal loop results in its sagging at this point to superimpose acute angulation as an added obstructing mechanism.

The content of the distended intestine is both liquid and gaseous. The liquid element is made up of accumulated secretions, food residues and the products of bacterial multiplication. The gas, as shown by many meas-

urements, consists largely of swallowed air (70 per cent). Both nausea and thirst aggravate the patient's tendency to swallow air. Air is in great part nitrogen (80 per cent) an inert gas which is absorbed extremely slowly. As a consequence, air swallowing is a major factor in abdominal distention. Only by the use of some decompressing device which recovers the air immediately as it is swallowed can distention be prevented.

In unrelieved intestinal distention, the progressive increase in intraluminal tension interferes eventually with the flow of blood through the wall of the bowel. Usually the anoxia affects the mucous membrane first, the submucosal vessels being particularly vulnerable to the flattening force exerted by the pressure from within the lumen. There is increasing edema which may be followed by actual necrosis even of the entire thickness of the wall. Such necrosis from excessive intraluminal tension can be observed in the experimentally produced closed loop of intestine (a segment which has been occluded at both ends by suturing). It is also seen occasionally as a late event in patients with neglected simple small bowel obstruction. Its greatest practical importance, however, lies in the necrosis and "spontaneous rupture" which may be encountered relatively early when complete obstruction of the left colon occurs in patients having a competent or "one-way" ileocecal valve. In this important group of patients, the development of obstruction distal to that valve amounts to completion of a closed loop, since the intervening bowel thus becomes shut off at both ends. Even though the secretions of the colon are not bulky, the formation of putrefactive gases and the continued entry of content from the ileum may combine to distend the loop until the intraluminal tension is dangerously high. Such patients demand prompt decompression by operation in order to avoid the risk of necrosis and perforation.

In all acute abdominal disorders, there is apt to be an initial reflex emptying of the stomach. In intestinal obstruction, the vomiting continues, however, and tends to be frequent and copious. It may be brought on

Prior to such decompression, care must be exercised not to administer diagnostic barium enemas under excessive pressure. Distention of the rectum normally evokes reflex peristaltic contraction of the colon, and spontaneous rupture of the distended bowel, usually at the rectum, may be precipitated by the violent reflex contraction evoked when the rectum is overdistended.

by attempts to ingest food or drink and it occurs typically after a crescendo of painful peristaltic contractions reversal of flow having finally regurgitated intestinal content into the stomach, which then ejects the material. This regurgitant nature of the vomiting in obstruction is illustrated by the frequent observation that when the obstruction is high in the small intestine the vomiting begins early whereas its onset often is delayed when the obstruction is at a lower level. Moreover vomiting may be entirely absent in colon obstructions.

In late cases of intestinal obstruction the distended and thin walled proximal loops lose all effective contractile power. The colicky waves of peristalsis diminish in vigor at this stage and often the vomiting amounts only to the stomachs emptying itself of a passive overflow from the distended intestine. The vomitus may be dark and odorous and this has given rise to the term fecal vomiting. Such vomitus however usually does not come from the colon but represents the results of stagnation and bacterial multiplication in the dilated small bowel.

A number of mechanisms take part in the severe water and electrolyte loss characteristic of advanced intestinal obstruction. The deficit from interruption of oral intake and the loss from vomiting are readily apparent and require no detailed comment. Less obvious, but equally significant, are certain internal or hidden depletions.

Since the absorptive power of the intestinal mucosa becomes increasingly defective as distention develops, gastrointestinal secretions which are poured into the obstructed intestine often are lost to the body just as effectively as are those which are vomited. Furthermore the dilatation of the bowel may serve by irritation actually to increase the flow of secretions. In low obstructions as a consequence the proximal intestine fills with an accumulation of water and essential electrolytes which is of no avail whatever to the body. In this situation, dehydration and electrolyte loss may be well under way even before the advent of vomiting.

At an early stage of obstruction, a sizeable loss of plasma may be in process. This loss, which consists in the escape of protein containing transudate through the edematous wall of the intestine and into the peritoneum,

Although reversed peristalsis has been observed in the duodenum, it is the consensus that in lower obstructions regurgitation actually is the result of normally directed peristaltic waves which force the intestinal content against the obstruction until finally the stream reverses itself to surge proximally

increases dehydration and adds the element of protein depletion.

In the relatively infrequent obstructions arising very high in the small intestine the vomiting tends to be immediate and constant and results in an imbalance approaching somewhat that seen in pyloric obstruction. Dehydration being accompanied by a greater loss of chloride ion than of sodium, results in hyponatremia and an alkalosis which is evidenced by elevation of the carbon dioxide combining power of the blood. In obstructions at lower levels the vomiting is usually intermittent and less copious at the start and since the loss of sodium ion equals or exceeds that of chloride ion the patient is prone to develop an acidosis unless there has been adequate parenteral replacement. In patients with neglected obstruction at either level, severe dehydration leads to hemoconcentration and to oliguria, and often renal damage is reflected in an elevated nonprotein nitrogen in the blood. Potassium deficiency is another complicating factor in late cases, as is also the sizeable loss of plasma through the bowel wall. The presence of hemoconcentration tends to mask these deficits and laboratory data must be interpreted with this effect in mind.

In simple obstruction, the intestinal wall is reddened and edematous, although the thinning effect of distention may make the edema less apparent. The distended loop often "weeps" so that a serous transudate collects in the peritoneum. If the obstruction produces strangulation, the affected loop shows the changes of hemorrhagic infarction. Its color varies from gray to bluish black depending upon the degree and duration of the vascular involvement. Ordinarily the veins are occluded first by the strangulating force and for a time the systolic pressure continues to drive arterial blood into the congested intestinal wall. There is a consequent rupture of venules with blood escaping into the intestinal lumen, sometimes in a quantity sufficient to cause hemorrhagic shock. There may be a similar escape of blood from the serosal surfaces of the strangulated intestine and mesentery so that the peritoneal fluid takes on a sanguineous character. Finally the mounting tension within the lumen of the strangulated loop stops all capillary circulation in the wall and frank gangrene follows. At this stage the arteries as well as the veins of the involved mesenteric sector have become thrombosed and release of the loop from the strangulating mechanism no longer restores their pulsa-

tions. The gangrenous intestine soon perforates but even before gross perforation is evident the necrotic wall has served as a portal through which bacteria and toxic agents have escaped into the peritoneal cavity.

The escape of bacteria and toxins through the wall of the gangrenous intestine contributes the final, and most deadly threat in intestinal obstruction. Once this stage is reached, the mortality rate will be formidable no matter how energetic and skillful the treatment. It is borne out by long experience of surgeons that intestinal resection necessitated by gangrene of an obstructed loop carries much greater risk to life than does intestinal resection performed for other reasons. The difference of course arises from the fact that, by the time gangrene has developed, the peritoneum has already been subjected to a heavy dose—perhaps a lethal dose—of noxious contamination by agents set free from the intestinal lumen.

When gangrenous intestine disintegrates within the abdominal cavity the intestinal content pours into the peritoneum. With rare exceptions the contamination becomes generalized and the outcome is overwhelming peritonitis. In that third or more of obstructions which are caused by strangulated hernias, however, the strangulated loop does not lie within the abdomen proper but in a separate peritoneal sac or compartment. In these cases there is the possibility that at the narrowed neck of the hernia the natural defenses will succeed for a time in confining the contamination to the hernial sac and that during this time peritonitis will remain localized. However one can never rely on such localization, even temporarily and hence there is necessity for prompt surgical intervention whenever strangulation is suspected.

The harmful effects of bacterial activity in intestinal obstruction are not limited to this late phase of peritonitis following bowel necrosis. Bacteria are also influential in earlier developments. Their mere multiplication in the stagnant content of the obstructed intestine contributes to distention by adding the bulk of bacterial growth as well as the gases created by their action. Bacterial activity also augments the liquid content since it increases intestinal secretion, an effect evidenced by demonstrations that, in experimentally produced closed loops of intestine, agents which inhibit bacterial activity serve to diminish secretion. Finally bacteria play an important role in initiating

and accelerating the gangrene which results from strangulation. There have been repeated demonstrations that control of bacterial activity through intestinal antiseptics delays and limits tissue necrosis when a loop is partially devitalized. Bacterial invasion of the bowel wall is a potent factor in causing venous thrombosis to precipitate tissue death. This is particularly telling in areas where blood supply is deficient but not entirely lacking. In such marginal and less extensive areas of defective circulation, collateral blood supply can sometimes be established in time to avert actual gangrene if invasion by bacteria can be prevented in the interim.

It has always been difficult to understand why certain patients die from simple or non-strangulating obstruction. As a consequence, a *toxemia theory* has often been proposed to explain such deaths on the basis of perverted absorption from damaged intestine. Thus, for example systemic effects were attributed at one time to products of protein breakdown not absorbed in the normal bowel. Histamine and histamine-like substances were suggested particularly as responsible toxins. Although experimental tests have failed consistently to identify any specific substance certainly the possibility exists that when intraluminal tension is greatly elevated the blood vessels and lymphatics of the edematous bowel wall may take up products which are toxic systemically. There has been little positive evidence however that such absorption is actually appreciable so long as the layers of the wall retain their viability. Too often the symptoms arising from severe dehydration and electrolyte losses have been attributed to a conjectured toxemia.

Once the viability of the intestinal wall is impaired, the situation is radically different. Necrotic tissue then offers an easy avenue for direct escape of bowel content into the peritoneum. Absorption through a serous membrane, such as the peritoneum, is much less selective than is absorption through the intestinal mucosa and there is little doubt but that many toxins of bacterial and non-bacterial origin are readily absorbed by the peritoneum. Consequently fever and toxemia are no longer a mystery once there is gangrene and peritonitis.

In summary from the standpoint of the pathologic processes which endanger the life of the patient as a result of intestinal obstruction, one can conclude that so long as the circulation through the intestine is intact the chief dangers from obstruction of the

lumen lie in the water and electrolyte losses and in the mounting intestinal distention. If distention progresses unrelieved, the intraluminal tension may eventually become increased to the point that the viability of the bowel wall is impaired. Bacterial influences then join in posing the threat of necrosis with perforation and peritonitis. Fortunately, except in closed loop cases this stage is encountered infrequently in simple or nonstrangulating, obstruction and only as a late event. In strangulating obstructions, in contrast, circulatory impairment constitutes the immediate danger. Although obstruction of the lumen usually precedes occlusion of the vessels, it is the latter which leads to irreversible gangrene, some times within a period of hours. Properly then, whenever there is a threat of strangulation that danger takes precedence over all others.

Diagnosis. The diagnosis of acute intestinal obstruction is based upon the symptom triad, *cramping abdominal pains, vomiting and failure to pass gas or feces by rectum*. Although distention becomes of great significance as obstruction advances in the initial stages of small bowel obstruction, when diagnosis is particularly to be desired, distention may be slight and does not afford a dependable diagnostic criterion.

The clinical picture of acute intestinal obstruction is seen in its typical form when the small intestine becomes obstructed. The patient, an individual of any age who may or may not have experienced a similar previous episode, is seized rather suddenly with *cramping abdominal pains*. These pains, which are synchronous with waves of forceful peristalsis, are usually felt neither to the right nor to the left but in the midabdomen, or "put of the stomach," at the level of the umbilicus or higher. The patient is apt to describe them as *gas pains* or *colic*. They are intermittent, recurring in a series of waves or paroxysms. Each painful cramp characteristically builds up to a peak of intensity and then subsides. Similarly it is not infrequent for the cramps in a sequence to mount in a crescendo fashion and then to taper off. In the earlier stages of obstruction, the patient does not appear particularly ill in the intervals between these pains, unless, of course, as in some obstructions arising during a postoperative period, the patient is already quite sick at the time obstruction develops.

The earliest paroxysms of abdominal pain are usually followed immediately by vomit-

ing. The first vomiting is neurogenic in nature the stomach emptying itself on reflex stimulation as it does at the onset of many acute abdominal disorders. The vomitus consists of whatever the stomach happens to contain. Following this initial reflex emptying of the stomach there is a variable interval before vomiting resumes in the regurgitant form which is typical of intestinal obstruction. So long as no additional material is taken by mouth, the length of this interval of freedom from vomiting depends largely upon the level of the obstruction. Proximal to the obstructed point, as noted previously, the intestine becomes distended with fluid and gas which it has been unable to drive forward. The powerful peristaltic contractions by which it attempts to force its content past the obstruction soon culminate in periods of reversal of stream which regurgitate this material proximally. In high jejunal obstruction as a consequence, there may be little or no pause in the vomiting since the stomach soon is ejecting the content regurgitated from the uppermost intestine. When the obstruction is low in the ileum in contrast, there may be a considerable interval before reflux reaches the gastric level. As regurgitant vomiting begins, the vomitus is at first bile stained by duodenal content. Then it becomes the thin brownish or greenish content of the distended small intestine. Late in obstruction the material vomited becomes thicker and foul smelling.

Although *failure to pass gas or feces by rectum* soon follows the development of an obstruction, there may be one or more initial movements as the distal bowel empties out its content. In like manner enemas given to encourage evacuation may at the start return fecal matter and gas which had already reached the colon. Once the intestine distal to the obstruction has been emptied, however evacuation comes to an end. Thereafter no gas is passed, provided, of course, that obstruction is complete. This point is of great practical importance in the observation of patients having threatened or suspected obstruction, continued passage of gas by rectum being a reliable indication that any obstruction present is but partial.

These three symptoms *colicky pain, vomiting and failure to pass gas or feces*, are the essential evidences of intestinal obstruction. Some of the additional manifestations are important though less dependable because of their variability. The most constant observation on physical examination is an *increase in peristaltic sounds*. Borborygmi

are heard at the height of the intermittent pains. Occasionally the sounds become so loud as to be audible without the aid of a stethoscope and the patient may even report that he hears the noises himself. The sounds are often of a gurgling or bubbling quality and sometimes the movements of intestinal content can be detected by palpation. As distention increases the sounds take on a higher pitch and assume a metallic or tinkling character. However, since loud peristaltic sounds are often heard in the absence of any distinct pathologic process, the borborygni heard in a patient with suspected obstruction must be interpreted with caution. Probably such sounds are of no particular significance unless they are synchronous with the peaks of the colic. In that case they serve only to establish the presence of hyperperistalsis or colic. It must still be determined whether the colic in the particular instance at hand results from a mechanical obstruction of the intestine or from an acute intestinal irritation such as from gastroenteritis or food indiscretion. In either instance, however, the fact that the abdomen is not silent but that peristaltic activity is readily audible with the stethoscope aids in diagnosis in that it tends to rule out paralytic ileus, an important condition which also is accompanied by distention, vomiting and constipation but in which the abdomen is relatively silent.

Inspection of the abdomen may show only the slight fullness of early distention. Of course, the abdomen will be tight and protuberant if distention has become advanced and there will then be marked tympany on percussion. An external hernia may be visible on inspection, and palpation may prove that it is irreducible, tense and tender so as to suggest immediately a diagnosis of strangulated hernia. In nonstrangulating obstructions, however, abdominal palpation ordinarily gives negative findings. Although the patient may voluntarily draw up his knees and tighten his abdominal muscles during a cramping paroxysm, examination during one of the pain-free intervals usually reveals neither rigidity nor definite tenderness.

It has already been noted that in the earlier stages of simple obstruction the patient does not appear particularly ill, except during the seizures of pain. Marked systemic changes are absent and the laboratory is of little help. The temperature is normal and the pulse rate is not materially increased. There is no significant leukocytosis and the water and electrolyte losses have not yet

been sufficient to be reflected in chemical determinations. Of the various special procedures only the roentgen ray examination of the abdomen is commonly of aid in establishing the diagnosis and in certain instances is of the greatest help. In other cases, however, roentgen ray findings are inconclusive and supply only presumptive evidence. This is particularly true in patients in whom differentiation between mechanical obstruction and paralytic ileus proves especially difficult. In problem cases such as these, the roentgen ray findings must always be correlated with the clinical observations. Again one returns to the conclusion that for intestinal obstruction to be recognized in its early phases when release can be accomplished most efficiently and at the minimum of risk to the patient, the diagnosis usually must rest upon the cardinal symptoms previously mentioned: cramping abdominal pains, vomiting and failure to pass gas or feces.

Late in intestinal obstruction, systemic effects are readily apparent. They consist chiefly of the physical signs of advancing dehydration. Laboratory studies reveal the resulting concentration of the blood. However, the dry skin and tongue, the sunken eyes, the scant and concentrated urine and the hemoconcentration, which the laboratory reports after copious vomiting has been in progress for several days, are in no way specific signs peculiar to intestinal obstruction. They are merely evidences of untreated dehydration, indicating the lack of adequate replacement therapy during a period when there has been excessive fluid loss from any cause.

In some instances the advent of strangulation in small bowel obstruction produces sudden and dramatic developments which are easily recognized. Immediately the abdominal pain becomes more severe and takes on a constant rather than intermittent character. The patient often complains of pain in the back. The general appearance rapidly becomes that of extreme illness. Varying degrees of circulatory collapse or shock develop with lowered blood pressure, accelerated pulse and subnormal temperature. In abrupt and massive strangulations, shock is particularly apt to be immediate and profound. Along with these circulatory changes, there are distinct alterations in the abdominal picture. Localized tenderness develops and the strangulated loop often becomes palpable as a tender indefinite mass. Roentgen ray examinations may,

tended loop to be discrete and to have an unusually smooth-edged appearance, the corrugations caused by the valvulae conniventes having been lost. Leukocytosis and fever develop and there is mounting rigidity to indicate peritonitis.

When the onset of strangulation is heralded by such changes as these the surgical emergency is obvious. Unfortunately however in many other patients, strangulation develops rather insidiously without striking and abrupt changes in the clinical picture and in such patients its early detection may be exceedingly difficult. This fact greatly complicates the frequent problem of proper management of the patient in whom marked distention and advanced water and electrolyte imbalance are already present when he is first seen by the surgeon. In this situation, delay in operative intervention for a day or longer while these complications are righted might well serve the patient's best interest but only provided the surgeon can be certain that strangulation is neither present nor impending. To aid in this dilemma, many studies have been made with the purpose of establishing dependable criteria so that strangulation can always be recognized immediately and accurately. Increased pain and pain of a constant type, increased pulse rate, the presence of a mass tenderness, leukocytosis and beginning abdominal rigidity are signs of strangulation most commonly cited. With such points in mind, some experienced surgeons find that they are able to diagnose strangulation with satisfactory accuracy while others report much less success in this regard. Moreover it should be pointed out that any emphasis placed upon the early signs and symptoms of strangulation must not be interpreted to mean that all is well so long as one proceeds with operation as soon as strangulation is recognized. To the contrary it can scarcely be denied that the surgeon does much better by his patient if the intestinal loop can be released before gross impairment of circulation has occurred, not after. Although the release of a strangulated loop may be followed by changes in the appearance attesting its viability in many instances this is not the case and resection becomes necessary in an extremely ill patient. It is well to emphasize that a number of the so-called signs of strangulation actually are signs of inflammation, that is, they are the signs and symptoms of the peritonitis which is developing as a result of strangulation.

Generalizations regarding diagnosis are

apt to be misleading in colon obstructions because these present a wide variety of clinical pictures ranging from the dramatically sudden onset and rapid course of sigmoid volvulus to the slow insidiously developing obstruction often seen in association with carcinoma of the colon.

Only in the sigmoid, which has a long mesentery is volvulus common. Elsewhere in the colon mobility is so limited by the peritoneal and omental attachments of the bowel that twisting with resulting strangulation is rare. Furthermore, because of the limited mobility colon loops seldom enter hernial orifices (except "sliding" hernias) or pass through windows between peritoneal bands. Instead, in the case of the colon, obstruction usually results from a disease of the bowel itself. Carcinoma, or less commonly chronic inflammatory disease slowly blocks the bowel lumen by stricture formation or by a combination of obturation and stricture. The block becomes complete only after the disease has been present for a considerable time. Clinical obstruction, therefore often develops gradually or with recurring episodes or threats of obstruction. The episodes become progressively more severe. Often the surgeon does not see the patient until he has finally become completely obstructed and is in a distended, cramping state amounting to that of an acute obstruction. If there is a competent ileocecal valve, extreme distention of the colon may develop rapidly with great danger of necrosis and perforation as a result of excessive intraluminal tension. There may be no vomiting whatever. In other adults, and in almost all small children, an incompetent valve permits reflux into the small intestine and the colon is protected to some extent against a sudden and excessive rise in pressure within its lumen. Even in these patients, the vomiting may be greatly delayed and scant. In partial and in intermittent forms of colon obstruction, the small intestine may gradually become greatly distended over a time and patients sometimes present themselves with an enormously distended abdomen which has caused surprisingly little discomfort or vomiting.

In view of the commoner causative lesions, patients with large bowel obstruction are apt to be in the older age group and to show such evidences of an underlying chronic disease as anemia and protein depletion. The patient often reports a change in bowel habit dating back some weeks or months followed by increasing difficulty from con-

stipation or by alternating bouts of constipation and diarrhea. He has cramping pains, often lower in the abdomen than is the case in small bowel obstruction. He is distended, usually markedly so and there is pronounced tympany on percussion. An intestinal pattern, with visible peristalsis is not uncommon. Peristaltic noises are audible and these may be low pitched and rumbling. Vomiting may have been scant or absent and there may be little evidence of dehydration. The roentgen ray "scout" film portrays the distended colon and, as described below fluoroscopy with the barium enema reveals the location of the obstruction.

In comparing the diagnostic criteria with those in obstruction of the small intestine the chief contrasts lie in the frequent absence of vomiting in colon obstruction and in the earlier appearance of meteorism or distention in many patients. In view of these differences the symptom triad given for obstruction of the small intestine might well be altered in the case of large bowel obstruction to read *cramping abdominal pains distention and failure to pass gas or feces by rectum*.

Roentgenographic Studies. Although, in the final analysis the diagnosis of intestinal obstruction must depend upon clinical fea-

tures, radiographic studies of the distribution of gas within the abdomen and of the configuration of the gas-filled intestinal loops are often of great aid in diagnosis. In the normal abdomen, except in infants and very small children, gas is visualized on roentgen ray film only in the stomach, duodenal bulb and colon. Within the small intestine, the bowel activity keeps the gas too intimately mixed with the content for it to be visible. When there is marked stasis for any reason, however the gaseous and liquid elements separate and then the gas collections become visible on the roentgenographic film. In the patient with intestinal obstruction, for example, within six hours after onset roentgen ray studies will usually portray the pockets of separated gas and thus serve to locate the intestinal loops which are proximal to the obstruction. Moreover as the distention of an individual loop progresses, the increased content of gas serves to delineate the intestinal walls bounding the gaseous accumulation and thus provides useful information regarding the contour and configuration of the loop.

A roentgen ray picture, taken with the patient sitting or erect, may show multiple fluid levels of the type characteristic of mechanical obstruction (Fig. 84) In other



Fig. 84



Fig. 85

Figure 84. Typical upright roentgenogram of abdomen in mechanical obstruction of the small bowel. Numerous dilated loops are distended by gas and fluid so that fluid levels are exhibited. There is absence of gas in the large bowel. In one distended loop the circular stripe-markings characteristic of distended jejunum are seen—the herringbone or accordion appearance. (Illustration drawn from an x-ray view)

Figure 85. A ladder arrangement of the shadows of the bowel coils, when the distended loops lie parallel, is pathognomonic of acute obstruction. (Illustration drawn from an upright x-ray view)



Fig. 96



Fig. 97

Figures 96. The gas-filled transverse colon is easily distinguished by its haustral markings. The distended small intestinal loops show fluid levels. (Illustration drawn from an upright x ray view of patient with obstruction of the descending colon.)

Figure 97. The enormously distended sigmoid loop can be recognized, surrounded by the distended loops of the proximal colon. (Illustration drawn from an x ray view of patient having acute sigmoid volvulus.)

cases, the typical stepladder pattern (Fig. 95) may be seen. Survey or scout films taken with the patient in a horizontal position are particularly useful in revealing the location and contour of the dilated loops. A loop of distended colon can often be distinguished by virtue of its haustral markings and its position and course within the abdomen (Fig. 96). Distended loops of small intestine tend to show the distinguishing transverse markings of the valvulae conniventes, an exaggeration of the normal "feathering" often producing the effect known as the accordion or herringbone appearance (Fig. 94). When a distinct loop of small intestine loses these valvular markings and shows smooth rather than corrugated margins, strangulation is to be suspected.

Sometimes the x-ray appearance is so typical that it permits identification of the mechanism causing the particular obstruction. For example, the contour and position of the limbs of the twisted loop of the sigmoid colon are often so characteristic as to permit a diagnosis of volvulus from the roentgen ray view alone (Fig. 97). Other peculiar configurations seen on roentgen ray film aid in the diagnosis of intussusception and of malrotation.

In some instances, the radiographic confirmation and portrayal of an intestinal obstruction are facilitated greatly by concomi-

tant administration of barium as a contrast medium (Fig. 98). When the barium is administered as an enema by rectum, its use is safe provided due care is exercised to avoid overdistention of the rectum by the balloon commonly used and overdistention of the large bowel as a result of administering the enema under excessive pressure. Such precautions are necessary lest perforation be



Figure 98. Appearance with barium enema in a patient with carcinoma of the splenic flexure of the colon. The column of barium often stops completely when the tumor is first encountered but passes through the narrowed area shortly thereafter to delineate the defect. (Illustration drawn from an x-ray view.)

caused in patients having colon obstruction either as a direct effect, the bowel perforating at a diseased area which is subjected to the immediate pressure of the enema or in directly owing to an excessively violent reflex contraction of the proximal colon having been elicited by distention of the rectum. In the latter instance the thin walled cecum proximal to an obstruction may "rupture" itself if the ileocecal valve is competent. However when performed in a safe manner the barium enema is so helpful in demonstrating the presence and location of a large bowel obstruction that it is considered an essential part of the preoperative study in colon obstructions. Without the information it provides the surgeon often would be unable to plan the operation and the surgical approach intelligently.

Aside from its negative value in demonstrating that the colon is not obstructed, the barium enema is of little aid in the study of obstructions of the small intestine excepting those involving the immediate vicinity of the ileocecal valve such as terminal ileitis and ileocolic intussusception. This has given rise to the temptation to administer the barium by mouth in obstruction or suspected obstruction. However when barium is introduced proximal to an obstruction it has a notorious tendency to form an impaction at and above the point of obstruction where it frequently becomes inspissated to form a hard mass or enterolith. The impaction immediately converts a partial obstruction into a complete one and the barium mass greatly complicates the surgical treatment. With few exceptions consequently the oral administration of barium in patients with possible intestinal obstruction is to be condemned. Such use has led to many deaths in persons having colon obstruction through operative and postoperative complications for which the barium is responsible. Moreover in obstruction at more proximal levels, oral administration of barium is usually unnecessary and the information to be gained by its use is seldom worth the risk involved. In the highest obstructions, pyloric and possibly duodenal, the cautious use of small quantities of a thin barium mixture may sometimes be permissible if one is sure he can recover the barium in washings. Even here however one must weigh the advantages against several risks. The latter include the risk of aspiration of barium into the lung, a particular danger in the infants in whom these very high obstructions are most common.

Differential Diagnosis. The conclusion that

obstruction is present in a given patient immediately poses several supplemental problems in diagnosis. Information bearing upon the degree, the approximate site and the probable cause of an obstruction provides a basis for intelligent estimates relating to the urgency of the situation, the course to be expected and the likelihood of early strangulation.

So long as the patient continues to pass gas by rectum the obstruction may be considered to be only threatened or partial. Similarly roentgen ray demonstration that gas reappears in the colon after it has been evacuated by enemas indicates that the obstruction is still incomplete. Since complete occlusion of the intestinal lumen nearly always precedes the advent of strangulation, continued passage of gas leads to the conclusion that time is available for additional study as well as for attempts to relieve the obstruction by nonoperative means.

The approximate site or level of an obstruction can often be conjectured on the basis of the history and symptoms. More over an obstruction of the colon can ordinarily be located accurately by means of the fluoroscope and barium enema, whereas the x ray scout film often serves to locate a small bowel obstruction within the limits of the upper middle or lower third of the small intestine. Even this approximation as to location permits a more intelligent selection of incision.

In some patients the specific cause of an obstruction is obvious on physical inspection. This is the case in strangulated external hernia or in imperforate anus. Other patients in whom the physical signs permit a strong inference as to cause include those with laparotomy scars to suggest obstruction by adhesions, or with a palpable tumor which is characteristic, as in intussusception. Again the x ray film of the abdomen may reveal findings typical of such lesions as congenital atresia or meconium ileus, or the appearance following a barium enema may justify a specific diagnosis such as volvulus, intussusception or diverticulitis. In a great many other patients, however little or no preoperative evidence will be obtained from any source to indicate the mechanism or cause of the obstruction. This is particularly apt to be the case when the interference is in the small intestine.

Obstruction must be distinguished from many other disorders which cause abdominal

The Richter type of femoral hernia is one of the few exceptions to this rule.

pain and vomiting. If the pain is of a cramping or colicky nature and is associated with inability to pass gas, there must immediately be suspicion of obstruction. The absence of the constitutional and abdominal signs of acute inflammation permits differentiation from such diseases as acute appendicitis, acute cholecystitis and peritonitis. In simple obstruction there is neither fever nor leukocytosis. Similarly there is no definite tenderness,* either localized or general and abdominal rigidity is absent. The sounds of increased peristaltic activity which are typical of mechanical obstruction moreover are rarely heard in association with the abdominal inflammations. Inflammation is usually accompanied instead by a reflex inhibition of intestinal motility or ileus.

Diagnosis becomes more difficult if strangulation has ensued since the signs of a local inflammatory lesion then begin to make their appearance and to become superimposed upon the symptoms of simple obstruction. Differentiation must be based upon the history and upon the other signs and symptoms of intestinal obstruction. Furthermore when a sudden strangulation leads to hemorrhage or to shock, it may be confused with other conditions responsible for abdominal hemorrhages and with conditions such as perforated peptic ulcer which produce a temporary shock state on a neurogenic basis.

A particular problem in diagnosis arises in differentiating intestinal obstruction from other abdominal colics. Renal or ureteral colic and biliary colic are usually recognized easily because of the characteristic location and radiation of the pain. Such syndromes as lead colic and the abdominal crises of tabes dorsalis, sickle cell anemia and porphyria are rare but must be kept in mind along with the visceral neuropathy associated with diabetes. The more frequent difficulty concerns differentiation of mechanical obstruction from the intestinal colic of acute enteritis or gastroenteritis resulting from poisoning, infection or dietary indiscretion. The history is of great help in recognizing enteritis of this sort in that it may reveal ingestion of a possible poison or of an injudicious combination of foods. A story of previous food allergy also may provide a clue. In food poisonings, moreover other associates or members of the household usually have been similarly and simultaneously stricken. In any case, the advent of diarrhea will re-

*Point tenderness at the site of attachment of an adhesion to the peritoneal parietum is an exception occasionally noted.

lieve all suspicion that the cramping pain is the result of mechanical obstruction. A delay in the onset of diarrhea, however might lead to premature laparotomy on the assumption that an obstruction is present.

The differentiation of mechanical intestinal obstruction from neurogenic obstruction, or *paralytic ileus* may be a very difficult diagnosis especially during the days immediately following an abdominal operation. Since a temporary inhibition of intestinal motility occurs regularly after major abdominal surgery and is aggravated and prolonged when there is any significant degree of peritonitis, a possible basis for protracted ileus is present after many operations. Inasmuch as mechanical obstruction also may develop during the postoperative period, one is concerned in differentiating the two conditions whenever distention with obstipation and vomiting appears after abdominal surgery. The most important point enabling such differentiation is the fact that in paralytic ileus the waves of forceful and violent peristalsis characteristic of mechanical obstruction are absent. Consequently the patient does not complain of cramping or colicky pain and on auscultation there is no increase in the sounds of peristaltic activity. Instead the abdomen is relatively silent. X ray scout films, moreover show that gaseous distention is generalized rather than limited to the intestinal loops proximal to any level. Perhaps most important of all is the x ray visualization of gas in the rectum and pelvic colon. In fact, in many cases of ileus, distention of the entire large bowel is a prominent feature. In advanced peritonitis, for example the colon loops may be enormously distended.

As the intestine recovers from a period of reflex inhibition or ileus, there may be transient "gas pains" particularly if the bowel has been distended. The irregularity of these cramps, and the fact that gas is soon passed per rectum ordinarily prevent confusion with the colic of a mechanical obstruction. Occasionally however the patient actually does enter a state of mechanical obstruction as ileus is subsiding. After peritonitis for example obstruction from adhesions is not infrequent and at a certain period the patient may be suffering from a "combined obstruction," both paralytic and mechanical.

Treatment. During the past twenty-five years there has been a remarkable reduction in the over all mortality rate in acute intestinal obstruction. A decrease from a former rate of over 40 per cent to a current rate of

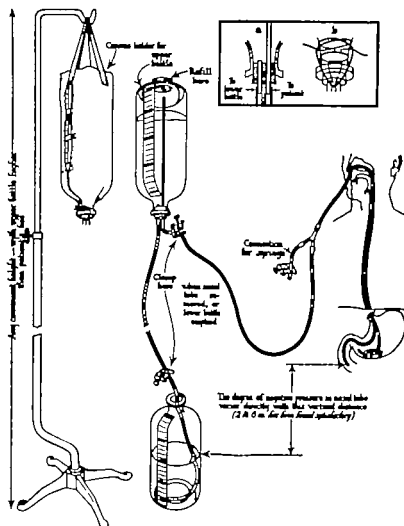


Figure 99 Diagram of suction apparatus employed in the treatment of intestinal distention. An effort should always be made to have the end of the catheter reach beyond the duodenum. Perforations are made so that suction may be exerted simultaneously on the stomach and on the duodenum. When the fluid aspirations are great, a third bottle should be used as a collecting receptacle. (Wangenstein and Faine J.A.M.A. vol. 110.)

perhaps 10 per cent has been cited recently. Although the discovery of antibiotics, the development of improved techniques in operative surgery and improvements of anesthesia have played parts in this reduction certainly the chief factors responsible have been the use of decompressing devices for prevention and correction of intestinal distention and the more effective replacement therapy made possible by constantly increasing knowledge of fluid and electrolyte balance. Since in the vast majority of patients the obstructing mechanism can be released only by operation, treatment of intestinal obstruction remains primarily surgical. However intestinal decompression and parenteral replacement therapy are vital

supportive measures often essential both to preparation for operation and to the after care.

Decompression of the small intestine is accomplished by continuous aspiration of content by way of a tube passed through the nose into the stomach or upper intestine. The method was introduced by Wangenstein (1931-32) using a tube which passed through the stomach into the duodenum whenever possible, and applying constant gentle suction through the tube by means of a simple water siphon apparatus such as that shown in Figure 99. Following demonstration of the value of this method, Miller and Abbott (1934) introduced a longer tube for decompression, the tube having a double lumen and carrying an inflatable balloon in its tip. Once the tip of the Miller Abbott tube has passed from the stomach into the

The fact that the mortality rate is still as high as 10 per cent is evidence of the critical dangers inherent in intestinal obstruction.

duodenum, the balloon is inflated to serve as a bolus which peristalsis propels distally along the intestine. If the intubation is ideally successful, the tip of the tube eventually comes to lie just proximal to the point of obstruction so as to effect complete emptying of the proximal intestinal loops. Passage of the tube through the pylorus continues to be a time-consuming and trying problem in intestinal intubation.

In discussing decompression it should be emphasized that prevention of distention is not nearly so difficult as is the relief of distention once it has become established. In patients in whom distention has not yet occurred, the institution of simple gastric suction by way of a Levin tube which stops short of the pylorus will serve to recover all air swallowed thereafter as well as any liquid content which is regurgitated from the duodenum, provided, of course, that the tube is kept open so that it can function. This suffices to prevent the development of distention. Once distention is present, however gastric suction, since it fails to empty the tract distal to the pylorus, serves only to prevent augmentation of the existing distention. If the Levin tube can be passed through the pylorus, decompression from within the duodenum is somewhat more efficient. However for satisfactory removal of the voluminous content from loops which are already distended, intubation to a point beyond the duodenojejunal flexure, where sharp angulation may be caused by the ligament of Treitz, is most effective. This requires the use of the long tubes such as the Miller Abbott tube.

After twenty four hours, one may find that only gastric intubation has been accomplished. This difficulty remains the chief hindrance to effective decompression therapy while the delay incident to unsuccessful attempts to intubate the intestine may be a very dangerous delay when treating patients having intestinal obstruction. Whereas, laparotomy admittedly is more hazardous when performed in the presence of marked distention, that danger to the patient is not so great as is the danger arising from delay when strangulation is present or imminent.

Although intestinal decompression is primarily a method for combating distention, the history of its development has been colored by constant attempts to substitute decompression for surgical operation in the definitive treatment of obstruction. These attempts are easily understood in the light

of the clinical experience that, in a number of patients having early or incipient obstruction of certain types in the small intestine decompression of itself actually does serve to release the obstructing mechanism so that an emergency surgical operation becomes unnecessary. Very naturally experience with such cases has led to trials in substituting decompression for operation in treating other patients with obstruction and many studies have been made to determine the possibilities and define the limitations of such non-operative management. Unfortunately however too little heed is given to repeated warnings pointing out the great difficulty in selecting patients in whom it is safe to defer operation while a satisfactory trial of decompression therapy is being made. Lack of appreciation of this difficulty continues to lead to costly misuses of decompression as a tentative substitute for surgery. Unless the surgeon is highly critical, a false sense of security is fostered by the cessation of pain and vomiting which follows institution of decompression and by the temporary improvement in the patient's condition which is brought about by the concurrent administration of water and electrolytes. Often these beneficial effects are interpreted as indicating a relief of the obstruction when in reality they mean only a relief of certain of its symptoms. In many instances consequently surgery is postponed unduly until a subsequent deterioration in the patient's condition finally forces operation upon the surgeon at a time when greater hazards are faced than would have been the case with prompt surgery. In these patients it is not unusual for the tardy laparotomy to reveal that an unrecognized strangulation has been present and that the delay which had been intended for the patient's benefit has actually been to his great harm. Thus, it is the unhappy paradox that intestinal decompression, a valuable form of supportive therapy through misuse becomes responsible for many of the deaths which continue to occur.

Decompression of the large intestine can only occasionally be accomplished by means of a tube passed through the nose into the small intestine. As previously noted, the ileocolic sphincter often serves as an effective one-way valve to prevent all regurgitation from the cecum into the ileum. Consequently acute distention of the colon demands immediate surgical decompression by open operation. However the use of nasogastric suction as an adjunct measure still

serves a useful purpose, since by recovering swallowed air it prevents additional distention of the small intestine.

In all intestinal obstructions, fluid is lost from the normal body fluid spaces whether it be by vomiting, by collection of fluid in the lumen of the obstructed bowel or by retention in edematous tissues. The prevention of water and electrolyte imbalances and the correction of depletions already existing are of immediate concern in the treatment of each patient. The extent of replacement needed and the time available for its achievement are influenced necessarily by varying factors. The level of obstruction influences replacement requirements. In obstructions located high in the small intestine losses by vomiting usually have been copious whereas in colon obstructions there may have been no vomiting at all. The elapsed time since onset of obstruction is another important consideration. When obstructions are seen very early replacement needs are correspondingly less than at later stages. The possible presence of strangulation is particularly important. It sharply limits the delay which is justified in order to effect replacement.

In treating patients having obstruction of the small intestine the basic problem is that of correcting the dehydration which has resulted from lack of intake coupled with a loss of intestinal secretions. I.e. a dehydration comprising deficits both of water and of salt. A quick examination of the urine with regard to color volume and specific gravity provides a rough measure of the extent of the dehydration. Correction is accomplished by intravenous administration of an adequate quantity of water and of sodium chloride. In patients with early cases in whom only moderate losses have taken place the administration of 1 or 2 liters of isotonic saline often suffices and this can be accomplished during the preparation for surgery and while the operation is in progress. In patients with late cases however several times this volume may be needed and the administration of such quantity ordinarily requires some hours. Glucose is added to reduce the caloric deficit. Appearance of dilute urine which contains chloride or ordinarily indicates that water balance has been restored. In the meanwhile, determination of the carbon dioxide combining power of the blood will have revealed the extent of acidosis or alkalosis. Potassium replacement, if needed, is deferred until the output

of urine is good and, in emergency cases, can often be postponed until the postoperative period.

Vitamins B and C are added to intravenous infusions as needed. When history or examination suggests malnutrition, attention is directed particularly to the treatment of avitaminosis.

During recent years there has been better appreciation of the need for adequate blood transfusions in the preoperative preparation of many patients having small bowel obstruction. Loss of whole blood into the lumen of strangulated intestine or loss of plasma through the wall of edematous bowel calls for replacement in patients in whom it appears likely that such loss has been substantial. Moreover transfusion of whole blood or plasma does much to sustain those patients in whom preparation for operation has been hurried of necessity because of obvious or suspected strangulation.

Large bowel obstruction usually presents quite different problems in the correction of physiologic deficits than does obstruction of the small intestine. As noted before, vomiting is not a prominent feature in colon obstruction therefore losses of water and electrolytes usually are not acute. In view of the commoner causes of obstruction of the large bowel, the typical patient is older and suffers from a chronic illness. For these reasons the surgeon is chiefly concerned with replacing blood, protein and vitamins rather than with administering water and salt. When the patient is seen before complete obstruction has become established, time may be available for reasonably adequate correction of the malnutrition and anemia, operation being deferred to a later time. This also permits the preparation of the colon with antibiotics. If the patient is not seen until acute obstruction is present, however time is not available for complete replacement therapy or for full antibiotic protection since necrosis and perforation may result from any considerable delay in preparing for an emergency operation. In such cases a chief concern lies in assuring an adequate output of urine. Furthermore, in lieu of time for better preparation blood transfusion is used freely to sustain the patient during surgery. Of great importance, moreover is the practice of limiting the surgical operation to simple palliative decompression of the distended colon. Fortunately a great majority of large bowel obstructions are of a nature to permit post

ponement of definitive treatment to a later date.

Digital examination of the rectum is an essential diagnostic step in all patients with suspected intestinal obstruction. Fecal impaction, rectal stricture and pelvic tumor are examples of conditions sometimes discovered which alter the entire plan of treatment.

The majority of patients having obstruction will have been given one or more enemas in the process of establishing a diagnosis. Any continued evacuation of gas and liquid means that obstruction is not complete. The use of the enema is not entirely devoid of danger however and at times it is overdone. After the colon has once been evacuated, the use of a rectal tube or a colon tube is often preferable to more enemas. Continued passage of gas or liquid through the tube indicates progress in relieving the obstruction. The colon tube is the more effective, but to introduce a tube beyond the rectum into the sigmoid it is usually necessary to guide it visually by proctoscope.

Aside from their general value as an adjunct in surgery antibiotic substances are particularly valuable in the treatment of strangulating obstructions since they help to delay the onset of necrosis as well as to combat its ill effects. For parenteral administration, a combination of penicillin and streptomycin with one of the broad-spectrum antibiotics such as Aureomycin is used frequently. A very promising development is seen in recent reports, both experimental and clinical, indicating that intestinal antiseptics are effective when introduced directly into the bowel during an emergency operation. For example, it is reported that when time has not been available for preoperative preparation of the bowel, neomycin is of value if used in this manner.

Not infrequently in certain types of obstruction complete relief will be effected, at least for the time being, during the process of preparing the patient for operation. Obviously an emergency operation becomes unnecessary if normal passage of gas by rectum is restored as a result of instituting upper intestinal decompression or as sometimes happens, during the course of evacuating the colon by enemas. However the patient should be observed for any return of symptoms and diagnostic studies planned to determine the cause of the obstructive threat. Often, an underlying lesion will be discovered which necessitates operation at a subsequent date.

When confronted with a case of complete obstruction the surgeon must first arrive at a conclusion relative to the likelihood of strangulation existing. If he concludes that strangulation is present or imminent, operative treatment is mandatory and he must proceed with a minimum of delay. In most situations an hour or longer will be required to prepare the operating room and to induce anesthesia. During this time isotonic saline solution is given intravenously while emergency laboratory determinations are made, antibiotics administered and blood transfusion started. Supportive treatment is continued during and after operation. In the presence of recognized strangulation greater delay is seldom justified. Within this short period, the correction of water and electrolyte depletion can hardly be complete and often will be very inadequate. Liberal use of blood transfusion helps to sustain the patient in the emergency.

Immediate surgery is also required when acute colon obstruction is accompanied by marked distention. Otherwise, since many colon obstructions are of the closed loop type delay is apt to result in necrosis and perforation. Tube-decompression of the small intestine must never be expected to relieve the greatly increased tension within a distended colon.

When, in the surgeon's opinion, the obstruction is of a simple or nonstrangulating variety he must remain alert to the possibility that it may change in character and frequent reassessment with respect to the possible presence of strangulation is essential. Furthermore, since it is notoriously difficult in some cases to recognize the advent of strangulation, he will be wise to avoid procrastination by setting in his own mind definite time limits for the preparation of the patient. This is particularly important when, in patients with distention decompression therapy is being given a trial, for it will mask many of the symptoms of obstruction. Some advise immediate operation if the obstruction has not been relieved within a six hour period, since that length of time will ordinarily suffice for minimal parenteral replacement.

Patients having early simple obstruction require the least in replacement therapy. Since distention is not yet severe they are ideal for immediate operative relief and, under modern conditions the surgical risk is not great. For these reasons, there is a good basis for recommending prompt surgery in all patients with early obstructions.

Although this will mean that a few patients will be operated upon unnecessarily the risk in these few will be slight indeed. In the other patients, the danger of overlooking occult strangulation will be avoided. The over-all risk will be less than when such patients are treated first by a lengthy trial of decompression in the hope of releasing the obstruction without operation.

Early postoperative obstructions must be considered separately since in such patients it may be impossible to observe the time limitations which are recommended for obstructions in general. After major abdominal surgery a period of reflex inhibition of intestinal motility may be expected for a day or two. If at the end of this period, symptoms arise which suggest mechanical obstruction, considerable additional time may be required to determine whether obstruction actually exists. Instead, the patient may be suffering from a prolongation of the paralytic ileus or may only be experiencing gas pains coincident with recovery of the intestine from ileus. A period of twenty four hours or more may be needed to arrive at a diagnosis, whereas developments during the subsequent hours may serve to reverse that decision. Inasmuch as inflammation and edema commonly play an important role in the early postoperative period, a number of these postoperative obstructions are relieved completely by decompression therapy. This fact and the widespread impression that there is very little risk of strangulation have resulted in overconfidence in dealing with such cases. However an early postoperative obstruction is not necessarily due to fresh adhesions; it may be the result of old adhesions, of volvulus or of hernia through a postoperative defect. Strangulation does result in 10 per cent or more of early postoperative obstructions. Therefore, once obstruction is diagnosed in these patients, surgical exploration is in order unless relief by intubation is prompt and unequivocal.

Operative treatment of mechanical obstruction is aimed primarily at restoring the physiologic continuity of the bowel by releasing the intestine from the interference which is occluding its lumen. Since any one of many and varied mechanisms may produce obstruction, the surgical steps required to relieve the situation obviously will vary greatly from patient to patient. The surgeon is concerned also with preventing a recurrence of obstruction, and treatment includes when practicable, the definitive care of any causative lesion which is found.

In some cases effective operative treatment is relatively simple. In patients with early obstruction by an adhesion band, for example there may be little difficulty in locating the junction of the collapsed distal intestine and the dilated proximal loops and in severing the adhesion which is causing the constriction deformity or the angulation at that level. Sometimes there are multiple obstructive sites where release must be afforded. Again, attachments which are not yet causing obstruction may also need liberation on the basis that they provide potential sites for future obstruction. Throughout the operation, all surgical manipulation must be gentle in order to avoid creating additional raw areas and thus encouraging formation of more adhesions. Moreover any areas which do become freshly denuded are recovered with peritoneum if possible, and the remnants of adhesive bands are not left dangling to invite reattachment. When the operation is performed carefully most patients having obstruction by adhesions can be relieved permanently and future difficulties avoided. Similarly relief is not particularly difficult in patients with early obstructions arising from many other causes such as volvulus intussusception and strangulated hernia.

Marked distention always makes surgery difficult technically and this results in greater trauma and hazard to the patient. When it has been impossible or impracticable to obtain relief by preoperative intestinal intubation, the use of nasogastric suction is important in order to prevent any increase in the distention already present. When surgery must be performed in the presence of severe distention, moreover many surgeons have adopted the practice of deflating the distended intestinal loops by *direct suction aspiration* before closing the abdomen. Various trocars have been designed for introduction through the bowel wall to accomplish this purpose. Due care is taken to prevent spill and contamination of the peritoneum. Such direct evacuation of the distended intestine at laparotomy sometimes makes it possible to locate and release obstructions which otherwise would be extremely difficult of access. In addition, the patient is left in a condition more favorable to recovery than if the abdominal wall were closed over greatly distended bowel. Enterostomy a procedure which consists in introducing a catheter into a distended loop of intestine—the loop immediately proximal to the obstruction if possible—the

catheter out through the abdominal wall to drain the intestinal lumen, often is disappointing by failing to relieve the distention.

When the obstructing mechanism has also interfered with the circulation to the obstructed loop so that *strangulation* exists surgical treatment may be greatly complicated. In patients with early cases of strangulating obstruction, the release of the obstructed intestine and its mesentery by unwinding the volvulus, severing the constricting ring at the neck of the hernia or reducing the intussusception may lead to immediate return of a good circulation. Although the affected loop frequently retains the evidence of its recent venous engorgement and appears "angry" for a time when it resumes its normal mobile position in the peritoneal cavity and is covered with warm, moist gauze, a few minutes often suffice to demonstrate its viability. The loop assumes a reddish hue, pulsations are palpable at the mesenteric margin and gentle mechanical stimulation elicits contractions which traverse the segment in question. These are the ordinary signs of adequate circulation. In more advanced instances of strangulation the intestinal loop may be frankly gangrenous or may have a borderline appearance which makes the decision as to viability very difficult. Again, sometimes the loop as a whole is clearly viable, but there are dark, lusterless spots which do not share in the general satisfactory appearance. Various special tests have been devised to determine the adequacy of the circulation in doubtful instances, but the safest rule for the surgeon to follow is to resect the intestinal loop when he has any doubt regarding its viability. After resection the continuity of the bowel is ordinarily restored by end-to-end anastomosis of the remaining intestinal segments. Occasionally in critical operative situations, the gangrenous intestinal loop is exteriorized by bringing it outside the abdomen and closing the abdominal wall beneath it. This shortens the operation since the necrotic segment of intestine can then be excised without further anesthesia and the remaining ends of intestine left protruding above the abdominal wall. Since external discharge of the content of the large bowel creates no insuperable problem, this form of management is entirely feasible when a segment of gangrenous colon has been excised, and it is practiced sometimes in treating late sigmoid volvulus. If the

gangrenous loop is one of small intestine, however such *exteriorization* is reserved for desperate situations because the copious discharge of small bowel content not only excoriates the skin but also is apt to create a critical problem in water and electrolyte replacement.

In obstructions of the small intestine it is usually practical and desirable to combine at one sitting the release of the obstruction and the definitive treatment of the causative lesion. While this is sometimes necessary in large bowel obstruction also as has just been mentioned in connection with sigmoid volvulus, in the usual case of *colon obstruction* the reverse is true. Experience has demonstrated that when large bowel obstruction is the result of a chronic disease, such as carcinoma or diverticulitis, the ill effects of the obstructive episode are superimposed upon the debilitation resulting from the causative lesion to the extent that surgical interference is not well tolerated and "one stage" or "primary" colon resection becomes unduly dangerous. Although these obstructions are not strangulating obstructions in the ordinary sense they do present an emergency situation since the rapidly mounting intraluminal tension may result in necrosis and perforation of the distended large bowel. Intubation of the small intestine, as previously stated, cannot be expected to relieve colonic distention and by far the safest procedure for these patients is immediate surgery in the form of palliative decompression of the distended colon. This consists of forming an artificial vent in the large bowel at a point proximal to the obstructing lesion. When the obstruction is in the left colon, which is the common site, a loop of transverse colon is brought out upon the abdominal wall and a transverse colostomy formed. Cecostomy is necessary if the obstruction is situated proximal to the transverse colon, e.g., at the hepatic flexure. Either procedure can be completed in a manner to provide immediate decompression of the distended colon. The emergency situation having been relieved in this fashion, time becomes available for adequate restoration of the patient by replacement therapy and for preparation of the intestine with antibiotics. The definitive treatment of the causative lesion can then be conducted at a later date. Such a "two-stage" operation, in which resection of the diseased bowel is attempted only after a period of decompression by proximal colostomy is much the safest form

Although this will mean that a few patients will be operated upon unnecessarily the risk in these few will be slight indeed. In the other patients, the danger of overlooking occult strangulation will be avoided. The over all risk will be less than when such patients are treated first by a lengthy trial of decompression in the hope of releasing the obstruction without operation.

Early postoperative obstructions must be considered separately since in such patients it may be impossible to observe the time limitations which are recommended for obstructions in general. After major abdominal surgery a period of reflex inhibition of intestinal motility may be expected for a day or two. If at the end of this period, symptoms arise which suggest mechanical obstruction considerable additional time may be required to determine whether obstruction actually exists. Instead, the patient may be suffering from a prolongation of the paralytic ileus or may only be experiencing gas pains coincident with recovery of the intestine from ileus. A period of twenty four hours or more may be needed to arrive at a diagnosis whereas developments during the subsequent hours may serve to reverse that decision. Inasmuch as inflammation and edema commonly play an important role in the early postoperative period, a number of these postoperative obstructions are relieved completely by decompression therapy. This fact and the widespread impression that there is very little risk of strangulation have resulted in overconfidence in dealing with such cases. However an early postoperative obstruction is not necessarily due to fresh adhesions it may be the result of old adhesions, of volvulus or of hernia through a postoperative defect. Strangulation does result in 10 per cent or more of early postoperative obstructions. Therefore, once obstruction is diagnosed in these patients, surgical exploration is in order unless relief by intubation is prompt and unequivocal.

Operative treatment of mechanical obstruction is aimed primarily at restoring the physiologic continuity of the bowel by releasing the intestine from the interference which is occluding its lumen. Since any one of many and varied mechanisms may produce obstruction, the surgical steps required to relieve the situation obviously will vary greatly from patient to patient. The surgeon is concerned also with preventing a recurrence of obstruction, and treatment includes when practicable, the definitive care of any causative lesion which is found.

In some cases effective operative treatment is relatively simple. In patients with early obstruction by an adhesion band, for example, there may be little difficulty in locating the junction of the collapsed distal intestine and the dilated proximal loops and in severing the adhesion which is causing the constriction deformity or the angulation at that level. Sometimes there are multiple obstructive sites where release must be afforded. Again, attachments which are not yet causing obstruction may also need liberation on the basis that they provide potential sites for future obstruction. Throughout the operation, all surgical manipulation must be gentle in order to avoid creating additional raw areas and thus encouraging formation of more adhesions. Moreover any areas which do become freshly denuded are recovered with peritoneum if possible, and the remnants of adhesive bands are not left dangling to invite reattachment. When the operation is performed carefully most patients having obstruction by adhesions can be relieved permanently and future difficulties avoided. Similarly relief is not particularly difficult in patients with early obstructions arising from many other causes such as volvulus, intussusception and strangulated hernia.

Marked distention always makes surgery difficult technically and this results in greater trauma and hazard to the patient. When it has been impossible or impracticable to obtain relief by preoperative intestinal intubation, the use of nasogastric suction is important in order to prevent any increase in the distention already present. When surgery must be performed in the presence of severe distention, moreover many surgeons have adopted the practice of deflating the distended intestinal loops by direct suction aspiration before closing the abdomen. Various trocars have been designed for introduction through the bowel wall to accomplish this purpose. Due care is taken to prevent spill and contamination of the peritoneum. Such direct evacuation of the distended intestine at laparotomy sometimes makes it possible to locate and release obstructions which otherwise would be extremely difficult of access. In addition, the patient is left in a condition more favorable to recovery than if the abdominal wall were closed over greatly distended bowel. Enterostomy a procedure which consists in introducing a catheter into a distended loop of intestine—the loop immediately proximal to the obstruction if possible—and the

portance from the standpoint of the abdominal surgeon are those cases of ileus resulting from varying degrees of irritation and inflammation of the peritoneum and those cases arising from other lesions which give a clinical picture readily confused with peritonitis. Among the latter group the injuries resulting in fractures about the ribs and spine or in retroperitoneal hemorrhage should have special mention. By irritation of spinal nerves or nerve roots such lesions very commonly give rise to abdominal pain and muscle spasm as well as to ileus and vomiting, so that the clinical picture may be particularly difficult to differentiate from that of peritonitis resulting from a ruptured viscus.

A temporary state of ileus is to be expected after abdominal operations as a result of the exposure of peritoneal surfaces and the manipulation of the viscera. This is the commonest form of traumatic peritonitis. When the operation is such that one anticipates an ileus persisting more than twenty-four hours, continuous gastric suction by way of a Levin tube introduced at time of operation is of great value. It prevents significant distention from air swallowing and minimizes postoperative vomiting. The tube is removed, of course, when the return of effective peristalsis is indicated by resumption of normal bowel sounds and passage of gas by rectum. Since it is much easier to prevent distention in this manner than it is to relieve distention which already has become established, such prophylactic gastric suction proves particularly valuable if peritonitis or obstruction arises later in the postoperative period. In fact, the absence of distention constitutes a distinct advantage in the management of almost every postoperative complication.

The severe form of paralytic ileus which accompanies bacterial peritonitis is illustrated by the sequence of events in neglected appendicitis. In the early stage of appendicitis when inflammation is limited to the appendix, the patient is usually constipated and may complain of inability to pass gas. As inflammation spreads onto the serosal surface of the appendix and then to the adjacent parietal peritoneum, "peritoneal irritation" usually results in a "quiet" abdomen with pronounced obstipation. An exception may be seen, however, in patients in whom the appendix lies adjacent to the rectum, where contact irritation may give rise to rectal tenesmus or diarrhea. If localization fails, so that there is diffuse and spreading

peritonitis complete ileus becomes established and is unrelenting for a time. In general peritonitis following appendiceal rupture for example the loss of bowel motility can be expected to persist unabated for four or five days. By the end of the second or third day the distention has become marked unless there has been effective gastric suction. The added complication of acute gastric dilatation was once very common now it is rarely seen in patients under treatment on surgical wards where some form of decompression is used routinely. Resumption of intestinal peristalsis at the end of the five-day period indicates usually that the patient has weathered the critical infection and probably will recover although complications such as localized abscess or mechanical obstruction from adhesions, may still interrupt convalescence.

The diagnosis of paralytic, or inhibition, ileus is based upon the lack of bowel sounds and the absence of cramping pains in a patient suffering from distention, vomiting and obstipation. When the situation is one of ordinary postoperative ileus, the condition is only temporary and mild cramps concurrent with the return of peristalsis soon are followed by the passage of gas. Occasionally such "gas pains" are severe, particularly in patients who have been permitted to distend themselves with swallowed air during the postoperative period, and in these patients the differentiation from mechanical obstruction may be more difficult. The roentgen ray film however shows that the gas has a general distribution and is visualized in the large as well as the small intestine.

The appearance of pronounced ileus always suggests the possibility that peritonitis is present. If peritonitis actually exists, one finds abdominal tenderness and rigidity either localized or general depending upon the extent of the inflammation. Pain is present but is constant rather than intermittent or colicky. Fever, tachycardia and prostration are constitutional signs of infection, along with leukocytosis. When ileus results from an extra-abdominal infection such as pneumonia, these same systemic signs appear but abdominal tenderness and muscle spasm are lacking. Not infrequently in obese and older individuals, however, the abdominal tenderness of appendicitis may be difficult to detect for a day or longer and the development of silent distention of the abdomen is always an indication for repeated abdominal examinations.

Treatment of paralytic, or inhibition, ileus

of management when carcinoma of the colon or sigmoid diverticulitis has resulted in intestinal obstruction.

NEUROGENIC OBSTRUCTION OR ILEUS

Intestinal obstruction may be the result of nervous interference with the normal propulsion of intestinal content, just as it may arise from mechanical obstruction of the bowel lumen. Normal passage down the intestinal tract is accomplished through physiologically coordinated waves of muscular contraction. These peristaltic waves are dependent upon the intrinsic nerve plexuses in the intestinal wall and neither vagotomy nor sympathectomy abolishes them. However, though peristalsis originates through these local bowel reflexes it is readily influenced by the extrinsic nerves. The vagus whose terminals connect with ganglion cells in Auerbach's plexus serves in general to augment peristaltic movements and to increase the tone of the smooth muscle of the intestine. In man, however the vagus is not constantly or tonically active in augmenting intestinal motility and interruption of the vagi, surprisingly is less apt to lead to intestinal stasis than to a temporary state of hypermotility and diarrhea. Moreover the participation of the vagal innervation in pathologic disturbances of peristaltic co-ordination and in production of spasm rings and other impediments to intestinal passage remains obscure and uncertain.

The inhibitory effect of the sympathetic innervation, in contrast, is evidenced clearly in various pathologic states. The sympathetic fibers to the intestine pass peripherally mainly in the lesser splanchnic nerves. They terminate in the lower part of the celiac ganglion and in the superior mesenteric ganglion. From these ganglia, inhibitory impulses are relayed to the bowel through postganglionic neurons which pass uninterrupted through the intrinsic plexuses of the bowel wall to end in direct relation to the smooth muscle cells. That there is a certain amount of tonic inhibitory activity by way of these fibers is witnessed by the observation that spinal anesthesia, which interrupts conduction in the thoracolumbar outflow of the sympathetic, usually serves to augment intestinal peristalsis. In many disease situations, moreover reflex sympathetic inhibition of intestinal motility is sufficient to produce a state of pronounced stasis leading to distention and vomiting. The effects of such reflex ileus may be fully as serious as are the effects of the comparable stasis

and distention arising from actual mechanical obstruction.

Paralytic ileus is the term commonly used in referring to the impairment of intestinal peristalsis which is seen, for example, when the peritoneum is the seat of an inflammatory reaction. A similar derangement of intestinal motor function may accompany traumatic or infectious lesions which are extraperitoneal or even extra abdominal. In addition, ileus sometimes appears in conjunction with a generalized toxic state such as uremia or it may be seen in connection with severe electrolyte imbalance, particularly when potassium is deficient. In the case of uremia and of hypopotassemia, possibly the intestinal musculature actually is parietic in the sense that its intrinsic ability to contract has been reduced temporarily. Further more in the advanced stages of distention from any cause the muscle of the bowel may be exhausted from fatigue and overstretching. An intestinal segment which has been deprived of its arterial blood supply soon passes into a state of paralysis from ischemia. With these exceptions, however the underlying fault in ileus ordinarily is not paralysis of the intestinal muscle but rather an excessive neurogenic interference by way of sympathetic inhibitory fibers. Therefore, the term *inhibition ileus* would usually be more appropriate.

A standard classification of the causes of ileus is given in Table 11. Of particular im-

Table 11 Classification of Causes of Ileus

- I. Intra-abdominal
 - A. Peritoneal irritation
 - 1 Traumatic peritonitis (including operative trauma)
 - 2 Bacterial peritonitis
 - 3 Chemical peritonitis
 - B. Vascular changes
 - 1 Strangulation
 - 2 Mesenteric thrombosis or embolism
 - 3 Prolonged simple obstruction
 - C. Extraperitoneal irritation
 - 1 Hemorrhage
 - 2 Infection
 - 3 Renal lesions
 - 4 Trauma to retroperitoneal nerves
- II Extra-abdominal
 - A. Toxic
 - 1 Pneumonia, empyema
 - 2 Uremia
 - 3 Systemic infection
 - B. Metabolic
 - 1 Porphyria
 - C. Neurogenic
 - 1 Spinal cord lesions
 - 2 Fractures of spine or ribs in driving nerve roots
 - 3 Lead poisoning
 - D. Electrolyte imbalance

testine the resulting obstruction actually represents a failure of propulsion due to ischemic paralysis of the muscle of the affected bowel. When arterial obliteration is slow and the affected segment is short the collateral circulation may be sufficient to prevent actual gangrene particularly with the help of antibiotics. Sudden and massive occlusions, however usually result in extensive infarction of the intestine and this is followed by peritonitis and its sequelae.

Mesenteric occlusion is seldom diagnosed prior to operation unless one has reason to expect embolism or venous thrombosis in the patient in question. Although the prognosis always is poor it is not hopeless if the patient's condition will permit immediate resection of the stretch of gangrenous intestine with re-establishment of the continuity of the remaining bowel by anastomosis. Survival of a number of patients has now been recorded and in some of these the entire midgut had been gangrenous, so that resection was necessary from a level a few inches distal to the duodenojejunal flexure down to the proximal transverse colon. Patients with such massive intestinal resection suffer from anorexia, cramps and very severe diarrhea after operation. They present critical problems in replacement therapy and nutrition for many months. Impairment in the absorption of fats remains particularly severe. However the fact that some have survived disproves the former belief that removal of more than three-fourths of the small intestine necessarily results in a fatal nutritional disturbance.

SPECIFIC LESIONS CAUSING INTESTINAL OBSTRUCTION

Intestinal Obstruction in Infancy When congenital malformations result in complete intestinal obstruction, symptoms appear during the first day or two of life. Operative relief must be prompt or the infant will not survive. Parenteral fluid administration should precede surgery and gastric suction should be instituted to prevent vomiting and pulmonary aspiration. Otherwise, chest complications are a frequent cause of death.

The congenital malformations causing intestinal obstruction in infants include atresia and stenosis of the small intestine or colon, imperforate anus, meconium ileus malrotation or incomplete rotation of the midgut and duplications of the intestinal tract.

Intussusception. The term intussusception refers to the telescoping or invagination of a portion of small or large intestine into a more

distal portion. The receiving sleeve is called the intussusciptens and the entering portion, the intussusceptum. Obstruction always results. Not only is peristalsis interrupted, but also the mesentery of the advancing intussusception becomes progressively compressed so that this entering bowel segment becomes edematous violently congested and finally necrotic. The commonest intussusception is that of the ileum into the cecum or of the ileum and cecum into ascending colon. Rarely the ileum telescopes into the ileum or the colon into the colon. Though the usual intussusception consists of three layers or cylinders double and compound varieties are described consisting of five and even seven cylinders. Intussusception which is secondary to the drag of a pedunculated tumor occurs in adults but only rarely. In infancy and early childhood, in contrast, intussusception which is "idiopathic," i.e., has no demonstrable cause is so frequent as to classify it as one of the most important surgical emergencies. The experience that 75 per cent of these patients are less than one year of age and that the peak of incidence occurs in babies between the ages of four and ten months suggests that the change from milk to a more solid diet may have some connection with the occurrence of many of the cases.

The infant with intussusception usually is a chubby well nourished baby more often male than female. It is suddenly noticed that he appears to be having recurring attacks of violent abdominal pain which he evidences by grunting, crying, drawing up his legs and perhaps holding his abdomen. After the seizure passes the child appears playful and well until the next episode five or ten minutes later. Usually vomiting is an early symptom. As obstruction progresses, the child becomes pale and listless, even severely shocked. After one or more normal stools the bowel movements consist of red or brownish mixtures of blood and mucus. Sometimes bleeding is sufficient to produce hemorrhagic shock. Abdominal palpation reveals very little tenderness or spasm, but ordinarily a round or sausage-shaped mass or tumor is felt along the course of the right, transverse or descending colon, a tumor which stiffens with the painful seizures. At the same time, the right lower quadrant appears empty on palpation. On digital examination of the rectum, blood is usually found and occasionally the cervix like tip of the advancing intussusceptum can be palpated per rectum.

is essentially prophylactic and palliative. By avoiding preoperative cathartics and purgatives by examining immediately all patients complaining of abdominal pain so that diagnosis and operation will be prompt in appendicitis by performing surgical operations as gently and as expeditiously as possible and by using gastric suction routinely after major abdominal surgery one can accomplish a great deal in preventing unnecessary occurrence of ileus and in minimizing its extent when it does occur. Once ileus is established, operative surgery has little to offer aside from the procedures which may be indicated in treating the underlying cause of the ileus. Enterostomy is usually disappointing in that it collapses only one loop of the distended intestine. Furthermore it has been the general experience that the inhibited bowel fails to respond to such stimulating drugs as Prostigmin. Attention should be concentrated primarily upon supportive therapy and upon the prevention of additional distention through decompression. Since in most instances the intestine is not actually paralyzed, a balloon tipped tube can sometimes be passed down the bowel. Otherwise gastric suction prevents additional distention from air swallowing, provided attention is directed toward maintaining the patency of the Levin tube. Along with the effort to prevent any increase in distention, parenteral replacement therapy and the administration of antibiotics are combined with whatever treatment is proper for the primary lesion. An additional measure sometimes found useful is the interruption of sympathetic inhibition by splanchnic block or spinal anesthesia.

Spastic or dynamic ileus a temporary obstruction caused by spasm in a segment of intestine, is only occasionally of surgical importance. Patients exhibiting such intestinal spasm are apt to be of an extremely nervous make-up and one desires to avoid laparotomy if it is at all possible. However, since the symptoms are those of mechanical obstruction and the patient often has had a previous operation, the situation is easily confused with obstruction caused by postoperative adhesions. Inasmuch as adhesions usually obstruct the small intestine whereas spastic areas are more common in the colon, x-ray demonstration of distention of the colon often helps in diagnosis. Knowledge regarding findings at a previous operation is also of aid in determining whether surgery is necessary. Functional disorders of digestion associated with a spastic descending colon often present similar though milder symp-

toms. A test with atropine or some other antispasmodic drug is of aid in differentiating them.

VASCULAR OBSTRUCTION

The term *vascular obstruction* refers to the relatively infrequent obstructions caused by mesenteric vascular occlusion. The vascular accident may be either arterial or venous. Most commonly the superior mesenteric vessels are involved. Embolism of the superior mesenteric artery may occur in patients having vegetations on the heart valves, or other forms of heart disease particularly when accompanied by atrial fibrillation. Thrombosis of the artery as a result of atheromatous plaques or arteriosclerotic narrowing at its aortic origin is not unusual. Mesenteric venous occlusion most commonly is a thrombophlebitis secondary to abdominal sepsis (pyelephlebitis). It can also result from trauma or from portal stasis, especially in individuals suffering from some disease such as polycythemia which predisposes to intravascular clotting. In still other patients with venous thrombosis no cause whatever is found.

During the past few years a number of cases of occlusive necrotizing inflammatory disease of the smaller distal arteries of the superior mesenteric system have been observed during the postoperative period following surgical correction of coarctation of the aorta. Whether this occurrence is related to alterations in the circulatory dynamics following operation or to some other factor clinically the cases present the problems of mesenteric thrombosis.

Although the development of mesenteric vascular occlusion sometimes is insidious, with surprisingly few symptoms for a number of days, in the typical case the onset is sudden and catastrophic. There is severe abdominal pain, either continuous or cramping, with profound shock and collapse when the occlusion is massive. Vomiting usually is copious and occasionally the vomitus is bloody. Peristalsis is more often absent than exaggerated. The roentgenogram usually reveals the presence of fluid in the peritoneal cavity. Otherwise it may be interpreted as showing either mechanical obstruction or paralytic ileus. Characteristically marked leukocytosis is an early finding, although tenderness and rigidity may not be pronounced until gangrene has occurred in the affected intestine.

Since mesenteric vascular occlusion amounts to a denial of circulation to the in-

ministering morphine. Gentle manual reduction (taxis) can often be attempted more intelligently by the patient himself who is accustomed to reducing his own hernia. The possibility of *en bloc* reduction must be kept in mind. Unless nonoperative reduction is unmistakably successful operation is required for reduction. If the patient has symptoms of obstruction or if the irreducible hernial contents are tender tense and painful, the operative section of the constricting ring at the neck of the sac must be immediate. If after operative reduction, the intestinal loop concerned does not regain its color sufficiently to be clearly viable it must be resected. Anastomosis follows usually end-to-end, and then the repair of the hernia.

Volvulus. Some degree of twisting or torsion is not unusual in obstruction due to abnormal fixation of a loop of small intestine at some point owing to the presence of an adhesion band. Such secondary volvulus is an added factor in occluding the intestinal lumen and is particularly dangerous because of its strangulating effect upon the vessels in the mesentery. In its purer or "primary" form, however volvulus is most apt to arise when there is a lack of adequate mesenteric fixation. In congenital malrotation for example, volvulus of the entire midgut of an infant may result from defective attachment of the mesentery of that portion of the bowel. In adults, volvulus of this type is more frequent in the colon than in the small intestine. The long sigmoid loop is by far the commonest site, the narrow base of its mesentery no doubt predisposing. When volvulus of an abnormally mobile cecum is encountered, it is usually found to be a twist upon the axis of the ascending colon. Volvulus of the broad-based transverse colon is exceedingly rare.

The symptoms diagnosis and treatment of volvulus of the colon are discussed in the section of this chapter dealing with the colon.

Gallstone Ileus. This is the name given to mechanical obstruction of the intestine by a very large gallstone and is uncommon since cholecystectomy has become widely practiced. Usually the stone has ulcerated its way into the duodenum from the gallbladder rarely from the common duct, and the ordinary site for its lodgment is in the terminal ileum, though occasionally it lodges elsewhere. Ulceration and spasm may occur at the site, as may volvulus of the distended proximal bowel. For these reasons, the obstruction is a very dangerous one, particu-

larly since the obstructive symptoms often have relented and recurred to the extent that the patient comes late to surgery. The possibility of gallstone ileus should be kept in mind when obstructive symptoms arise in female patients having a history of gall bladder disease. Operation should be prompt and the stone removed from the intestinal lumen with minimal contamination of the peritoneum.

Diverticulitis. Diverticulitis of the sigmoid colon is a common inflammatory disease sometimes difficult to differentiate from sigmoid carcinoma in fact, although unrelated, the diseases sometimes coexist. Obstruction in diverticulitis is seldom a complete obstruction so that surgery is performed most commonly as an elective procedure in patients having recurring partial obstructions and in whom barium enemata in the past have demonstrated diverticulitis. With each recurring threat of obstruction in diverticulitis there is usually some degree of exacerbation of the inflammatory aspects of the disease so that tenderness in the left lower quadrant, "left-sided appendicitis," helps to differentiate it from sigmoid cancer. Occasionally however both the clinical evidence and that from the barium enema are borderline and confusing, so that exploratory operation is necessary. If diverticulitis is encountered under these circumstances and the bowel has been properly prepared for surgery a primary resection is feasible so long as there are no signs of acute inflammation. In the presence of marked inflammation however or in the event of acute obstruction from diverticulitis, it is much safer to limit emergency surgery to proximal decompressing colostomy since resection of the unprepared bowel in the presence of inflammatory edema is fraught with many dangers.

Fecal Impaction. Very rarely concretions formed within the intestine cause intestinal obstruction. The term *fecal impaction*, however does not refer to actual obstruction but to a disagreeable distention of the rectum by a hard mass of inspissated feces which the patient is unable to pass. Often there is a complaint of rectal tenesmus with constant desire to stool, which attendants may misinterpret as diarrhea. The diagnosis is obvious on digital examination. The mass must be broken up and removed digitally, with the help of irrigations of oil or of dilute hydrogen peroxide. When caring for elderly patients, especially when they are bedfast, one must guard against impaction

In the typical cases of intussusception the diagnosis can be made on the history and physical findings alone. When it is the rare ileocolic intussusception, x ray films of the abdomen reveal obstructed loops of small intestine. In other doubtful cases the findings on barium enema are very helpful if the intussusceptum has entered the colon. Obstruction of the barium column, a cupping of the head of the column a thin barium coating over the tip of the intussusceptum and spiral-shaped or ringlike stripes of barium are reported.

Operative reduction of intussusception is not difficult in patients with early cases. Through a right abdominal incision, the head of the intussusceptum is located in the left abdomen and gently milked backward into the right colon and thence through the ileocecal valve. Traction upon the proximal end of the intussusceptum is best avoided. If the reduced bowel is viable nothing more need be done. When irreducibility or gangrene necessitates resection and anastomosis of the remaining ends of bowel does not seem feasible at the first operation, the gangrenous loop can be exteriorized upon the abdominal wall. After the wall is closed about the limbs the gangrenous portion of the intestine is resected and the two limbs left as a double barreled Mikulicz enterostomy stoma. The enterostomy opening is closed as soon as practicable to alleviate the critical problems in water and electrolyte loss.

It has been known for a long time that in a great many patients with early cases of ileocolic intussusception, reduction can be accomplished safely by low pressure colonic inflation with fluids or air. There has been recent revival of this method, using a low pressure barium enema under fluoroscopic control. However surgical opinion still remains sharply divided as to the wisdom of attempting such nonoperative reduction routinely before resorting to a surgical operation. If one desires to use the method, the nonoperative reduction must be promptly and unequivocally successful and great care must be exercised to assure that there has been no significant delay if surgical reduction still proves necessary.

Obstruction by Adhesions. Bands or adhesions are a common cause of intestinal obstruction. Some peritoneal bands are congenital, but many result from earlier operations, chiefly upon the appendix and female pelvic organs. Obstructions occurring during convalescence from surgery or from

peritonitis usually are a result of adhesive attachments which are still soft, vascular and only partly organized. With decompression therapy recovery often follows without operation. However strangulation does ensue in 10 per cent or more of these patients and operative intervention is indicated unless relief is prompt under nonoperative management.

Obstructions which occur months or years after operation result from firm cicatricial bands stretching as bridges between two peritoneal surfaces. In some instances a knuckle of bowel becomes wedged into the window beneath the band, while in others a loop passes through sufficiently long to invite twisting or volvulus ending in strangulation. The majority of conditions giving rise to such adhesion bands are situated in the right iliac fossa or in the pelvis, although occasional obstructions due to attachment of a Meckel's diverticulum to the umbilicus are at a higher level on the right. A right paramedian incision is appropriate in exploring for such obstructions.

Strangulated Hernia. This is perhaps the commonest cause of acute intestinal obstruction. With the exception of rare internal and concealed hernias the site and cause of the obstruction are quickly apparent so that there is no clearer indication for immediate surgery. Although the intestine can become devitalized within two hours after the onset of strangulation, ordinarily gangrene is unlikely if the hernial "ring" is divided within four to six hours. In strangulated inguinal and femoral hernias, the trapped loops are usually ileal, while in strangulated umbilical hernias involvement of jejunal loops is more common. Although strangulation may be considered to be the logical end to which all hernias tend, those having a narrow and rigid neck particularly invite its occurrence. Femoral hernia is more apt to become strangulated than is any other on account of the rigidity of the anatomic structures about the neck of the femoral sac, and the most rapidly developing gangrene of all may be encountered in the Richter type of femoral hernia, in which only a small area of bowel wall is pinched tightly in the rigid hernial orifice.

In all patients having acute abdominal pain one must think of hernia and examine its usual sites of occurrence carefully. If an irreducible hernia is found, a trial of nonoperative reduction may be made adjusting the patient's position so as to relax the anatomic structures concerned and ad-

THE LIVER AND BILIARY SYSTEM

Anatomy and Physiology of the Liver and Biliary System and Diseases of the Gallbladder and Bile Ducts

By FRANK GLENN M D

FRANK GLENN was born in southern Illinois, attended Washington University and its medical school. Surgical training at the Peter Bent Brigham Hospital and The New York Hospital preceded his investigative interests in surgical problems of immediate importance to the care of the surgical patient. Successor to George Heuer he has brought a midwestern background to the Lewis Atterbury Stimson Chair of Surgery at Cornell University and The New York Hospital.

The liver and biliary system are so intimately associated with digestion and the body metabolism that impairment of their function diminishes the efficiency of the total body economy. An understanding of these processes requires an accurate knowledge of the anatomy physiology and pathology of these complete systems. Departures or variations from the normal range of structure and function are the basis for considerable

disability. Of course Nature allows for some latitude in both of these, but beyond a certain degree of deviation from the normal, which we call the pathologic, signs and symptoms appear that experience has taught us are indicative of failing health.

The interruption of these changes is important to the patient, first in that the conditions they produce must be corrected to release him of his symptoms, second, because

of feces in the rectum by regular attention to the bowels and by periodic rectal examinations

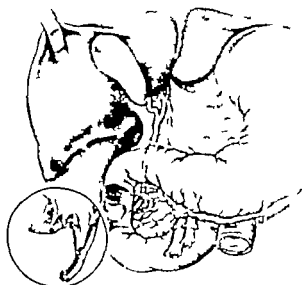
READING REFERENCES

- Abbott, W. O. and Johnston, C. G. Intubation Studies of Human Small Intestine: a Non-surgical Method of Treating, Localizing and Diagnosing the Nature of Obstructive Lesions. *Surg. Gynec. & Obst.* 66:691, 1938.
- Allen, A. W. Editorial. Acute Obstruction of the Small Intestine. *Arch. Surg.* 70:476, 1935.
- Barnes, J. P. Trocar Decompression in Acute Small Bowel Obstruction. *Surgery* 37:542, 1955.
- Becker, W. F. Acute Adhesive Ileus. A Study of 412 Cases with Particular Reference to the Abuse of Tube Decompression in Treatment. *Surg. Gynec. & Obst.* 95:472, 1952.
- Blais, A., III, Kennedy, J. D., Caliban, R. J. and Har-kins, H. N. Effect of Penicillin in Experimental Intestinal Obstruction. Cure of Strangulated Ileal Obstructions Treated with Penicillin Prior to Late Resection. *Arch. Surg.* 53:378, 1946.
- Bollinger, J. A., and Fowler, E. F. Results of Treatment of Acute Small Bowel Obstruction. *Arch. Surg.* 66:888, 1933.
- Case, J. T. Roentgenological Aid in the Diagnosis of Ileus. *Am. J. Roentgenol.* 19:413, 1928.
- Crowley, R. T. and Johnston, C. G. Physiological Principles in Intestinal Obstruction. *S. Clin. North America* 26:1427, 1946.
- Dennis, G. Current Procedure in Management of Obstruction of the Small Intestine. *J.A.M.A.* 154:463, 1954.
- Farber, S. Congenital Atresia of the Alimentary Tract. Diagnosis by Microscopic Examination of Meconium. *J.A.M.A.* 100:1733, 1933.
- Frimann-Dahl, J. Roentgen Examinations in Acute Abdominal Diseases. Springfield, Ill. Charles C. Thomas, 1951.
- Gardner, C. E. The Surgical Significance of Anomalies of Intestinal Rotation. *Ann. Surg.* 131:879, 1950.
- Gross, R. E. The Surgery of Infancy and Childhood. Philadelphia, W. B. Saunders Company, 1953.
- Ladd, W. E., and Gross, R. E. Abdominal Surgery in Infancy and Childhood. Philadelphia, W. B. Saunders Company, 1941.
- McKittick, L. S. The Diagnosis and Management of Acute Obstruction of Small Intestine. *New England J. Med.* 225:647, 1941.
- Meyer, H. W. Acute Superior Mesenteric Artery Thrombosis, Recovery Following Extensive Resection of Small and Large Intestines. *Arch. Surg.* 53:298, 1946.
- Michel, M. L., Jr., and others. Acute Obstruction of the Colon. *Ann. Surg.* 139:806, 1954.
- Miller, T. C. and Abbott, W. O. Intestinal Intubation. a Practical Technique. *Am. J. Med. Sc.* 137:593, 1934.
- Moore, R. M. and Kirby, O. T. Jr. One-Stage Resection in Selected Cases of Sigmoid Diverticulitis. *Ann. Surg.* 139:826, 1954.
- Moore, T. C., and Lawrence, E. A. Congenital Malformations of the Rectum and Anus. I. Clinical Features and Surgical Management of 120 Cases. *Surgery* 32:352, 1952.
- Morton, J. J. Diverticulitis and Carcinoma of the Sigmoid. *Surgery* 32:765, 1952.
- Noer, R. J., Robb, H. J. and Jacobson, L. F. Circulatory Disturbances Produced by Acute Intestinal Distention in Living Animals. *Arch. Surg.* 63:520, 1951.
- Poth, E. J. Intestinal Antisepsis. *Am. J. Surg.* 86:903, 1954.
- Poth, E. J., and McClure, J. N. Intestinal Obstruction, the Protective Action of Sulfasuxidine and Sulfathiazole to the Ileum Following Vascular Damage. *Ann. Surg.* 131:159, 1950.
- Ravitch, M. M. Reduction of Intussusception by Barrum Enema. *Surg. Gynec. & Obst.* 99:431, 1954.
- Sarnoff, S. J. and Fine, J. Intestinal Obstruction. I. The Protective Action of Succinylsulfathiazole Following Simple Venous Occlusion. *Surgery* 18:927, 1944.
- Snyder, H. H. and Chaffin, L. Embryology and Pathology of the Intestinal Tract. Presentation of 40 Cases of Malrotation. *Ann. Surg.* 140:368, 1954.
- Urickio, J. F., Calenda, D. G., and Freedman, D. Mesenteric Vascular Occlusion. *Ann. Surg.* 139:206, 1954.
- Wangensteen, O. H. Suction Siphonage in Acute Intestinal Obstruction. *J.A.M.A.* 101:1532, 1933.
- Wangensteen, O. H. *Intestinal Obstructions: Physiological, Pathological and Clinical Consideration with Emphasis on Therapy Including Description of Operative Procedures*, 3rd ed. Springfield, Ill. Charles C. Thomas, 1955.
- Webster, D. R., Hendrickson, H. W. and Carne, D. J. The Effect of Potassium Deficiency on Intestinal Motility and Gastric Secretion. *Ann. Surg.* 132:779, 1950.
- Weich, C. E., Allen, A. W., and Donaldson, C. A. An appraisal of Resection of the Colon for Diverticulitis of the Sigmoid. *Ann. Surg.* 138:332, 1953.

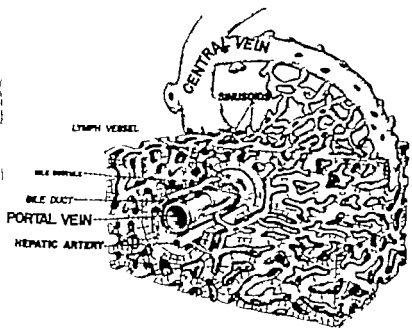
rate—estimated to be near 40 cc. per hour—and save for that time when active digestion is going on it goes to the gallbladder. There it is concentrated by the absorption of water and some salts through the wall of the

gallbladder. Thus the bile in the gallbladder may be concentrated five to ten times with a marked increase in the concentration of bile salts, pigment, calcium and cholesterol (Fig. 1B).

LIVER, GALLBLADDER AND EXTRAHEPATIC BILIARY DUCTS —



A



B

Figure 1. A, Gallbladder and extrahepatic ducts. Inset, Schematic detail of junction of cystic with common duct. B, Schematic diagram after Hans Elias portraying the liver as a spongylike structure of cells penetrated by communicating system of cavities or lacunae through which courses the blood from the portal tract to the central vein. These sinusoids are lined with the all-important Kupffer cells.

if they persist he may become more susceptible to additional disease processes and, finally, the longer their duration the more resistant they are to interruption or reversal.

The early recognition and proper interpretation of the signs and symptoms of biliary tract disease are essential to efficient therapy. The surgeon who undertakes to treat these patients must clearly understand the objectives, principles and technical procedures involved and their associated hazards. Furthermore the maximum success with the minimum risk will be provided the patient by the surgeon who is firmly grounded in the significance and frequency of anatomic abnormalities and the underlying pathologic processes of injuries and disease of the liver and biliary tract.

ANATOMY OF THE LIVER AND BILIARY SYSTEM

The liver, an essential organ of the body, may be considered as made up of four lobes—the right, left, caudate and quadrate. Each has a ductal system that receives tributaries from its functioning units and unites with those of the other lobes near the hilum to form the common hepatic duct (Fig. 1). Closely associated in this area are the hepatic artery and portal vein which enter the liver.

Until recently it was commonly accepted that the functioning units of the liver were the lobules that make up the lobe. It was believed that each lobule consisted of polygonal cells arranged about the ramifications of the portal vein, while on the opposite side or periphery were the small branches of the hepatic artery, hepatic vein and bile canaliculi. The lobules were considered as individual functioning units separated from each other by connective tissue. Now, chiefly because of the research of Hans Elias, the liver is looked upon as a spongelike structure of cells penetrated by a communicating system of cavities or lacunae. These contain the blood capillaries of the liver, the sinusoids. The endothelial lining is formed by the Kupfer cells. These are traversed by two mesenchymal tracts arranged about the vessels and bile ducts, one being the portal tract, the other the central vein.

In the liver cell, many complex functions take place that have far-reaching effects upon the body as a whole. Blood, both portal and systemic, arterial and venous, provides the materials in large quantity. The bile produced and secreted into the canaliculi is but a small part of these complicated processes.

The common hepatic duct is formed by the ductal systems from the lobes as they emerge from the liver in the hilum as the right and left hepatic ducts, about 4 cm. distal from the junction of the right and left hepatic duct the cystic duct unites to form the common bile duct. The common bile duct (ductus choledochus) measures about 8 cm. from the cystic duct to where it empties into the duodenum. It extends through the edge of the lesser omentum, passes behind the upper margin of the duodenum and through a portion of the head of the pancreas to enter the duodenal wall obliquely and empty into the intestine through the ampulla of Vater. In almost 70 per cent of individuals the main pancreatic duct empties into the common bile duct within 2 cm. of the ampulla of Vater. In the remainder the pancreatic duct may open directly into the duodenum. In addition to the blood from the portal vein, which lies medial and posterior to the common duct, the liver also receives systemic arterial blood from the hepatic artery which comes through the edge of the lesser omentum slightly to the left. The cystic artery which supplies the gallbladder usually passes posterior to the common duct and between the cystic duct and liver to enter the gallbladder wall. If one places the index finger in the foramen of Winslow and compresses the structures between it and the thumb, the flow within the cystic and hepatic arteries will be readily occluded. This is sometimes desirable when there is bleeding from the cystic artery in the course of surgical procedures in this area.

The gallbladder is a pear shaped structure attached to the inferior surface of the liver at the junction of the right and quadrate lobes. Its extrahepatic portion is covered with peritoneum, communicating with the common duct by the cystic duct; it receives bile as it is produced in the liver, concentrates it and later returns it through the same channel to the common duct and into the duodenum.

PHYSIOLOGY OF THE GALLBLADDER

The functions of the extrahepatic biliary system include the transportation, concentration and storage of bile after its production in the liver and before its delivery into the duodenum as needed. Bile as it comes from the liver is made up of 97 per cent water, 1 to 2 per cent bile salts and about 1 per cent of pigment, cholesterol and fatty acids (Table 1). The bile is produced at a constant

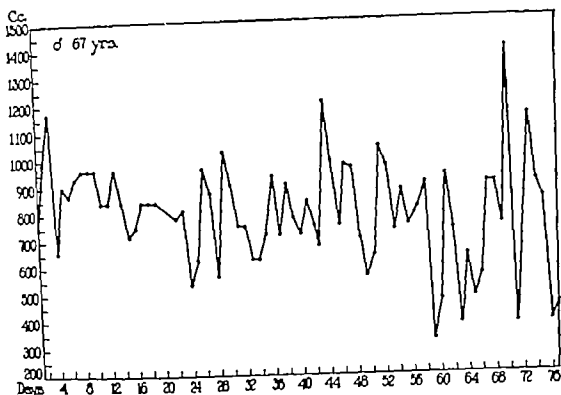


Figure 2. Biliary fistula, bile output in seventy-six days demonstrating considerable fluctuation from day to day but averaging slightly over 800 cc for twenty-four hours.

derived from the breakdown of hemoglobin by the Kupffer cells as well as bilirubin. By phagocytic activity they also remove bacteria, pigment and cellular elements from the blood.

Formation of Fibrinogen and Prothrombin. Prothrombin formation in the liver is governed by vitamin K. This substance in association with fibrinogen is essential for the clotting of blood. Fibrinogen is believed to be produced in the liver.

Red Blood Cell Production. Red blood cells are produced in the liver during embryonic life. Sometimes with the destruction of the bone marrow in later life, red cells may again be formed in the liver.

Detoxification. The function of detoxifying injurious substances brought to the liver by the portal circulation from the intestinal tract is not well understood. It is probable that some of the injurious substances are destroyed and others are conjugated with other substances to render them nontoxic, while still others are excreted as are some bacteria.

Vasodepressor Substance Elaboration. In the presence of hypoxia or anoxia, the liver elaborates a vasodepressor substance (VDM) that has been studied by Shorr. Since the production of this material may be initiated by the hypoxia caused by anesthesia and because toxic anesthetic agents

may cause liver necrosis, this function of the liver is of great concern to the surgeon.

CONGENITAL ANOMALIES OF THE GALLBLADDER AND EXTRAHEPATIC DUCTS

The gallbladder may be, but is rarely absent. It may vary greatly in size, shape and its attachment to the liver. An occasional gallbladder may be completely intrahepatic; it may be deeply embedded in liver substance or loosely attached by a mesentery. Bile ducts from the liver may enter the gallbladder directly or indirectly through the cystic duct.

Congenital anomalies of the extrahepatic ducts and the arterial system of this region are numerous and great emphasis has come to be placed upon them. The anatomic relationships vary so much that the surgeon must always be alert not to mistake structures. The cystic duct may be so short that it can hardly be identified, it may be very long and extend parallel to the common duct for several centimeters; it may enter the common duct from any point in its circumference. There may be duplication of a segment or of the entire common duct. There may be accessory ducts from the liver to the gallbladder in addition to the cystic duct and the common duct. There may be congenital cystic dilatation in any of these,

Table 1 The Composition of Bile

	LIVER BILE	GALLBLADDER BILE
Specific gravity	1.009-1.015	1.026-1.032
pH	7.1-8.5	5.5-7.7
Total solids (%)	1-3.5	4-17
Mucin (%)	0.1-0.9	1-4
Bile acids (%)	0.2-2	1.5-10
Bile pigment (%)	0.05-0.17	0.2-1.5
Total lipid (%)	0.1-0.5	1.8-4.7
Cholesterol (%)	0.05-0.17	0.2-0.9
Phosphatide (%)	0.05-0.08	0.2-0.5
Inorganic (%)	0.2-0.9	0.5-1.1
Total base (mEq/liter)	150-180	
Chloride (mEq/liter)	75-110	15-30
Calcium (mg %)	4-9	10-14
Iron (mg %)	0.03-7	

The flow of the bile from the gallbladder is believed to be stimulated by a hormone, cholecystokinin, which is secreted into the blood stream when acid chyme comes into contact with the duodenal and jejunal mucosa. Cholecystokinin causes an increase in the tone of the gallbladder increasing its intraluminal pressure from 300 mm. of water to nearly 375 mm. and returning the bile to the common duct. At the same time that the gallbladder contracts to return the bile to the common duct through the cystic duct, the sphincter of Oddi relaxes so that the bile flows through the ampulla of Vater into the duodenum without an increase in pressure within the common bile duct. When ingested, certain foods such as fats, egg yolk and those high in cholesterol act as a strong stimulus for the gallbladder to empty.

Loss of the gallbladder is compensated for by the dilatation of the extrahepatic ductal system. While it is probable that the digestive process is somewhat reduced in efficiency by the more constant flow of the bile into the duodenum after cholecystectomy no untoward symptoms appear. Many animals do not have gallbladders (e.g., horses, rats).

The concentrating function of the gallbladder is invoked in preparation for Graham-Cole cholecystography a test in which an iodine-containing compound administered to the patient and excreted in the bile becomes sufficiently concentrated in the gallbladder to be visualized on the x-ray film and enables one to outline gallstones and estimate the degree of normal function of the intact gallbladder if the dye enters it. If the gallbladder does not receive the dye, then it may be assumed that there is obstruction of the cystic duct or the ampulla of the gallbladder.

PHYSIOLOGY OF THE LIVER

The liver has many functions that are known and we believe there are many to be discovered. It is essential to life. Mann has demonstrated that death preceded by hypoglycemia follows within ten to twelve hours after hepatectomy. The liver also has great regenerative power. In the experimental animal as much as 70 per cent of the liver has been removed with apparent complete regeneration taking place in a few weeks. This same degree of regeneration has also been observed to take place in man.

Of the many known functions of the liver the following are of the greatest importance.

Carbohydrate Metabolism. The liver is one of the major body reservoirs for glycogen storage and consequently plays an important role in the regulation of the quantity of sugar in the blood. Glucose and other sugars are converted and stored in the liver as glycogen. Adequate glycogen aids the liver to resist the injurious effects of many toxic and noxious substances. Protein and a part of fat can also be used by the liver to make glycogen and are therefore important in carbohydrate metabolism.

Protein Metabolism. Protein metabolism, including deamination of the amino acids with the formation of urea and the destruction of uric acid, as well as the storage of proteins takes place in the liver. Here many of the serum proteins are formed.

Fat Metabolism. Fat is received from the circulation and may be stored in limited amounts in the liver. It may be broken down with the formation of ketone bodies. In fat deficiency the liver may form fat from other substances.

Vitamin Metabolism. Prothrombin, which is of such great importance in the clotting mechanism, is formed in the liver. Vitamin K is essential to its formation. Vitamin A is formed from carotene in the liver and vitamin B is stored and utilized in the liver.

Formation of Bile. Bilirubin, one of the important constituents of bile, is manufactured by the liver from the hemoglobin of broken-down red cells. The liver also forms bile salts and acids. These with cholesterol, a fatlike substance which is actually a higher alcohol, are also excreted by the liver and make up the other important constituents of bile. Bile is secreted continuously during a twenty-four hour period at about the rate of 30 to 50 cc. per hour for a total daily production of 800 to 1200 cc. (Fig. 2).

Gamma globulin and immune bodies are

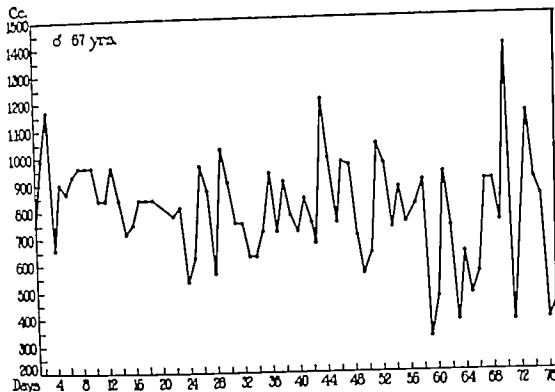


Figure 2. Biliary fistula, bile output in seventy-six days, demonstrating considerable fluctuation from day to day but averaging slightly over 900 cc for twenty four hours.

derived from the breakdown of hemoglobin by the Kupffer cells as well as bilirubin. By phagocytic activity they also remove bacteria, pigment and cellular elements from the blood.

Formation of Fibrinogen and Prothrombin. Prothrombin formation in the liver is governed by vitamin K. This substance in association with fibrinogen is essential for the clotting of blood. Fibrinogen is believed to be produced in the liver.

Red Blood Cell Production. Red blood cells are produced in the liver during embryonic life. Sometimes, with the destruction of the bone marrow in later life, red cells may again be formed in the liver.

Detoxification. The function of detoxifying injurious substances brought to the liver by the portal circulation from the intestinal tract is not well understood. It is probable that some of the injurious substances are destroyed and others are conjugated with other substances to render them nontoxic, while still others are excreted as are some bacteria.

Vasodepressor Substance Elaboration. In the presence of hypoxia or anoxia, the liver elaborates a vasodepressor substance (VDM) that has been studied by Shorr. Since the production of this material may be initiated by the hypoxia caused by anesthesia and because toxic anesthetic agents

may cause liver necrosis this function of the liver is of great concern to the surgeon.

CONGENITAL ANOMALIES OF THE GALLBLADDER AND EXTRAHEPATIC DUCTS

The gallbladder may be but is rarely absent. It may vary greatly in size and its attachment to the liver. An occasional gallbladder may be completely intrahepatic. It may be deeply embedded in liver substance or loosely attached by a mesentery. Bile ducts from the liver may enter the gallbladder directly or indirectly through the cystic duct.

Congenital anomalies of the extrahepatic ducts and the arterial system of this region are numerous and great emphasis has come to be placed upon them. The anatomic relationships vary so much that the surgeon must always be alert not to mistake structures. The cystic duct may be so short that it can hardly be identified, it may be very long and extend parallel to the common duct for several centimeters. It may enter the common duct from any point in its circumference. There may be duplication of a segment or of the entire common duct. There may be accessory ducts from the liver to the gallbladder in addition to the cystic duct and the common duct. There may be congenital cystic dilatation in any of these,

although the common duct is the more frequent site for such to occur. Defects in continuity and atresia of the bile ducts are not infrequent and are to be considered in young infants with persistent jaundice and inspissated bile and mucus.

CONGENITAL ATRESIA OF THE BILE DUCT

Complete obliteration of the lumen of any segment of the biliary tract is labeled biliary atresia. This may involve a part or all of the gallbladder, the cystic common or intrahepatic ducts. Dependent upon the location and extent, some of these abnormalities may be corrected surgically. Gross reports that in 146 patients with this condition twenty-seven or 18 per cent were subjected to a remedial anastomotic procedure.

If the atresia involves the common duct or one of its major hepatic tributaries obstructive jaundice results. Since jaundice in the newborn is relatively frequent it becomes indicative of atresia only if it persists and/or increases after two to three weeks. If the obstruction is in the extrahepatic common duct the stools from birth will be acholic. Thus as weeks elapse the jaundice and the pigmentation of the tissues become more marked. The circulating pigment in the blood stream may impart some color to the feces but this is minimal. Intermittent jaundice and intermittent normal-colored stools rule out the likelihood of atresia.

The obstructive jaundice is accompanied by progressive changes in the liver. It enlarges, becomes nodular, firm and a deep green color. The end result is portal cirrhosis with all gradations of liver cellular degeneration, together with considerable hepatic cell regeneration. As a result of this progressive extensive liver damage over a matter of months, a bleeding tendency develops because of a lowered prothrombin level. If the condition is uncorrected, the infant with biliary atresia, while maintaining a fair state of nutrition, develops slowly physically and mentally and is distinctly lethargic. As the liver enlarges splenomegaly and ascites appear and death usually occurs within the first two to three years of life as a result of liver deficiency.

The surgical treatment, when feasible, consists in establishing a satisfactory pathway for the bile to reach the intestine. When feasible, the most promising procedure consists in the excision of the atretic portion of the ductal system followed by establishment of continuity by an end-to-end anastomosis. If the atresia is in the common

duct distal to the cystic duct, then one may anastomose the gallbladder to the intestine to provide a pathway for the bile.

Over a twenty year period we have encountered twenty seven patients with congenital atresia of the ductal system at The New York Hospital-Cornell Medical Center. The diagnosis was confirmed at operation in seventeen, at necropsy in three, and at operation and autopsy in seven. The diagnosis was established between the ages of four days and one year. Twenty four of the patients were operated on, but in only ten cases was some form of surgical correction feasible. Although nine of these lived for from two months to nine and three-fourths years, only two are known to have a satisfactory result. Eight patients unsuitable for surgical correction lived for from fourteen months to six years. Because the usual microscopic sections of the liver in these patients with congenital atresia show the parenchyma to be composed of hepatic cells, separated by broad bands of fibrous tissue containing proliferating bile ducts, it is not likely that the results of surgical procedures will improve.

TRAUMA

An increasing number of automobiles traveling at greater speeds than ever before accounts for the more frequent occurrence of highway accidents which result in serious injuries. Among the victims of such accidents are persons who sustain major trauma to the abdomen without a penetrating or open wound. A very high mortality rate is associated with injuries to the liver and biliary system in this type of trauma.

Nonpenetrating trauma which produces rupture of a viscus may cause little evidence of external injury. When rapidly moving automobiles suddenly collide, occupants of the car may sustain injuries to the viscera with little evidence of external trauma, particularly if they are fully clothed. The liver under these circumstances, is frequently injured. Its location and attachment which prevent its escape from the forces involved, as well as its weight, consistency and shape, are factors contributing to the vulnerability of the liver.

Liver injury with hemorrhage and/or escape of bile is accompanied with an increase in pulse rate. The rapidity with which the pulse rate increases is often an index to the magnitude of the injury. An increasing pulse rate is indicative of an approaching fall of blood pressure and shock.

A degree of liver injury sufficient to produce shock usually will result in considerable hemorrhage and some escape of bile. However it may be that injury to other parts of the body caused the shock and the liver injury may have been sufficient to have resulted immediately in blood loss or bile escape. Thus the shock may be temporary and by the time the patient arrives at a hospital he may have a stable blood pressure and pulse. In either of these two situations, hemorrhage may again begin and continue. This together with the escape of bile into the peritoneal cavity can result in rapidly progressive shock which within a short time is irreversible. In addition this group of patients has usually sustained partial to complete interruption of the many normal physiologic functions of the liver. These are complex not only individually but in relation to the total body economy.

If the diagnosis of injury to the liver is made or is strongly suspected following such trauma as may be sustained in an automobile accident, the patient should be prepared and operated upon with as little delay as is compatible with anticipating the common exigencies. Delay increases the risk that the injury entails as well as the risk of the operative procedure.

There are all manner and variations of the extent and nature of these injuries. In general it may be said that free and partially detached fragments should be removed. In cases in which the injury is limited to the left lobe extensive lobectomy may be the procedure of choice. Rupture and superficial lacerations may be closed with any one of the various suture methods. Where there is complete rupture through capsule and parenchyma after removal of free fragments of liver massive sutures that seek to control hemorrhage and occlude the biliary ducts may be effectual. All traumatic wounds of the liver and biliary system should be provided with drainage which will permit the escape of bile through the abdominal wall to the exterior.

A gallbladder which is lacerated or detached from the liver bed should be excised. Lacerations of the extrahepatic ductal system should be meticulously repaired with fine interrupted silk sutures and proximal decompression instituted by means of a rubber T tube.

Following operation for trauma to the liver all patients will have some degree of peritonitis due to bile, hemorrhage or infection. Their treatment should include all

measures commonly employed, including gastrointestinal decompression maintenance of proper water and electrolyte requirements and chemotherapy.

Based on a review of the literature and our observations, we estimate that of those persons in automobile accidents who as a result of nonpenetrating trauma, sustain injury to the liver and biliary system which is not treated surgically, one-third die at the site of the accident, one-third die within six hours and one-third survive.

GALLSTONES

Incidence. It may be safely estimated that 10 per cent of the entire population of the United States and 20 per cent of those over forty years of age have gallstones.

If one looks at the incidence of the disease over the entire life span it is one that increases the longer life continues. In infants and young children gallstones are so rare that they are seldom given serious thought in a differential diagnosis. They have been reported in the newborn at autopsy. Before the age of puberty their occurrence is usually associated with some type of blood dyscrasia such as hemolytic jaundice. During adolescence, disturbances of metabolism may be present and stones may develop. In the female pregnancy is accompanied or followed by the appearance of gallstones with sufficient frequency to have established it as one of the most frequent diseases associated with gestation. Between the ages of thirty five and fifty-five years, men, and women who have not been pregnant, show an equally high incidence of symptoms of gallstones far beyond those of the younger age group. Studies of routine autopsy material have confirmed the clinical impression that there is a precipitous increase of gallstone incidence in individuals of the thirty-five-to-fifty-five-year group. Thereafter although the incidence does increase, it is more gradual. It has been estimated that one out of each three individuals who reach the age of seventy-five years will have gallstones. This should make us think about the changes in the physiologic functions of the body as it grows older. Indeed, the appearance of stones in adult life and their increased incidence with increased age are strongly suggestive that defects from the normal physiologic functions are involved.

It is to be observed in the analysis of various reported series of patients with gallstones that if the number of women who have been pregnant is subtracted from the

although the common duct is the more frequent site for such to occur. Defects in continuity and atresia of the bile ducts are not infrequent and are to be considered in young infants with persistent jaundice and inspissated bile and mucus.

CONGENITAL ATRESIA OF THE BILE DUCT

Complete obliteration of the lumen of any segment of the biliary tract is labeled biliary atresia. This may involve a part or all of the gallbladder, the cystic, common or intrahepatic ducts. Dependent upon the location and extent, some of these abnormalities may be corrected surgically. Gross reports that in 146 patients with this condition, twenty-seven or 18 per cent were subjected to a remedial anastomotic procedure.

If the atresia involves the common duct or one of its major hepatic tributaries obstructive jaundice results. Since jaundice in the newborn is relatively frequent, it becomes indicative of atresia only if it persists and/or increases after two to three weeks. If the obstruction is in the extrahepatic common duct, the stools from birth will be acholic. Thus as weeks elapse the jaundice and the pigmentation of the tissues become more marked. The circulating pigment in the blood stream may impart some color to the feces, but this is minimal. Intermittent jaundice and intermittent normal-colored stools rule out the likelihood of atresia.

The obstructive jaundice is accompanied by progressive changes in the liver. It enlarges, becomes nodular firm and a deep green color. The end result is portal cirrhosis with all gradations of liver cellular degeneration, together with considerable hepatic cell regeneration. As a result of this progressive extensive liver damage over a matter of months, a bleeding tendency develops because of a lowered prothrombin level. If the condition is uncorrected, the infant with biliary atresia, while maintaining a fair state of nutrition, develops slowly physically and mentally and is distinctly lethargic. As the liver enlarges, splenomegaly and ascites appear and death usually occurs within the first two to three years of life as a result of liver deficiency.

The surgical treatment, when feasible, consists in establishing a satisfactory pathway for the bile to reach the intestine. When feasible the most promising procedure consists in the excision of the atretic portion of the ductal system followed by establishment of continuity by an end-to-end anastomosis. If the atresia is in the common

duct distal to the cystic duct, then one may anastomose the gallbladder to the intestine to provide a pathway for the bile.

Over a twenty-year period we have encountered twenty-seven patients with congenital atresia of the ductal system at The New York Hospital-Cornell Medical Center. The diagnosis was confirmed at operation in seventeen at necropsy in three, and at operation and autopsy in seven. The diagnosis was established between the ages of four days and one year. Twenty-four of the patients were operated on, but in only ten cases was some form of surgical correction feasible. Although nine of these lived for from two months to nine and three-fourths years, only two are known to have a satisfactory result. Eight patients unsuitable for surgical correction lived for from fourteen months to six years. Because the usual microscopic sections of the liver in these patients with congenital atresia show the parenchyma to be composed of hepatic cells, separated by broad bands of fibrous tissue containing proliferating bile ducts, it is not likely that the results of surgical procedures will improve.

TRAUMA

An increasing number of automobiles traveling at greater speeds than ever before accounts for the more frequent occurrence of highway accidents which result in serious injuries. Among the victims of such accidents are persons who sustain major trauma to the abdomen without a penetrating or open wound. A very high mortality rate is associated with injuries to the liver and biliary system in this type of trauma.

Nonpenetrating trauma which produces rupture of a viscus may cause little evidence of external injury. When rapidly moving automobiles suddenly collide, occupants of the car may sustain injuries to the viscera with little evidence of external trauma, particularly if they are fully clothed. The liver, under these circumstances, is frequently injured. Its location and attachment which prevent its escape from the forces involved, as well as its weight, consistency, and shape, are factors contributing to the vulnerability of the liver.

Liver injury with hemorrhage and/or escape of bile is accompanied with an increase in pulse rate. The rapidity with which the pulse rate increases is often an index to the magnitude of the injury. An increasing pulse rate is indicative of an approaching fall of blood pressure and shock.

volves the cells adjacent to the smallest bile canaliculi. The reparative process that takes place in the liver is remarkable but the end result of repeated episodes of this nature is the biliary cirrhosis not infrequently seen in the old patient (over sixty five years of age) with a small contracted gallbladder containing stones and with stones in the common duct. Morphologic changes are evidence of advanced liver changes hepatic function, however may be markedly impaired before these are evident.

Finally malignant disease—carcinoma of the biliary tract—follows in the wake of gall stone formation. The incidence of stones associated with carcinoma is over 50 per cent, regardless of site and ranges from 50 to 90 per cent in reported series of patients with carcinoma of the gallbladder.

Gallstone Formation. Gallstones are concretions that form anywhere in the biliary tract, although it is believed that most of them form in the gallbladder. They vary greatly in content, size and shape.

They may be of almost pure cholesterol, inorganic calcium salts, calcium in combination with bilirubin, or any combination of a mixture of these substances and they often include cellular debris and bacteria. The cholesterol stones may occur singly tend to be rounded, of yellow white color and consist of radiating crystals. They are usually fairly large. Bilirubin calcium stones are black in color and assume various shapes, being irregular faceted or rounded. They vary greatly in size from that of sandlike particles to calculi a centimeter or more in diameter. They are of a homogeneous structure, either crystalline like the cholesterol stones or laminated like the mixed calculi. They are encountered in patients with hemolytic disease. The most common stones are the mixed type. These represent a combination of the two previously described and may contain cholesterol, bilirubin calcium, cellular debris and bacteria. They also vary greatly in size shape and consistency. They are usually laminated and have a central nucleus that is soft with superimposed concentric layers of varying amounts of cholesterol, bilirubin and calcium. Each concretion is considered to have an individual nucleus of a precipitate, cellular debris, bacteria or other foreign body material.

The mechanism whereby stones are formed in the gallbladder and biliary ductal system is not completely understood. Much study has been devoted to this problem. Many isolated and easily comprehended

facts have been revealed. Many more associated and complex fragments of information difficult to interpret have been brought forth by numerous investigators. No single theory provides an adequate explanation for all gallstone formation. Perhaps the most satisfactory explanation is that stone formation is the result of general variations from the normal functioning of the liver and biliary system. These may be the result of disturbances in metabolism of infection or of stasis or obstruction.

Bile metabolism. There are a group of diseases—congenital hemolytic anemia, sickle cell anemia and similar disorders—in which hemolysis of red cells produces an increase in the bilirubin excreted in the bile. In patients with such conditions the incidence of gallstones is high. The stones consist chiefly of bilirubin and are believed to form by crystallization as the result of hyperconcentration. The mechanism in disturbances of cholesterol metabolism does not appear as evident. However a few facts are known, for example bile acids keep the cholesterol of the bile in solution. In normal bile the ratio is about 20:1. If the concentration of the bile within the gallbladder reduces this to 13:1 cholesterol precipitates out in crystalline form and may be the nucleus of a stone.

Stasis and infection may play an important role in this change in concentration. Much investigation and study have failed to demonstrate a direct and constant relationship between the cholesterol content of bile and that of the blood serum. In pregnancy however it is common for both to be elevated, but all pregnant women do not develop gallstones. While some defect in cholesterol metabolism probably accounts for many instances of gallstone formation, we cannot be specific just where in the long and intricate chemical process it occurs.

Infection. Seventy years ago Naunyn was convinced that ascending infection in the biliary tract, when there was stasis, led to stone formation. Bacteria can be isolated from gallbladders containing stones removed at operation in over 50 per cent of the patients and from 20 per cent of stones. *Streptococci*, *Escherichia coli* and various members of the typhoid group are found most frequently but a wide variety of organisms may be encountered, including staphylococci.

Stasis. The role of stasis of bile alone in calculi formation has not been determined. It has been observed that there is a tendency for stone formation among those in whom there is evidence of comparative stagnation

total number of female subjects there will remain a number of women approximately equal to the number of male subjects. For example of every five patients with gall bladder disease four will be women and one a man, three of the women will have been pregnant, one will not have been. These conclusions are derived from studies made over the past fifty years. It is probable that as the old age group comes to constitute a greater proportion of the population, the ratio of gallstones in women who have not been pregnant and in men will increase.

Stones in the biliary tract seem to increase in size with the lapse of years. The presence of one or several stones seems to predispose to the formation of additional stones. Not only do the stones seem to increase in size and number but also they appear in locations other than the gallbladder. Indeed, stones may form in the gallbladder or in any part of the ductal system. Most of the stones form in the gallbladder but as time passes, more subjects are found to have stones in the ductal system. The size of the stones in the common duct of a person eighty to ninety years of age is unquestionably greater than that of the stone obtained from a younger patient.

Stones may be found in the biliary tract from birth to death. They are rare in infants, infrequent in children and adolescents, occasionally found in young adult males and in females who have not been pregnant, very common in all women recently pregnant, and they occur with gradual and increasing frequency in each decade of life of older individuals.

Relationship of Gallstones and Biliary Tract Disease. Gallstones are associated with the majority of disorders of the biliary tract encountered among the people of America. Thus in the consideration of biliary tract disorders, one may more concisely present a comprehensive picture of the subject by first following the natural course of gallstones and noting specifically the instances in which calculi are not involved or are relatively unimportant. Briefly stated, we may begin with stone formation in the gallbladder—the usual site of origin of most gallstones. It is true that cholecystitis, either acute or chronic, may occur without calculi formation, but such instances are relatively few. In the usual patient, stones in the ductal system are observed to appear and produce symptoms at varying periods after cholelithiasis has occurred. There is much evidence to indicate that the incidence of choled-

cholithiasis increases appreciably each decade after stones have appeared in the gallbladder.

To find stones in the common duct and none in the gallbladder is relatively rare. More frequently some time following cholecystectomy stones are found in the ductal system that were either overlooked at operation or formed subsequently. Common duct obstruction the result of benign stenosing fibrosis of unknown cause but frequently associated with a history of cholelithiasis, is an extremely rare entity. In the majority of subjects postoperative stenosis of the common duct is the result of injury to that structure when cholecystectomy was performed.

Another important relationship between gallstones and biliary tract disease is that existing between gallstones and liver damage, generally labeled cirrhosis. Probably no single mechanism accounts for the numerous variations of impaired liver function that result from cirrhosis. The variations appear to range from minimal derangements which are difficult to recognize clinically to complete liver failure. Liver damage is of great concern because much of the risk in undertaking surgical operations for biliary tract disease arises from impaired liver function. Almost 50 per cent of carefully prepared patients who die following operations on the biliary tract exhibit advanced liver damage.

When there has been long-standing disease of the gallbladder there are changes in the ductal system and the liver. It is believed that calculi, obstruction and infection in the gallbladder produce an inflammatory process which extends throughout the ductal system to involve the liver cells probably by way of the lymphatics. This has been labeled "cholangitis" and may vary in degree from minimal involvement evidenced only in edema microscopically to gross suppuration. Some scarring results from each episode of inflammation and therefore over the years, there is an accumulative injury to the liver that may eventually result in full blown biliary cirrhosis. The exact role and significance of the presence of calculi, infection and stasis or obstruction are difficult to establish because cholangitis may be the result of blood-borne infection from the portal or systemic circulation. An early example of this is to be observed in the young woman recently pregnant who has an attack of acute cholecystitis with a mild degree of jaundice. This attack is due to cholangitis which in

that colic may occur when there are stones in the gallbladder or biliary tract or when the gallbladder is inflamed with or without stones; (3) that the afferent nerve supply from the gallbladder and extrahepatic bile ducts is through the right and left splanchnic nerves to the posterior roots of T7 through T10; (4) that the more severe the pain, the greater is the area referred to by the patient. For example, the pain may begin in the epigastrium and as it increases in severity may be felt by the patient to be extending along the costal margin into the back and up beneath the right scapula and shoulder and then to the substernal area and sometimes to the left precordial region, left costal margin and left shoulder.

Attacks of biliary colic, acute cholecystitis and cholangitis may justify a diagnosis of gallstones. The demonstration of cholesterol and calcium bilirubinate crystals on microscopic examination of bile obtained from the duodenum by intubation is further evidence in establishing their presence. However, cholecystography according to the method devised by Graham and Cole in 1924 is by far the most widely used and the most satisfactory means of demonstrating directly or indirectly the presence of calculi in the gallbladder. The patient is given a dye (tetraiodophenolphthalein) that is excreted by the bile. The normal gallbladder concentrates the dye and may be visualized on the x-ray film. A diseased gallbladder does not concentrate the dye and, if the cystic duct is blocked, the dye will not enter. If there are stones in a gallbladder that has good function, they will be readily visualized. If the gallbladder is not visualized because of a lack of function or because the cystic duct is blocked, it may be assumed that the organ is diseased and contains stones. Cholecystography properly done will enable one to confirm the absence or presence of gallbladder disease in 90 to 95 per cent of the subjects (Fig. 3).

Recent developments in the technique of cholecystography, including intravenous administration of dye substances containing a higher iodine content, have made it possible to visualize the intact biliary ductal system and demonstrate stones and other abnormalities.

CHRONIC CHOLECYSTITIS

Chronic cholecystitis is a term designating disease of the gallbladder characterized by evidence of a chronic inflammatory process on gross or microscopic examination. Gall-

bladder function, as demonstrated by cholecystography, is usually impaired, but this is not always the case. Stones are present in a high percentage of the patients (Fig. 4). The term is a wide and comprehensive one and ranges from the process in which chronic inflammation is attended with minimal changes and vague symptoms of dyspepsia to that in which the gallbladder wall has been reduced to dense scar tissue contracted over calculi, the gallbladder is without function and frequently choledocholithiasis and liver damage are associated. Between these two extremes there are inexhaustible varieties and combinations of pathologic changes. The gallbladder with minimal microscopic involvement usually produces few symptoms but this is not invariable and occasionally such a gallbladder gives rise to severe symptoms. The most frequently encountered example of chronic cholecystitis is that of the patient who has had for months or years indigestion manifest by discomfort after eating. If a larger amount of food than is usual is ingested or if food high in fat content is included, there is in addition, belching and regurgitation for several hours. These symptoms may increase to be followed by pain and colic with nausea and vomiting. The duration of such episodes may be short or prolonged over hours or days. Many of these patients have had repeated episodes of acute cholecystitis. Some, however, may never have had an acute inflammatory process involving the entire organ. Rather the condition is believed to have been a slowly extending low-grade infection.

Symptoms. In early adult life, the symptoms may be of little concern to a patient when they first appear. Over a few years they may increase so insidiously that the exact time when they began to cause actual disability is difficult to determine. Under such circumstances, patients tend to accept their symptoms as something they must tolerate. They learn to keep them to a minimum by avoiding overeating and by abstinence from an ever increasing number of foods that they correctly or incorrectly believe cause their complaints. Many patients are little disturbed by these symptoms throughout their more robust years of life. During this period, the changes of biliary tract disease, namely increase in number and size of calculi, scarring of the gallbladder wall and reduction in functional efficiency of the gallbladder progress and, what is probably of greater significance, there is injury to the liver which may range from

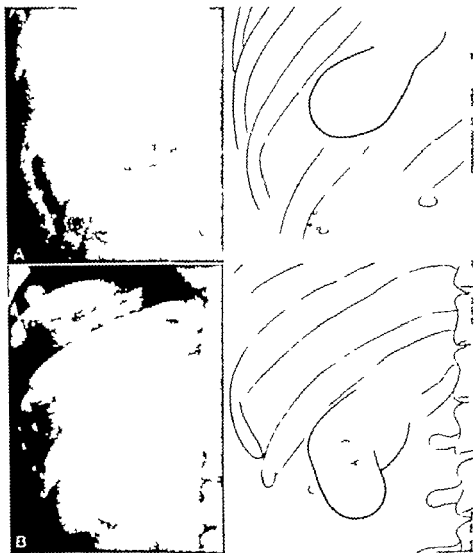


Figure 3. Cholecystograms made following the oral administration of the dye. A, Normally functioning gallbladder. B, Gallbladder with numerous nonopaque calculi and fair function.

of bile, as in pregnant women, obese individuals and those with organic obstruction to bile flow. As Naunyn pointed out, stasis and infection are frequently associated and this combination may provide the nucleus for stone formation.

Diagnosis. Gallstones usually give rise to a group of symptoms which render their presence evident. The sudden onset of pain in the epigastrium or near the right costal margin which radiates through to the back and sometimes to the right shoulder is typical of "gallstone colic." The pain is usually severe and may be agonizing. Its duration varies from minutes to hours and sometimes days. It may subside as quickly as it appeared, but there commonly exists a residual soreness in the area. The majority of attacks come on several hours after eating and at night. Large meals and those containing considerable fat such as the typical American Sunday dinner consisting of meat,

potatoes, fatty gravy and ice cream, are believed to play an important role in precipitating an attack. If the pain persists, nausea and vomiting often accompany it. The severity of the pain is characteristic and enables one to distinguish it from that due to other conditions encountered in this region. The patient is reluctant to move or take a full breath. If the pain persists, he or she may become despondent as did Sir Walter Scott, who after an attack of many hours "called his family and bidding them farewell turned his face to the wall to await death." Sir Walter Scott's attack, however subsided and the poet went to sleep to awake the next morning in fair spirits.

The mechanism whereby the pain of gallbladder colic is produced is not completely understood. Certain facts are known (1) that, in the conscious patient, distention of the gallbladder or any part of the ductal system by a rubber balloon can pain, (2)

disturbance or chemical irritation. Of these, the most significant is infection.

Role of infection. There are several channels through which infection may involve the gallbladder—these are through the bile, the blood stream and the lymphatics and by direct extension from adjacent viscera. In addition, there may be an ascending infection via the ductal system from the duodenum. Once the gallbladder is infected by bacteria, it may become the focus for infection which may manifest itself in different ways in various parts of the body giving rise to arthritis, neuritis or other disability. Sometimes the removal of a gallbladder which has been the site of long-standing infection is followed by a relief of symptoms due to affection in some other portion of the body. The role of infection in the pathogenesis of chronic cholecystitis has been subjected to numerous interpretations.

INFECTION FROM BACTERIA IN THE BILE. Many different bacteria are known to pass through the liver and into the bile. Many of these are destroyed or rendered inactive in the bile, while others seem to live, grow and seemingly are uninhibited. These bacteria may be grown from bile obtained at operation and include *Escherichia coli*, staphylococci, streptococci and typhoid groups. In acute cholecystitis without stones, the incidence with which positive cultures are made from material such as the wall of the gallbladder, the bile or the sentinel glands is rather high. And although the incidence is far less in the gallbladder of chronic cholecystitis, the same general bacterial groups are to be found. It is likely that if we had adequate means of taking material for culture from the ductal system, the gallbladder or the bile when there was no gross evidence of disease, the same organisms would be demonstrated. One may properly question whether the presence of such bacteria is pathologic or of clinical significance. Rolleston points out that in typhoid fever the bacilli are always present in the gallbladder but that cholecystitis is comparatively infrequent. I have removed the gallbladders from patients who were known typhoid carriers and whose bile prior to operation contained *Salmonella typhosa*, at operation the gallbladders were found to be normal both on gross and microscopic examination, but they did contain *S. typhosa*. Postoperatively these patients were free of this microorganism.

One may reason that as bile is concentrated in the gallbladder the number of organisms may be increased and that where

stasis is present for any reason optimum conditions would exist for the active invasion of the tissues by what may be considered passive organisms under other circumstances. There are many factors which may play a role in this process. There are the nature and virulence of the organisms as well as their actual number or concentration. One would need also to consider the over-all condition of the individual as to general resistance and susceptibility. Injury to the wall of the ductal system varying from that produced by simple distention to that sustained from a direct blow or piercing wound may provide the minimum needs for the invasion by these organisms to produce a clinical infection. The placing of large numbers of what have been considered as virulent organisms into the lumen of a gallbladder in animals has only infrequently produced cholecystitis.

One may conclude that many different bacteria are normally found in the biliary tract. In various types of cholecystitis—acute, chronic, calcareous and noncalcareous—there is ample evidence that these organisms may be playing an important role in the process. Certainly they are to be found in the bile, the wall of the gallbladder, the wall of the bile duct, in the smaller radicles of the liver causing cholangitis, and in the lymph glands such as the one that drains the gallbladder, the sentinel gland adjacent to the cystic duct and cystic artery.

HEMATOGENOUS INFECTION OF THE BILIARY TRACT. The arterial blood is supplied to the liver by the hepatic artery. The cystic artery, a branch of the hepatic, supplies the gallbladder and some individuals have small vessels passing directly to the gallbladder from the hepatic artery. In severe bacteremia, organisms may be found in both the liver and gallbladder as well as in other locations throughout the body. Under such circumstances, there may be multiple metastatic infections evidenced by true abscess formation. These may involve any system, organ or tissue throughout the body. The mechanisms which determine whether staphylococci that gained their primary focus in the throat may cause osteomyelitis or acute fulminating cholecystitis are not understood. There are plausible theories advanced. Wilkie observed the appearance of gallstones and acute cholecystitis in rabbits that were given intravenously large numbers of bacteria grown from the biliary tract of patients with biliary tract disease. Such observations do not establish a relationship

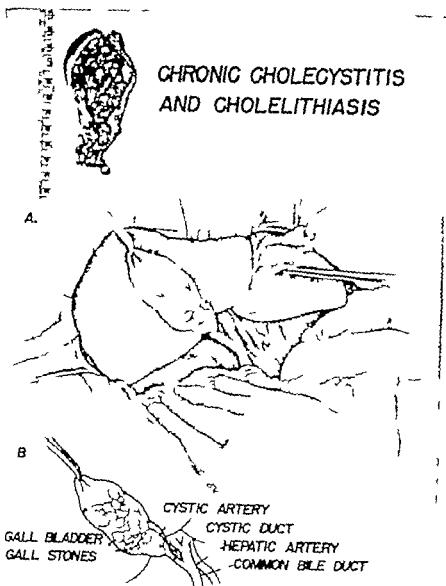


Figure 4. Chronic cholecystitis with cholelithiasis. A, Diseased gallbladder containing stones. B, Line drawing to emphasize anatomic relationships.

negligible damage to frank cirrhosis. Undoubtedly there are instances in which the process seems to subside and the patient appears to have been symptom free for years. If stones are present, the patients are candidates for an obstructive type of acute cholecystitis which is one of the most frequent conditions leading to abdominal operation in persons over sixty five years of age. Although these individuals may not have been aware of symptoms they have had progressive injury to their liver and, if this has been considerable, they may be poor operative risks when they present themselves for surgery. Such situations are frequent because many patients with chronic cholecystitis are able to tolerate their symptoms for years and only when the degree of

disability becomes marked do they seek relief.

Diagnosis. The diagnosis of chronic cholecystitis associated with cholelithiasis is established by a history of the symptoms as listed above and the demonstration of cholelithiasis and/or the impairment or absence of function. Cholecystography is one of the most accurate aids in diagnosis. With careful and proper evaluation, chronic cholecystitis and cholelithiasis may be accurately diagnosed in over 93 per cent of the patients.

Chronic cholecystitis without stones may be difficult to diagnose. Its pathogenesis is important.

Pathogenesis. It is believed that most cases of chronic cholecystitis with or without stones are the result of infection, metabolic

On microscopic examination the cholesterol is to be seen within the epithelial cells of the mucosa and beneath them in large macrophages. It is very easy for one to reason that this condition portrays a phase in the development of cholecystitis.

Role of chemical irritation. Derangement of the normal chemical reactions in the gall bladder may lead to irritative substances that play a role in the development of chronic cholecystitis. Ordinarily acidification of the bile occurs in the gallbladder if it does not, calcium carbonate is precipitated. The presence of calcium carbonate is indicative of irritative chemical activity and this compound may constitute a nidus for the precipitation of other constituents of the bile.

MILK OF CALCIUM GALLBLADDER. Calcium carbonate in almost pure form may be found in the gallbladder as a milk white paste. It may or may not be accompanied by stones but is almost invariably associated with microscopic changes in the gallbladder wall that justify a diagnosis of acute or chronic cholecystitis. On x-ray films the calcium material may be seen outlining the entire gallbladder or concentrated in the fundus. In cholecystography there is poor concentration of the dye. If calculi are present they may be laminated because of deposition of calcium over a previously formed cholesterol stone. These produce a spectacular picture on the x-ray film with the laminated calculi displacing the more dense milk of calcium or calcium carbonate.

CALCIFICATION OF THE GALLBLADDER. In the aged with long standing biliary tract disease, the gallbladder may appear to be calcified on x-ray examination. Such a gallbladder usually contains stones with laminations of calcium and there are varying degrees of calcium deposition in the wall. The origin of the calcium is believed to be from the bile and gallbladder mucosa. At operation the gallbladder is almost stonelike. It may be enlarged with a thickened wall and contain many stones or it may be small and contracted over one or a few small stones. In either event the likelihood of associated malignant disease is great and should be looked for both on gross and microscopic examination. The calcified gallbladder containing calculi cemented together with calcium carbonate is an impressive lesion at operation. It may be considered as due in part at least to derangement of the normal chemical change that goes on within it.

HYDROPS OF THE GALLBLADDER. It is probable that hydrops of the gallbladder is an

other example of cholecystitis due to chemical derangement secondary to obstruction of the cystic duct. This may be due to stones in the valvular portion that had their origin in the gallbladder. Hydrops may occur. It may occur in the absence of infection or stones and without evidence of changes in the cholesterol metabolism. The cystic duct is obliterated by scar tissue and is likely the basis of this condition. The bile can no longer enter the gallbladder but the mucous cells continue to secrete. The gallbladder becomes enlarged and distended with a viscid-like mucus. The wall is thin but edematous grossly and on microscopic examination a single layer of epithelium is seen lining it.

Differential Diagnosis. The differential diagnosis of chronic gallbladder disease requires careful evaluation of the patient's history and correlation with the findings on physical examination and the laboratory data, including x-ray examinations such as gastrointestinal series and intravenous pyelograms. The demonstration of gallbladder disease by cholecystography does not exclude the possibility of other conditions as the cause of symptoms referred to the epigastrium. The more common of these are peptic ulcer, renal disease, lesions of the large bowel including diverticulitis and neoplasm, pancreatitis and diaphragmatic hernia. The association of gallstones and heart disease has long been recognized. Stewart and Paterson have recently re-emphasized that the differential diagnosis between the two may be difficult, that the two may co-exist, and that when they do cholecystectomy often reduces the symptoms due to coronary disease.

Treatment. The treatment of chronic cholecystitis with cholelithiasis is surgical. Long experience has demonstrated that if stones are removed from a gallbladder and the organ remains stones are likely to form within a period of months. Cholecystostomy for the removal of stones had been a common practice prior to Langenbach's report of cholecystectomy for chronic cholecystitis and cholelithiasis in 1884. Not long thereafter Kümmell (1860) reported an exploration of the common duct with removal of stones. Over the past seventy five years an increasing experience coupled with improved facilities has made surgical treatment of biliary tract disease less difficult. Operative procedures are now well standardized and when carried out in carefully selected and well prepared patients are associated with minimal risk.

Indeed, the over all mortality for chole-

between the biliary tract disease in the human being and that found in the rabbit. Nevertheless one is not justified in saying that there is no relationship or that bacteria isolated from the gallbladder of a human being may not be grown in the blood stream of the experimental animal and be found in considerable concentration in its biliary tract.

SPREAD OF INFECTION THROUGH ADJACENT LYMPHATIC SYSTEMS WHICH INTIMATELY ANASTOMOSE WITH THOSE OF THE BILIARY TRACT The lymphatics of the liver and of the biliary ductal system including the gall bladder are connected by a luxuriant network of anastomosing channels with those of the duodenum, right colon and appendix, pancreas and lower stomach. Although there have been many investigations of these lymphatics, their true importance has been neglected. The commonly known facts are that the lymphatics here as elsewhere are concomitantly associated with the blood vessels both arterial and venous. In the lower stomach, duodenum and large bowel they conform to those elsewhere in the gastrointestinal tract. The structure is much the same in the extrahepatic biliary ductal system and gallbladder. The intrahepatic lymphatics however are a part of the complexity of the liver. These delicate tubules it is believed, provide the channels for the transportation of protein from the interstitial spaces of the liver to the blood vessels. When functioning to capacity and filled, they are evident structures but in the experimental animal and after death they are difficult to visualize. In their fine ramifications these lymphatics surround the functioning liver cells. If this concept is kept in mind one can readily imagine how simply infection due to organisms that may have their primary focus at some distance as for example in the appendix, may spread in the liver and biliary tract. There are, it is believed, many protective mechanisms with which we are not familiar that tend to prevent the extension of these infections.

The mechanism that is evident in an attack of acute cholecystitis is of importance in considering the relation of gallstones and liver damage. Disregarding for the moment the origin of gallstones and recalling that bacteria may be obtained from the gallbladder wall, the bile, the stone and the lymphatic glands in chronic biliary tract disease then it is easy to understand how the stone providing trauma and irritation, may play a role in repeated episodes of inflammation

within the gallbladder in which infection extends via the lymphatics to the liver and in turn leads to liver cell injury and scarring. The liver with its ability to recover from such insults and even to "regenerate by hypertrophy" may for a long time tolerate these episodes without clinical manifestations. On the other hand, the degree of liver involvement may at times be sufficient to cause jaundice. This is seen in young women with acute cholecystitis who have recently been pregnant. These patients may be so jaundiced that the bilirubin rises to 3 mg. per 100 cc of blood serum. This possibility should be kept in mind and the jaundice should not be considered as evidence of a common duct stone. Over the years a patient with gallstones may never be free from low-grade cholangitis and hepatitis resulting from an infection in a gallbladder containing stones. Eventually the amount of liver injury may become clinically evident as cirrhosis. When reduction of the functional capacity of the liver has not been recognized and the patient is subjected to an operative procedure hepatic failure may occur.

Liver damage associated with biliary tract infection and calculi is believed to be best explained by extension and reinfection through the lymphatics. A fair proportion of the deaths which follow surgery on the biliary tract are due to the liver damage that is the result of long standing gallstones.

Role of metabolic disturbances. Metabolic disturbances may be the basis for chronic cholecystitis as well as for the production of stones in the gallbladder. It is possible that these disturbances may occur and be the cause of chronic cholecystitis without stone formation. There is considerable evidence that disturbance in the metabolism of cholesterol is one of the most important of the metabolic anomalies.

CHOLESTEROSIS. The deposition of plaque-like accumulation of cholesterol on the mucosal surface of the gallbladder is known as cholesterosis of the gallbladder. It is believed that while this is seen in a normal and well-functioning gallbladder it may be seen also in association with chronic cholecystitis. Because the cholesterol plaques are opaque yellow white and are attached to the deep red mucosa, the term "strawberry gallbladder" has become synonymous with cholesterosis. Although these cholesterol deposits may be related to calculi formation, the relationship has not been clearly established. It is doubtful if symptoms or clinical signs are produced by this condition.

examination, the entire organ is seen to be involved by the acute inflammatory process scattered areas of ischemia and necrosis are noted.

Acute cholecystitis may occur in the absence of calculi and accounts for 5 to 10 per cent of reported cases. There are several etiologic factors that contribute to this condition. They include bacterial infection that is derived from the liver the blood stream and the lymphatic channels. Regurgitation of pancreatic juice is held by some to be of importance as is also the reaction produced by bile-salt concentration within the gall bladder. Then there are circulatory disturbances such as arteritis and arteriolitis seen in association with terminal hypertensive cardiovascular disease and severe bacteremia.

There is frequently a history of a systemic infection due to one of many organisms such as *Escherichia coli*, staphylococci and hemolytic streptococci. An upper respiratory infection with superimposed secondary invaders is for example more frequently a precursor than is a localized infection such as osteomyelitis. An infection caused by a virulent organism which overwhelms the body resistance no matter where it gained its foothold may be accompanied by acute cholecystitis. In years past, typhoid fever was a classical example of this.

An attack of acute cholecystitis in a gall bladder without stones is less sudden in onset. There is discomfort and then pain in the hepatic region with anorexia in the beginning. As the pain increases the area becomes

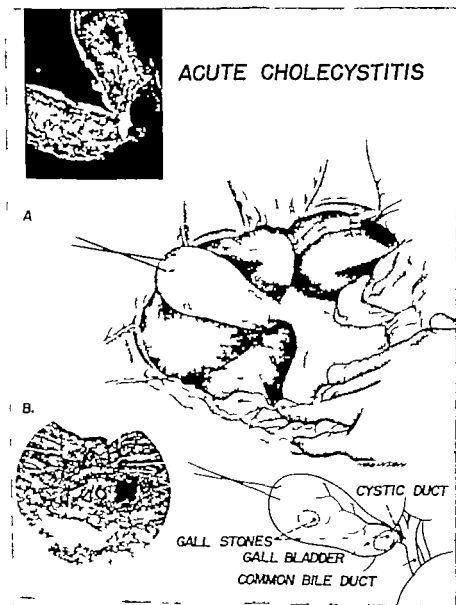


Figure 5. Acute cholecystitis with stone impacted in the ampulla of the gallbladder A, Photograph of specimen with stone in the ampulla and marked degenerative change. B Photomicrograph ($\times 20$) Acute inflammation of the gallbladder wall with necrosis.

cystectomy for chronic cholecystitis with cholelithiasis is less than 1 per cent. If operation were to be done in these same patients when the diagnosis was first established, it might be reasonable to expect the present operative mortality rate to be reduced by 50 per cent. The basis for this statement is that in a study of deaths (sixty-six) following 3439 operations for nonmalignant disease of the biliary tract, the largest single cause of death was hepatic insufficiency resulting either from acute or chronic infection, necrosis or combinations of these factors. There were nineteen deaths or 30 per cent of the total, due to hepatic insufficiency. Hepatic insufficiency is but one of the results of long standing disease.

The problem of chronic cholecystitis without stones is sometimes a difficult one. If there are gross changes such as thickening of the gallbladder wall, scarring and distortion, even though there are no stones encountered at operation in a patient in whom a diagnosis of chronic cholecystic disease has been established, cholecystectomy should be done. However the removal of a gallbladder without stone is so frequently followed by persistence of the presenting symptoms that we are reluctant to do a cholecystectomy unless we are convinced that the changes are definite. Actually few gallbladders are removed for chronic cholecystitis without stones.

Another problem that is encountered with increasing frequency because a greater proportion of our population is being subjected to periodic complete clinical evaluation including cholecystography is the demonstration of unsuspected cholelithiasis. Because one cannot predict the future course of the "silent stones" which may cause obstructive acute cholecystitis of a rapidly fulminating type in the older age group and because of the low mortality rate associated with cholecystectomy in such patients operation is indicated.

ACUTE CHOLECYSTITIS

Acute cholecystitis is often superimposed upon chronic cholecystitis that is associated with cholelithiasis. However the acute phase of the disease may also appear as the primary involvement of the biliary tract and it may develop in the absence of stones. In contrast to chronic cholecystitis the acute type presents the clinical manifestations of an acute inflammatory or obstructive process. Though the acute attack usually subsides it can progress and, if it does, may result in

perforation, abscess formation or local or generalized peritonitis. In addition to involvement of the gallbladder there is almost always extension of the infection to the biliary ductal system, namely cholangitis of varying degree. Each episode of acute cholecystitis results in some injury to the liver.

The clinical diagnosis is established by careful evaluation of the patient's history of his symptoms and of the signs elicited on examination. In the typical case a fairly long history of recurring episodes of biliary colic frequently precedes the onset of the acute attack. In some subjects however there is no record of previous symptoms referable to the gallbladder. The pain is severe and is located in the midepigastrium and the right upper quadrant and may radiate to the shoulder or back. Nausea and vomiting frequently accompany the onset of pain in these patients.

The physical examination reveals marked tenderness and sometimes muscle rigidity in the right upper quadrant. The gallbladder may be palpable as a distended tender mass. The patient looks ill and has a rapid pulse, some fever and an elevated leukocyte count. Some patients whose attacks have lasted more than twenty-four hours have a mild degree of jaundice. There is considerable variation as to clinical findings among patients with acute cholecystitis, particularly in those of the older age group. Some have no fever, others no leukocytosis and in still others there may be little tenderness over the enlarged and palpable gallbladder.

At operation the gallbladder is found to be reddened and distended, its wall is thick and edematous. Besides one or more stones the organ usually contains bile mixed with blood, colorless bile or pus under pressure. Usually there is a stone lodged in the ampulla of the gallbladder (Fig. 5) or the entrance of the cystic duct, blocking the outflow. On close inspection, areas of necrosis and gangrene may be noted and in some a frank perforation will be found with inflammatory reaction about the entire gallbladder and adhesions to neighboring structures. Free perforation with general peritonitis also may occur. The favorite location for such a perforation is in the fundus. This avascular area in the presence of inflammation of the gallbladder and compression of its blood vessels is most likely to become gangrenous first. Necrosis of this portion of the gallbladder in the presence of increased intracystic pressure may early result in perforation and escape of the contents into the abdominal cavity. On gross

tis is surgical. There has been considerable controversy during the past twenty years as to when surgery should be embarked upon; however the trend has been toward early operation. Some prefer to encourage the subsidence of the attack by keeping the patient quiet, allowing nothing by mouth and keeping the stomach decompressed with an indwelling tube and then at some later time removing the gallbladder. We prefer early surgical treatment of acute cholecystitis rather than the delayed.

The early surgical treatment consists of cholecystectomy or cholecystostomy as soon as the patient has been so prepared as to be considered in optimum condition for operation, unless there is some condition present which is not immediately reparable and which contraindicates operation.

Although cholecystectomy is the procedure of choice in acute cholecystitis the surgeon should keep an open mind as to just what is to be done until he has evaluated the findings. Cholecystostomy, a lesser procedure, may be indicated in the presence of peritonitis following a perforation of the gallbladder when the inflammatory process obscures the anatomic relationship of vital structures in the biliary fossa, in the presence of severe jaundice due to common duct obstruction and when the general condition of the patient is so grave that a general anesthetic and a prolonged operative procedure are contraindicated. Cholecystostomy under such circumstances is a compromise procedure but may be lifesaving, tiding the patient over a critical period. Definitive surgery that is cholecystectomy should be done a few months later. Cholechootomy may sometimes be indicated in the presence of acute cholecystitis, particularly in patients of the older age group in addition to cholecystectomy or cholecystostomy. The usual indications for cholechootomy are both less easily recognized and not as reliable in the presence of acute cholecystitis. Jaundice is frequently unassociated with cholecholethiasis, dilatation and induration of the common duct are difficult to evaluate because of inflammatory reaction, and stones are not as readily palpable for the same reason.

Acute cholecystitis constitutes a problem of varying importance and urgency depending on whether it occurs as an early phase of biliary tract disease, in a young woman recently pregnant, for example, or as an episode in the later stages of chronic biliary tract disease, as with men and women in the older age group or as a complication of a

terminal systemic disease or following some operative procedure unrelated to the biliary tract. For acute cholecystitis in the young, recently pregnant woman, since the postoperative morbidity of complications and the mortality rate are minimal and the complications of perforation and peritonitis are infrequent we believe that operation is the best method for interrupting biliary tract disease and that early surgical treatment should be employed. In both women who have not been pregnant and men in a slightly older age group but still under the age of fifty years acute cholecystitis appears to be a somewhat more severe process. Complications under nonsurgical treatment are more likely to occur but this group will tolerate operation almost as well as the first unless they are suffering from some systemic involvement such as cardiovascular disease, diabetes or renal impairment. A third group consists of those over fifty years of age the majority of whom have a long history of biliary tract disease. The older the patient and the longer the duration of biliary tract disease the more common are the complications of acute cholecystitis if not treated by surgery. By and large the older the patient the more difficult it is to estimate the exact nature of the process by physical examination, temperature elevation or white blood cell count and thereby determine whether the wall of the gallbladder is gangrenous, or whether perforation is impending. Furthermore, the incidence of common duct stone is greater in the older group. These patients are also more prone to develop postoperative complications which contribute to a higher mortality rate. Here again, however, early surgical treatment is the best protection for the patient.

In a final group are those patients who develop acute cholecystitis as a complication of the terminal phase of a systemic disease, such as cardiovascular disease with marked arteritis and arteriolitis. Whether or not operation is justified for such patients depends chiefly on the probable expectancy of life at the time. Since this is extremely difficult to estimate and since catastrophic perforation of the gallbladder with resulting bile peritonitis can be averted by cholecystostomy performed with the patient under local anesthesia, surgery to us seems indicated. When acute cholecystitis develops as a complication following operation for conditions unrelated to the biliary tract, the problem is primarily one of diagnosis and the type of surgical treatment is dependent upon the

tender and the gallbladder enlarges. Although there may be waves of increased pain similar to that due to biliary colic, such is not the common course of events. Systemic manifestations such as temperature elevation and leukocytosis are present. As the gallbladder enlarges the peritoneal reaction increases and the local signs become marked. In many instances the peritoneal reaction is indistinguishable from the reaction produced by a perforation. Sometimes, patients operated upon for presumed perforation have been found to have noncalculareous acute cholecystitis with a very flagrant and intense inflammatory process which extends to and involves the adjacent viscera and omentum.

The gallbladder in the early hours of an attack is edematous; there is a rapid increase in thickness of the wall of the gallbladder and obliteration of the cystic duct. The cystic duct may be occluded so that no bile enters it and none is emptied through it into the common duct. On microscopic examination, edema is noted as well as polymorphonuclear infiltration about the vessels. Bacteria may be demonstrated in these tissues. As the process progresses the organ becomes injected, distended and tense and the wall phlegmonous. Ischemic areas become apparent and necrosis with perforation may take place. In the beginning the bile in the gallbladder is normal in appearance and consistency. It may become diluted with thin mucoid material and then there may be hemorrhage. Usually the same organisms are to be obtained from the bile as are found in the wall or sentinel gland. At any point in this inflammatory reaction the process may subside either because of the natural resistant mechanisms of the body, a decrease in the virulence of the organism or specific chemotherapy.

Following the subsidence of an initial attack, there is healing of the ulcerations of the mucosal surface and scarring of the wall. Depending on the amount of tissue destroyed by the infection, the function of the gallbladder may be impaired. This may be so slight that it is of no particular importance or it may be so marked that the gallbladder is no longer capable of receiving or concentrating bile. In either event, this initial episode may be but the beginning of a long series of events in the advancement of biliary tract disease. It is likely that the organisms persist in the gallbladder and biliary passages and there will be recurrent attacks. The alterations in function and the residual

debris from the infection at its height may provide a nidus for gallstone formation.

Acute cholecystitis has been produced in animals by a variety of ways. A number of materials administered intravenously such as Dakin's solution, have produced it. Bacterial toxins of pathogenic organisms have produced ulcerative lesions in the mucosa. Various substances have been introduced into the gallbladder directly in an attempt to duplicate the regurgitation of pancreatic juice or duodenal contents into the biliary ductal system and gallbladder. These substances have been introduced directly with a needle and have produced acute cholecystitis of varying degree followed by healing that completely obliterates any evidence of what took place. This may be of clinical significance in that it emphasizes the fact that the causative factors in biliary tract disease are likely to be long standing and persistent.

The reflux of pancreatic juice containing enzymes into the gallbladder is believed by some to be the cause of a fulminating type of acute cholecystitis. The mechanism for such a reflux requires that the pancreatic duct empty directly into the common duct and that there be some obstruction between this and the opening into the duodenum. Thus a stone in the ampulla of Vater or spasm of the sphincter of Oddi could provide the immediate cause for the reflux.

In descriptions of operative findings in patients with acute pancreatitis it is occasionally stressed that there was also acute cholecystitis. The degree of gallbladder involvement seems to vary greatly. Only minimal edema is described in some while in others the description of a well advanced stage of acute cholecystitis has been reported. One could well consider that when the pancreatic duct joins the common duct proximal to the ampulla of Vater and there is an obstructive stone bile might pass into the pancreatic ductal system and produce acute pancreatitis and at the same time a mixture of pancreatic juice and bile could be regurgitated into the gallbladder resulting in acute cholecystitis. This probably does occur and material removed from such gallbladders has been demonstrated to contain pancreatic enzymes. However in a rather large series of patients I have treated surgically for acute cholecystitis it was by no means a clear-cut entity and is regarded as being very rare.

Treatment. Treatment of acute cholecysti-

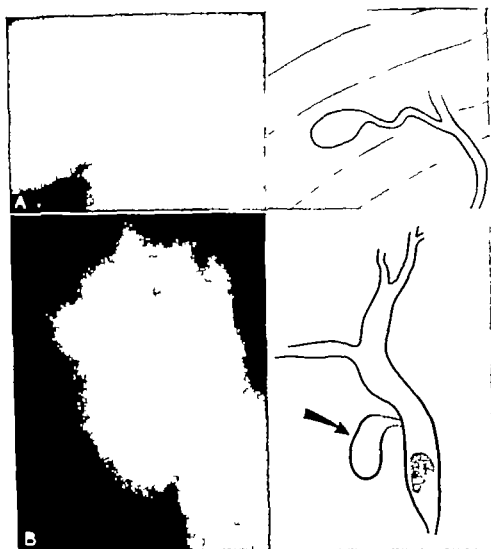


Figure 7 Intravenous cholangiogram. A, Normal biliary tract. B, Stone in common duct and residual cystic duct remnant.

intensity of the jaundice vary greatly. We believe that a stone may be passed through the ampulla of Vater during an "attack of biliary colic." If the stone produces obstruction for a short time, and if there is little infection in the ductal system, there may be no elevation of temperature and no clinical jaundice. At the other extreme, if the stone is large, is not passed and causes complete obstruction, then there is prolongation of the pain with persistent tenderness in the right upper quadrant. If the stone remains engaged and obstruction is complete, the jaundice persists and becomes more intense. If it acts as a ball valve mechanism, then there is a spontaneous subsidence of the symptoms all of which tend to reappear within varying periods and whenever the stone blocks the flow of bile again, as described by Charcot.

The surgical removal of stones from the ductal system by incising the common duct just distal to where the cystic duct joins it

and exploring the intrahepatic tributaries provides our only satisfactory means of treatment. There have been numerous regimens elaborated in an attempt to increase the flow of bile and at the same time produce relaxation of the sphincter of Oddi and then flush the stones into the duodenum. These have been of little benefit. The surgical removal of stones is sometimes difficult because they are easily overlooked in the more inaccessible part of the ductal system. The greatest proportion of common duct stones is found in the distal portion of the choledochus near the ampulla of Vater. They may at the time of operation be located within the hepatic radicles and escape the most exhaustive search. Visualization of the ductal system by filling it with a radiopaque medium, such as Diodrast, and taking x ray pictures (cholangiography) at the time of operation is a distinct aid in locating the elusive stone (Figs. 6 and 7). Recently the introduction of more concentrated contrast

local process and the patient's general condition. Probably surgical treatment during the early phase of cholecystitis, whether it be acute or chronic, is the best means of interrupting biliary tract disease.

CHOLEDOCHOLITHIASIS

If stones in the common duct are looked upon as a complication or sequela of gall stones, then it follows that the longer cholelithiasis has been present the greater is the likelihood of choledocholithiasis. The older the patient is and the longer the disease has existed, the greater may be the expected incidence of common duct stones. If all age groups are included and allowance is made for silent stones not omitting the findings from autopsy studies, it is our opinion that the true incidence of retained common duct stones is about 10 per cent. In other words of all patients with stones in the gallbladder one in ten will also have stones in the common duct. Choledocholithiasis is seldom if ever demonstrated at operation in the presence of a normal appearing gallbladder without stones. The common sequence of events is that stones form in the gallbladder then either pass into the common duct or form there. It is logical to assume that a stone which would pass through the cystic duct from the gallbladder would in turn travel through the common duct and ampulla of Vater. There are wide differences between what is possible and what actually takes place.

The incidence of common duct stone increases as the life span increases. Its occurrence is infrequent in patients with cholelithiasis under forty years of age (less than 6 per cent) and then gradually increases decade after decade so that in those over eighty years of age one-half of the patients with cholelithiasis may be expected to have choledocholithiasis. The number and size of the stones found in the older age group are likewise greater.

Because of the frequent coexistence of gallbladder disease and common duct stones, the presenting symptoms are often those of cholelithiasis. Unless the stones in the duct produce obstruction—complete, partial or intermittent—resulting in biliary colic and jaundice, the symptoms are the same. Furthermore, a mild degree of jaundice (bilirubin of less than 3 mg. per 100 cc. of blood serum) is to be seen as the result of an exacerbation of cholangitis associated with an attack of acute cholecystitis without stones in the common duct. Stones in the common duct which do not cause obstruction and thereby do not result in jaundice may persist unsuspected for years.

The typical manifestations of choledocholithiasis are, in addition to those of cholelithiasis, episodes of jaundice following bouts of biliary colic. Following a period of pain and before jaundice is evident, there may be a chill and elevation of temperature. The duration of the pain, the severity of the chill, the elevation of the temperature and

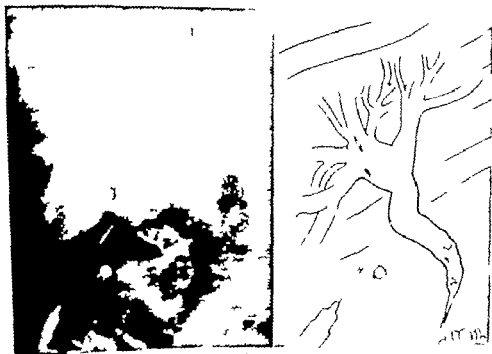


Figure 8. Operative cholangiogram showing stones.

under these circumstances, however does not necessarily indicate that common duct obstruction will result from them. It should also be mentioned that stones may exist in the common duct and yet not produce symptoms. However the fact that a patient is without symptoms of common duct stones following cholecystectomy does not rule out their possible presence.

The patient who has cholelithiasis and choledocholithiasis following cholecystectomy and the removal of stones in the common duct may be completely well. After varying periods, some may develop signs and symptoms of common duct obstruction. At operation a stone or stones are again re-

moved. There then arises the question of whether these formed following the first operation or whether they escaped detection by the surgeon. There is ample evidence that either may occur. Stones may be overlooked and the patient have a typical attack of common duct obstruction within a few days. Re-exploration reveals a stone of good size. If the patient goes for months or years and then exhibits signs of obstruction, one may reason that small stones were overlooked at the first operation and that these gradually increased in size and eventually reproduced the original clinical picture. Then of course, new stones may form and obstruction result. We have no means of



CONTRACTED GALL BLADDER CONTAINING STONES AND ASSOCIATED CHOLEDO- CHOLITHIASIS

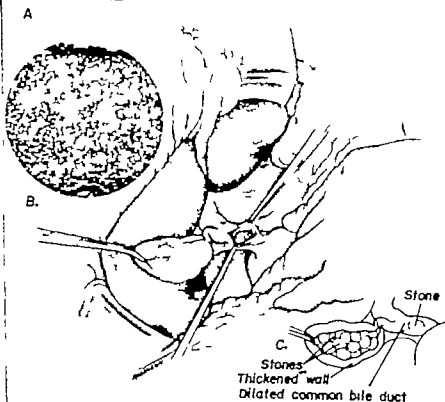


Figure 9 Contracted gallbladder containing gallstones with associated choledocholithiasis. A, Photograph of scarred contracted gallbladder containing calculi. B, Photomicrograph ($\times 20$) showing scar and atrophy of gallbladder wall. C, Line drawing of gallbladder wall thickened and contracted over stones. The common duct is dilated and contains a calculus.

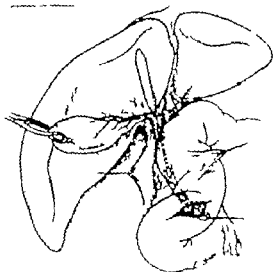


Figure 8. If common duct exploration is to be done at the time of a cholecystectomy the gallbladder will serve a purpose if it is removed downward from the fundus. The cystic artery and the cystic duct are first identified and secured and the gallbladder then is dissected from its bed in the liver. The cystic duct is left intact with a ligature about it just distal to the ampulla of the gallbladder to prevent small stones from passing through the cystic duct into the common duct. After exploration of the common duct, if for any reason a free passage into the duodenum cannot be demonstrated, then transduodenal exposure of the ampulla through a longitudinal incision should follow.

materials, which may be given orally or intravenously for visualization of the biliary tract before operation has been useful in identifying the number, size and location of calculi. Surgeons of experience in biliary tract disease are well aware of the probability of overlooking calculi and have developed various elaborate procedures in an effort to avoid this.

Exploration of the common bile duct and its tributaries for stones is a surgical procedure of sixty years standing. One of the first to report its successful accomplishment

was Kimmell in 1890. It is commonly employed today but the frequency of its use varies greatly from one clinic to another. Some explore the common duct for stones only in the presence of certain indications, whereas others carry out choledochotomy almost routinely in conjunction with cholecystectomy for cholelithiasis (Fig. 8).

A review of the literature on this subject would lead one to assume that there is general agreement as to the indications for exploring the common duct. In fact, many authors list the same criteria. However, a wide difference is found in the frequency with which the duct is opened in quest of stones and in the incidence of calculi recovered. By and large those who report the greatest proportion of explorations also report the highest incidence of stones recovered.

Table 2 tends to lend emphasis to this statement. McKittrick and Wilson, for example, report 100 choledochotomies combined routinely with cholecystectomy for cholelithiasis and show recovery of stones in the common duct in 52 per cent of these subjects. The other extreme is the experience at The New York Hospital-Cornell Medical Center. Of the 4365 patients subjected to either cholecystectomy or cholecystostomy choledochotomy in search of stones was done as well in only 522, or 11.9 per cent. Stones, however, were recovered in 61 per cent of those explored, or 7.3 per cent of the total series.

It is evident that there is considerable variation in the experience of surgeons who explore the common duct. Part of the difference may lie in the fact that when a gallbladder is manipulated at operation, small stones may pass through the cystic duct into the common duct and are then found when the duct is opened. Finding small stones

Table 2. Reported Incidence and Results of Common Duct Exploration Combined with Cholecystectomy

	EXPLORATION			STONES RECOVERED		
	NO. OF CASES	NO.	PER CENT	NO. OF CASES	PER CENT OF KEPT.	PER CENT OF TOTAL
Lacey 1910-1926	619	96	13.8	52	54.0	8.4
1927-1931	653	211	32.3	88	41.7	13.5
Cattell, 1930-1933	495	198	40.2	98	49.4	20.0
1934-1937	634	284	44.8	103	36.2	16.2
1938-1941	909	444	48.8	128	28.7	14.1
1942-1945	1104	504	45.7	186	36.9	16.8
Walzer, 1946	1259	301	24.0	—	50.0	12.0
McKittrick and Wilson, 1949	100	100	100.0	52	52.0	52.0
McLaughlin and Kleeger 1950	230	71	30.8	—	53.8	10.0
Glenn, 1932-1938	895	100	11.2	55	53.0	6.1
1939-1950	2472	259	10.5	179	69.1	7.2
1951-1956	660	154	17.9	78	50.6	9.07

groups of patients. One is made up of patients usually in the older age group who have had no previous operation and who have rather uniform findings at operation as described above. Then there are those who have had, many years previously, a cholecystectomy and/or choledochotomy with relief from their symptoms for varying periods and then have developed jaundice in an interval varying from three months to seventeen years. Operation reveals diffuse or segmental fibrosing stenosis of the biliary ductal system.

The narrowing or obliteration of the ductal system to the extent that jaundice results constitutes a grave situation with poor prognosis. Although stricture may be produced by a primary neoplasm and diffuse inflammation, by far the most common cause

is operative injury. Almost invariably associated with stricture formation and its obstructive jaundice is infection. The two once established seem inseparable and together result in liver destruction which is incompatible with any degree of well being.

The diagnosis of stricture due to operative injury is readily established. The original injury is usually either partial or complete interruption of the continuity of the common duct by actual division or ligation. The sequence of events following operation is therefore important. If the duct has been partially or completely divided and not ligated profuse bile drainage becomes evident within a matter of hours and persists, if unmolested, for weeks or months. If the drainage ceases, jaundice appears. This is

STENOSIS OF COMMON DUCT INDICATING OPERATIVE REPAIR

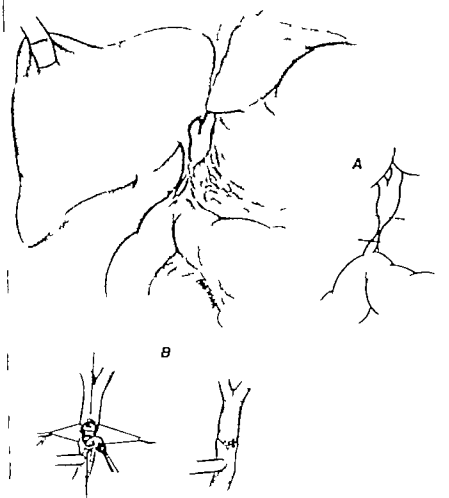


Figure 10. Stenosis of the common duct secondary to operative injury showing enlarged liver due to complete obstruction. A, Schematic outline of structure. B, Method of end-to-end anastomosis with T tube in distal segment.

differentiating between the two. It is of importance however that they be removed whenever they occur regardless of previous operations because they can cause great disability and lead to rapid damage to the liver.

Obstructive jaundice. Jaundice may be a difficult and perplexing problem to both internist and surgeon. Numerous laboratory tests have been developed in an effort to render diagnosis more accurate. There is a vast literature on the subject. Despite this we are frequently unable to arrive at a definite conclusion as to whether or not jaundice is due to common duct obstruction or some other cause.

Jaundice associated with a diminished amount or absence of bile in the intestinal tract may be caused by ductal obstruction or liver cell damage. If it is due to the former then soon after it is established the increased intraductal pressure (to over 375 mm. of water) results in failure of the Kupffer cells to function. Thus there is only a short period when "pure" obstructive jaundice is to be found. In the latter hepatitis cirrhosis or toxic necrosis produces jaundice by eliminating the function of the Kupffer cells. Differentiation between these two is at times most difficult.

There is general agreement that each patient should be evaluated in a meticulous manner. Abnormalities should be sought for in the blood and corrected and the nutrition of the patient should receive immediate and continuous attention. Each step in the management of the patient with jaundice should be well planned and done with deliberation. For those patients who after critical study are felt to have obstructive jaundice operation should be embarked upon when it is believed that all measures needed to bring his or her condition to the optimum for operation have been carried out. Delay based on the hopes that complete common duct obstruction will subside too often results in further liver damage. One of the most important contributions to the management of the jaundiced patient with an increased hemorrhagic tendency has been the introduction of vitamin K. Measured in terms of prothrombin time, the bleeding tendency is recognized in degree. When the liver parenchyma has not been destroyed, the administration of vitamin K corrects the bleeding tendency. The mortality following surgical operations upon jaundiced patients was formerly very high. Since the introduction of vitamin K by Dam and others in 1937 this hazard has been greatly reduced.

An increasing number of patients over sixty years of age are being seen with common duct obstruction due to large gallstones (Fig. 9). Because of long-standing biliary tract disease they also have liver damage. Delay in the relief of obstruction of the common duct is not well tolerated and should be kept at a minimum.

There is a group of patients who defy our diagnostic armamentarium and we are confronted with the question of operating to establish a diagnosis. This may be hazardous because death may follow in the patient who is suffering from severe hepatitis. On the other hand improvement follows the removal of an unsuspected stone producing complete common duct obstruction. No rule of thumb seems practical, rather one must consider each such case individually with care and thoroughness and then do what he believes best.

STRICTURES DUE TO DIFFUSE INFLAMMATION AND INJURY OF THE BILIARY TRACT

Benign, diffuse, stenosing fibrosis of the ductal system is not frequent. However from time to time the condition is encountered at operation and at post-mortem examination. The etiologic basis is not known. The onset is insidious and the development slow until jaundice has become definite and marked. Patients give a history of digestive disturbances similar to and undistinguishable from those due to gallbladder disease. Pain in the early course of the condition may be absent or insignificant. With the onset of jaundice there is usually discomfort in the right upper quadrant and sometimes attacks of mild colic. The gallbladder is not visualized on cholecystography. At operation the wall of the gallbladder and the extra-hepatic bile ducts are found to be thickened and appear edematous. The lumen of the common bile duct may be reduced to less than 1 mm. in diameter and will admit a small probe only with difficulty. The bile may vary from that of normal appearance to a colorless viscid mucoid material. On microscopic examination the liver reveals extensive biliary cirrhosis. Operation accomplishes diagnosis only and the progress of the disease continues until the patient dies because of impaired liver function. It should be emphasized that there is such great variation in the findings at autopsy and the clinical course in the reported cases that one is justified in wondering whether all the cases are due to the same process. There are two

widespread metastases. The next most common type, the papillary adenocarcinoma is not as malignant. This tumor grows more slowly is more bulky and metastasizes late in its very nature, namely that it grows slowly often ulcerates and is associated with infection, occasionally results in its unexpected discovery at an operation for acute or sub-acute cholecystitis. Scirrhous adenocarcinoma of the gallbladder occurs slightly less frequently than does the papillary type. It possesses about the same degree of extension. Mucous adenocarcinoma and squamous cell carcinoma are also found in all reported series of carcinomas of the gallbladder and differ very little in their course.

Treatment. The surgical treatment in recent years provides for radical cholecystec-

tomy including resection of the gallbladder bed. The surgical treatment of nondisseminated carcinoma of the gallbladder in recent years includes the resection of all the liver tissues which comprise the gallbladder bed en bloc with the gallbladder. The portal vein hepatic artery and extrahepatic ductal system are identified and skeletonized with the resection of the peritoneum and retroperitoneal tissue near these structures and the gallbladder. Partial hepatectomy has been done in an attempt to completely extirpate the disease. The reported results are discouraging because at the time operation is undertaken about 60 per cent of the subjects will have spread of the disease beyond the local tumor. Less than half of those who have been considered suitable for surgical

GARCINOMA ARISING FROM EXTRA- HEPATIC BILIARY SYSTEM

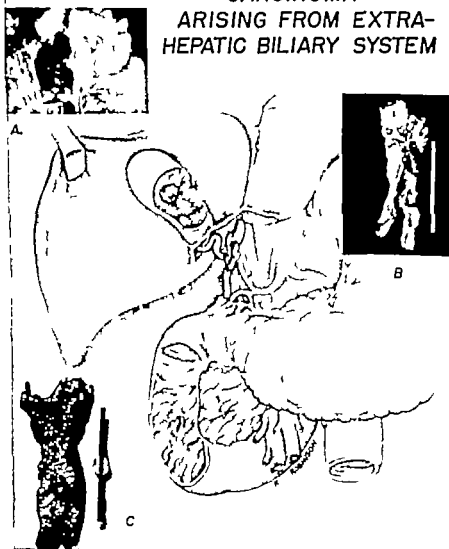


Figure 11. Carcinoma arising from the extrahepatic biliary system. A, Photograph of advanced carcinoma in the gallbladder containing stones. B, Photograph of common bile duct carcinoma invading the portal vein. C, Photograph of carcinoma of the ampulla of Vater.

associated with intermittent and colicky pain accompanied by chills and fever. If the obstruction is complete, the stools are acholic and the liver enlarges. There is anorexia and consequent loss of weight. If the injury at operation immediately resulted in obliteration of the lumen of the duct, as may occur by placing a ligature about the duct, then there follows increasing jaundice with out bile drainage. Of course there may be variations and combinations of these two types of injury. Sometimes a duct obstructed by a ligature may open and discharge bile and a fistula gradually contracts down to complete obstruction. Scar formation and adhesions near the site of injury increase as time goes on. Infection is commonly present and, together with the obstruction, is associated with cholangitis and subsequent liver damage.

Once the diagnosis has been established, operation should be undertaken when the patient has been adequately prepared. The operative correction may be difficult and is not a task for the novice or casual surgeon. If it is feasible resection of the obliterated segment of the common duct and re-establishment of continuity by end-to-end anastomosis, thus preserving a functioning ampulla of Vater as advocated by Cattell, is the ideal procedure (Fig. 10). It is often difficult to locate the distal segment and duodenotomy with retrograde exploration of the duct through the papilla of Vater may be indicated. The duodenum must be mobilized in most instances to enable one to approximate the ends of the duct. Walters and his associates at the Mayo Clinic have reported considerable success with anastomosing the duodenum to the proximal segment of the common duct or even the hepatic ducts in the hilum of the liver. In an attempt to reduce the hazard of regurgitation of intestinal contents into the biliary tract, Cole has popularized the anastomosis of the proximal ductal fragment to the Roux en Y arm of jejunum. Hepaticojejunostomy has been described by Longmire for those patients in whom the proximal ductal system in the hilar area could not be identified. All these procedures leave much to be desired. Many surgeons, including Lahey, Walters, Cattell, and Cole, have made notable contributions to the surgical correction of stricture of the common bile duct due to operative injury. These are commendable. However the best solution to the problems would seem to be their prevention. This is to be accomplished by careful surgery performed

by one who knows the variations of the anatomy of this area and the seriousness of common duct injury.

TUMORS OF THE BILIARY TRACT

The majority of tumors of the gallbladder and bile ducts are malignant. The few benign tumors which are encountered are sometimes difficult to distinguish from inflammatory responses such as hypertrophy of villi and reaction about calcareous material. Papillomas, adenomas and fatty tumors have been observed throughout the biliary system; they are rare, infrequently producing symptoms and occasionally diagnosed incidentally in the course of cholecystography.

The malignant tumors of the extrahepatic biliary tract include those which arise in the gallbladder, extrahepatic ducts and ampulla of Vater (Fig. 11). They are infrequently diagnosed before operation or autopsy. With the exception of those of the ampulla, they are extremely resistant to therapy and are associated with a short period of survival.

Carcinoma of the Gallbladder. Rolleston and McNee cite Maximilian de Stoll, Professor of Medicine in Vienna, as being the first to describe carcinoma of the gallbladder. He reported observations on three post-mortem subjects in 1771, but it was generally considered a rare condition until Graham stated, in 1931, that carcinoma of the gallbladder comprised 8 to 10 per cent of all malignant tumors found in women. Since that report, other studies have been published placing the incidence of this condition at about 4 per cent of all cancers. Carcinoma of the gallbladder accounts for 63 to 75 per cent of all malignant lesions of the extrahepatic biliary tract. The majority of patients with carcinoma of the gallbladder are over fifty-five years of age and more than 90 per cent have gallstones. The symptoms associated with this disease are indistinguishable from those of cholecystitis with cholelithiasis. Actually since the incidence of stones is so high, it is suggested that the symptoms arise from the gallstones rather than from the malignant process. Although there are a few instances of survival of these patients beyond five years, the majority die within a year of the establishment of the diagnosis. Often the diagnosis of carcinoma of the gallbladder is made by the pathologist who examines the gallbladder removed because of stones. Most carcinomas of the gallbladder are of the infiltrating adenocarcinoma type, which rapidly invades all layers of the gallbladder and extends into the liver and produces

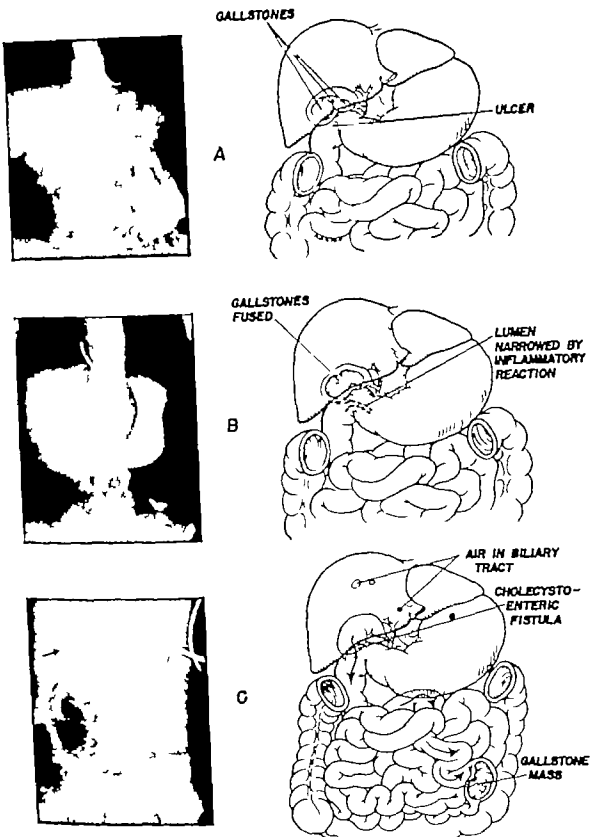


Figure 12. Genesis of gallstone ileus. A, Gastrointestinal series reveals duodenal ulcer and incidental gallstones. B, Five years later gastrointestinal series shows apparent coalescence of previously noted gallstones associated with inflammatory reaction of the pyloric canal resulting in almost complete obstruction. C, Twenty four hours after B and a period of severe abdominal pain followed by marked abdominal distention: there is air in the region of the biliary ductal system, marked distention of the small bowel and a gallstone mass in the left abdomen.

treatment have survived more than one year. There are a few reports of survival of five or more years.

Carcinoma of the Extrahepatic Bile Ducts. Carcinoma of the extrahepatic bile ducts includes tumors arising from the common bile duct, the cystic duct and the ampulla of Vater. Articles on these tumors and case records indicate that difficulty is often encountered in determining the exact site of the tumor's origin. Primary tumors of the pancreas or duodenum may also be confused with those arising from the ampulla of Vater. Reishaw credits Durand Fardell with reporting the first case of cancer of the common duct in 1840.

Carcinoma of the common bile duct and cystic duct is much less common than that of the gallbladder occurring about one fifth as frequently. Half of these are associated with gallstones. Women and men are more equally affected than in carcinoma of the gallbladder in which there is a ratio of 3:1 with predominance in the female. Resection of the tumor with restoration of ductal continuity is indicated. A few successful operations with several years survival of the patient have been reported, but they too are rare.

Carcinoma of the ampulla of the choledochus has a far better prognosis than either that of the gallbladder or of the bile ducts. In our experience and it parallels that of many other clinics patients with carcinoma of the ampulla without spread treated by radical pancreatoduodenectomy have had a much longer survival period than do those with carcinoma of the gallbladder or bile ducts, many going over five years without evidence of recurrence.

Although stones are not found to be associated with carcinoma of the bile ducts as frequently as with carcinoma of the gallbladder, Marshall, Kirschbaum and Stewart have reported their presence in from 21 to 55 per cent of their subjects. This is a higher frequency than is found in nonmalignant disease. The role of stones and their association with carcinoma of the bile ducts is without adequate explanation thus far but a derivative of cholic acid in the bile methyl cholanthrene is known to be a carcinogen and may play a part.

Infiltrating and medullary adenocarcinomas compose the major type of tumors in this group. Leiter found the infiltrating type to be predominant in the common bile duct with papillary adenocarcinoma most frequent in the region of the ampulla of Vater.

The recognition of these tumors at a resectable stage is difficult and depends largely upon a policy of early exploration in patients with mild or transient jaundice and upon a lively suspicion and acute observation of the operating surgeon. Cytologic examination of duodenal content has not been of much aid in preoperative diagnosis. Preoperative intravenous cholangiography may help to localize an incomplete obstruction of the common duct at the ampulla of Vater.

Except in rare instances early diagnosis of these tumors is difficult and so is seldom made. Many tumors are discovered unexpectedly or in a late phase when they have extended and metastasized beyond any possible cure by surgical therapy. Untreated, the life expectancy of the patients after onset of the first symptoms, or from the time the tumor is discovered, is usually measured in months. But as interest in these tumors has widened, their recognition has increased and surgical therapy has been developed, first palliative and later curative.

Palliation has been directed toward relief of jaundice and its associated complications, and toward relief of pain. In carcinoma of the ampulla of Vater pancreatoduodenectomy or similar more extensive radical resection is indicated in all individuals in whom excision of the gross tumor seems initially possible. Although the immediate postoperative mortality and complication rates are high, the palliation provided those patients who survive is long standing and effective. The absolute curability is probably not greatly affected. Resections known to be palliative seem justified in selected subjects.

The prognosis to patients with carcinoma of the gallbladder and extrahepatic biliary ducts is not greatly improved by the adoption of radical surgical procedures. Because of the high incidence of stones in these patients and because so many are over sixty years of age prophylactic cholecystectomy for all patients with symptomatic biliary tract disease may be further justified on the basis of the dangers of malignant change.

GALLSTONE ILEUS

Intestinal obstruction due to gallstones and associated cholecystoduodenal fistulae have in recent years been reported with increasing frequency. McLaughlin collected reports of 366 cases from the literature in 1951. There have been several reports of small series stressing the higher incidence of this type of obstruction in the older age group. This is in keeping with the now rec-

- McLaughlin, C., Jr., and Raines, M.: Obstruction of the Alimentary Tract from Gallstones. *Am. J. Surg.* 81:424 1951
- Nannyn, B. *Klinik der Cholelithiasis*. Leipzig, Verlag von F. C. Vogel, 1892. Translated by Garrod, A. E., New Sydenham Soc. 1896.
- Patterson, H. A.: The Association of Gallstones and Heart Disease. *Ann. Surg.* 139:683, 1951
- Popper, H., and Steigman, F.: Differential Diagnosis between Medical and Surgical Jaundice by Laboratory Tests. *Ann. Int. Med.* 29:469 1948.
- Robertson, H. E.: The Preponderance of Gallstones in Women. *Internat. Abstr. Surg.* 80:1 1945
- Rolleston, H., and McNeer, J. W.: Diseases of the

- Liver Gall Bladder and Bile Ducts. New York, Macmillan Company 1929
- Shorr, E., Zweifach, B. W., and Furchgott, R. F.: On the Occurrence, Sites and Modes of Origin and Destruction of Principles Affecting the Compensatory Vascular Mechanisms in Experimental Shock. *Science* 102:189 1945
- Stewart, H. J. *Cardiac Therapy*. New York, Paul B Hoeber 1952, p. 368.
- Walters, W.: Physiologic Studies in Cases of Stricture of the Common Bile Duct. A 28 Year Survey with Data on 254 Patients. *Ann. Surg.* 138:609 1953.
- Winkle, A. L.: Bacteria as a Cause of Cholecystitis. *Brit. J. Surg.* 15:450, 1927-28.

Diseases of the Liver

By ARTHUR H BLAKEMORE M D and
ARTHUR B VOORHEES JR M D

ARTHUR HENDLEY BLAKEMORE is a Virginian. Able men inherit additional distinction when born in certain states of the union. Virginians and Texans will dispute the inclusion of other states in that statement. The College of William and Mary and Johns Hopkins University were the sources of his education. He has led the revival of interest in operations for the relief of portal hypertension and the use of substitutes for resected, diseased blood vessels.

ARTHUR BOSTWICK VOORHEES, JR., was educated at the University of Virginia and trained to be a surgeon at Columbia University College of Physicians and Surgeons, and Presbyterian Hospital, New York. He has been active in surgical research in problems of vascular surgery and portal hypertension.

PORTAL HYPERTENSION

The term portal hypertension can be applied to any state in which the blood pressure within the portal venous bed is above the normal range. There is no strict agreement as to the precise range of normal pressure or as to the best method of measuring the pressure. The clinician circumvents these uncertainties by basing his diagnosis on anatomic deformities known to be associated with portal hypertension. The surgeon is frequently in a position to validate the diagnosis by comparing direct portal pressure measurements obtained by a uniform technique performed before and after an operation designed to correct portal hypertension. Generally speaking, manometric pressures are considered normal if below 150 mm. of water and abnormal if above 200 mm.

of water when the right atrium is the zero point.

Etiology Portal hypertension may be transient or constant, related to a definite pathologic state or part of a normal physiologic response. The major causes are

1. Physiologic
 - a. Hormonal (response to histamine epinephrine)
 - b. Increased intrathoracic pressure (response to Valsalva maneuver)
 - c. Neurogenic (hepatic plexus stimulation)
2. Pathologic
 - a. Reversible
 - Congestive heart failure
 - Bronchial asthma
 - Pericardial effusions

ognized higher incidence of acute cholecystitis in the aged and the tendency to develop complications without impressive clinical manifestations.

In the majority of instances there is perforation of the gallbladder with the discharge of a stone or stones into the lumen of an adherent adjacent viscus usually the intestine. If the stone is too large to pass through the intestinal tract it becomes lodged in the area where the lumen is smallest and produces a typical acute intestinal obstruction. Because the lumen of the ileum is the narrowest, it is a frequent site for obstruction. Stones perforating into the stomach may be vomited and those into the large bowel are passed in the feces.

The common site for perforation is into the first or second part of the duodenum, leaving a residual fistula behind. If the stone obstructs the small bowel in its terminal portion a triad of findings is to be observed (Fig. 12): distended small bowel, a gall stone in the small intestine usually in the terminal ileum, and air in the biliary tract.

Intestinal obstruction requires immediate surgical evaluation and, in most instances, operation. Intestinal obstruction due to a gallstone requires the same type of management. The commonly associated high mortality which has been reported should be reduced by proper supportive measures followed by operation. Because the diagnosis is incorrect in so many of these patients the condition needs greater consideration in differential diagnosis of intestinal obstruction. If one keeps the triad, small bowel obstruction, gallstone within the small bowel and air in the biliary tract, in mind when seeing patients, particularly in the older age group, with intestinal obstruction the diagnosis may more frequently be made.

Since it is rare for a stone which is large enough to produce intestinal obstruction to pass from the common duct through the ampulla of Vater it should be assumed that a cholecystointestinal fistula exists. At a later operation cholecystectomy and closure of the fistula should be done.

READING REFERENCES

- Andrews, E., Schoonheimer, R., and Hrdina, L. Etiology of Gallstones: Chemical Factors and Role of Gall Bladder. *Arch. Surg.* 35:798, 1932.
- Best, R. R. The Acute Gall Bladder. *Surg. Gynec. & Obst.* 73:312, 1941.
- Bisgard, J. D. and Baker, C. P., Studies Relating to Pathogenesis of Cholecystitis and Cholelithiasis and Acute Pancreatitis. *Ann. Surg.* 113:1006, 1940.
- Cattell, R., and Warren, A. W. *Surgery of the Biliary Tract.* New England J. Med. 255:698, 1956.
- Charcot, J. M. *Leçons sur les Maladies du Foie et des Vésicules Biliaires et des Reins.* Paris, 1877. Les. 12 et seq. 125.
- Clifford, W. J. Acute Gangrenous Cholecystitis. *New England J. Med.* 241:640, 1949.
- Cole, W. H. Recent Trends in Gall Bladder Surgery. *J.A.M.A.* 150:631, 1952.
- Cole, W. H. Precautions in the Treatment of Structures of the Common Duct. *Am. Surg.* 20:354, 1954.
- Cushing, H. Typhoidal Cholecystitis and Cholelithiasis. *Bull. Johns Hopkins Hosp.* 9:91, 1906.
- Dann, H. The Antihemorrhagic Vitamin of the Chick: Occurrence and Chemical Nature. *Nature* 155:852, 1935.
- Doubilet, H., Reed, C. and Mulholland, J. H. Delayed Operative Management of Acute Cholecystitis. *J.A.M.A.* 155:1590, 1954.
- Dumphy, J. E., and Ross, F. P. Stitches in Acute Cholecystitis. I. Surgical Management and Results. *Surgery* 25:539, 1949.
- Elias, M. H. Morphology of the Liver in Liver Injury. *Transactions of the Tenth Conference*, edited by F. W. Hoffbauer. New York, Josiah Macy Jr. Foundation, 1951. p. 111.
- Fallis, L. S., and McClure, R. D. Acute Cholecystitis. A Review of 320 Cases. *Surg. Gynec. & Obst.* 70:1022, 1940.
- Glenn, F. Surgical Treatment of Acute Cholecystitis. *Surg. Gynec. & Obst.* 90:843, 1950.
- Glenn, F. Common Duct Exploration for Stones. *Surg. Gynec. & Obst.* 95:431, 1952.
- Glenn, F., and Hays, D. M. The Causes of Death Following Biliary Tract Surgery for Nongallstone Disease. *Surg. Gynec. & Obst.* 94:283, 1952.
- Glenn, F. and Hays, D. M. The Scope of Radical Surgery in the Treatment of Malignant Tumors of the Extrahepatic Biliary Tract. *Surg. Gynec. & Obst.* 69:329, 1954.
- Graham, E. A. The Prevention of Carcinoma of the Gallbladder. *Ann. Surg.* 93:371, 1931.
- Graham, E. A., and Cole, W. H. Roentgenologic Examination of Gall Bladder: Preliminary Report of New Method Utilizing Intravenous Injection of Tetrabromophenolphthalein. *J.A.M.A.* 82:813, 1944.
- Graham, E. A., Cole, W. H., Copher, C. H., and Moore, S. Diseases of Gall Bladder and Bile Ducts. Philadelphia, Lea & Febiger, 1928.
- Gross, R. E. The Surgery of Infancy and Childhood. Philadelphia, W. B. Saunders Company, 1953.
- Holmes, J. B. Congenital Obliteration of the Bile Ducts: Diagnosis and Suggestions for Treatment. *Am. J. Dis. Child.* 11:405, 1918.
- Kirshbaum, J. D., and Kozell, D. D. Carcinoma of the Gallbladder and Extrahepatic Bile Ducts: A Clinical and Pathological Study of 117 Cases in 13,300 Necropsies. *Surg. Gynec. & Obst.* 73:740, 1941.
- Kummell, H. Zur Chirurgie der Gallenblase. *Deutsche med. Wochenschr.* 16:237, 1890.
- Leiter, H. E. Carcinoma Involving the Common Bile Duct, A Report of Four Cases of Successful Resection. *Surgery* 29:627, 1947.
- Lichtenman, S. S. Diseases of the Liver, Gall Bladder and Bile Ducts. Philadelphia, Lea & Febiger, 1951.

regenerating hepatic cell of a blood supply by reducing portal flow and shunting arterial flow about the regenerating nodule which lead to further hepatic cell anoxia and death. This concept of the progression of liver damage is a possible explanation of the progressive pattern of liver damage often exhibited by individuals with cirrhosis long after the primary cause of hepatic cell death has disappeared.

Much of our present-day therapy of cirrhosis is based on efforts to improve oxygenation of the liver cell by correction of anemia, to control hemorrhage and to reduce demands upon the liver through bed rest and control of intercurrent disease.

The next most common disease process that produces portal hypertension is occlusion of the portal vein prior to its entry into the liver the so-called extrahepatic block. Although it accounts for perhaps only one of five cases in any over all group it is the most common cause for portal hypertension in children. The most common causes of extrahepatic block are cavernomatous transformation of the portal vein, spontaneous thrombosis associated with polycythemia vera, periphlebitis and thrombosis associated with omphalitis of the neonatal period, congenital atresia, inflammatory or neoplastic extrinsic pressure. The clinical manifestation of the venous block is similar to that of the intrahepatic block, i.e., dilatation of the collateral venous circuit, splenomegaly and occasional ascites however there is absence of liver damage.

The least common disease process that produces portal hypertension is occlusion of the outflow tract associated with constrictive pericarditis, inferior vena cava thrombosis and hepatic vein thrombosis (Chiari's disease). The first two levels of obstruction can be suspected if in addition to portal hypertension, there is a coincident significant lower extremity venous pressure elevation. In the case of the latter if there is evidence of rapidly developing portal hypertension and sudden tender enlargement of the liver associated with progressive failure, Chiari's syndrome must be considered.

Clinical Picture. In the early stages of portal hypertension the patient is often unaware of any abnormality but occasionally he may note upper abdominal fullness associated with enlargement of the spleen prominence of the anterior abdominal wall veins, particularly in the periumbilical region. More often than not, any symptoms that the patient may experience are over

shadowed by symptoms related to the basic process which, as in the case of cirrhosis may be weight loss anorexia, fatigability and vague digestive difficulties. Even this is not constant and often the patient's presenting symptom is massive gastrointestinal hemorrhage.

Hints as to the etiology and duration of the disease process can occasionally be obtained from the history however more often than not these remain obscure. Nevertheless, it is of importance to establish, if possible, a history of chronic dietary inadequacies, alcoholism, infectious hepatitis, homologous serum jaundice previous biliary tract disease or operation or contact with hepatotoxins. A previous history of jaundice or long standing splenomegaly with or without evidence of liver disease, is important. Two of the most common coincident diseases peptic ulceration and diabetes mellitus must be excluded by historical and laboratory evaluation.

On physical examination the findings that are of importance in establishing the diagnosis are jaundice, spider angiomas, abdominal wall collateral circulation ascites, peripheral dependent edema and hepatosplenomegaly.

The immediate status of the individual can be rapidly assessed by the presence or absence of active bleeding and of shock. The extent of the underlying disease if this is cirrhosis can be roughly judged by the presence or absence of jaundice, new "crops" of spider angiomas soft tissue wasting, ascites and, of most importance liver tenderness.

Special and laboratory examinations are of great importance again in confirming the diagnosis of hypertension and in completing the etiologic picture. A thick barium swallow may demonstrate radiographically esophageal and gastric varices in perhaps seven out of ten individuals examined. Two of the remaining three will require esophagoscopy to demonstrate varices, and in the tenth and last individual varices will not be demonstrated.

Laboratory studies which reflect liver function are of major importance in determining the site and most likely cause of the block and in estimating, as in the case of cirrhosis, the degree of liver damage and, therefore, indirectly the operative risk.

Differentiation between hepatic vein thrombosis (Chiari's syndrome) and intrahepatic venous block is difficult and depends on the nature of onset. The former evolves in a matter of days and is accompanied by

b Irreversible

- Constrictive pericarditis
- Inferior vena cava obstruction
- Hepatic vein obstruction
- Intrahepatic portal obstruction
- Extrahepatic portal obstruction
- Extrahepatic portal arteriovenous fistula

Pathogenesis of Portal Hypertension The surgeon is infrequently called to see a patient with hypertension arising from a normal physiologic response or from a completely reversible pathologic state. On the other hand, portal hypertension arising from an irreversible pathologic state is being brought more often to the attention of the surgeon because of his increasing capacity to correct some of its causes or results.

The anatomy of the blood supply to the liver is unique in that there are two afferent systems and one efferent system. As a result of the dual inflow tract, circulatory studies, summarized by Child, are unusually difficult to make and to interpret. Despite the complexities there is fair agreement of the estimates that approximately one-fourth of the minute cardiac output passes through the liver. One-third of this flow is delivered by the hepatic artery and two-thirds by the portal vein. Although portal vein blood has a lower oxygen content than hepatic artery blood, it does have an oxygen content higher than peripheral venous blood. This fact, coupled with the greater minute flow in the portal vein makes the afferent venous system a significant source of oxygen which accounts for more than half of the oxygen supplied to the liver. If it were not for the numerous communicating channels within the substance of the liver between the two afferent systems and a more or less reciprocal flow relation between the two systems during periods of normal or abnormal stimulus, then intrahepatic or extrahepatic obstruction in the portal vein system would be of greater significance than it is. Whether permanent or temporary partial or complete, occlusion of either portal vein or hepatic artery will usually lead to an increase in flow through the other vessel. However this reciprocal relationship is usually insufficient to sustain complete integrity of the liver if either artery or vein is suddenly occluded. Any obstruction in the inflow or outflow tracts of the liver will lead to an elevated pressure within the afferent system. This seems to be of little consequence in the case of the hepatic artery however in the

portal system the changes are striking. When portal hypertension has developed gradually it is characteristic to find splenomegaly dilatation of the portal vein and of the existing collateral circuits between the coronary and azygos veins, between the falciform ligament and anterior abdominal wall between portal radicles retroperitoneal veins of Retzius and hemorrhoids. On the other hand, when sudden occlusion by thrombosis or by trauma has occurred in the hepatic vein or portal vein, engorgement of the entire portal vein or hepatic vein drainage bed is the outstanding feature in the early phase of the pathologic process (Popper). If the patient survives long enough, the collateral system will become significantly dilated.

The most common and clinically significant disease process that produces portal hypertension is cirrhosis biliary or portal. In this instance, the obstruction to afferent portal flow occurs within the substance of the liver. At present, it is generally believed that hypertension is due to deformed intrahepatic vascular anatomy and that this deformity is caused by accelerated hepatic cell death followed by irregular and eccentric nodular regeneration of the parenchyma. The expanding regenerative parenchymal nodules of the cirrhotic liver obliterate or compress sinusoids and portal vein radicles. Other sinusoids following death of liver cell cords, dilate and bring about ease of communication between hepatic arterioles and portal branches. After death of portions of a lobule and eccentric regeneration, the central (hepatic) vein comes to be situated near the periphery of the parenchymal nodule. Portal venous and hepatic arterial blood passing directly to this hepatic (central) vein thus by passes much of the parenchyma in the central part of the nodule. The over all blood volume within the vessels of the liver diminishes. These vascular changes, summarized by Popper are important in two respects because they set the stage for portal hypertension and for diversion of the blood supply from the regenerating hepatic cell.

Portal pressure elevation probably arises from three major factors: (1) the diminution in number of vessels assigned to carry a definite amount of blood, (2) the increase in resistance to flow in the remaining tortuous vessels (3) the presence of small, but presumably definite, arteriovenous fistulae between the hepatic arterioles and portal venules. The vascular deformity deprives the

graphically small varices whereas large and extensive varices may persist for years with out incidence of rupture. On the other hand, the incidence of subsequent hemorrhage from varices following an initial episode is very high suggesting that there are other factors still unknown that predispose the patient to repeated hemorrhage. It seems im probable that these factors can be assigned to chance alone. Occasionally bleeding may occur following violent intra abdominal or intrathoracic pressure changes associated with vomiting or coughing, but the cause is not usually so simple.

Bleeding may be the first indication of the presence of portal hypertension and the presenting symptom that brings the patient to the physician. The ultimate outcome of the hemorrhagic episode is frequently determined by early therapeutic measures. There is debate as to the therapeutic steps to take in the face of acute massive hemorrhage. Balloon tamponade of the esophageal varices and whole blood replacement are the important steps. Balloon tamponade techniques vary but take two general forms (1) traction tamponade by a large inflated gastric balloon attached to a double-lumen tube snugged up into the cardia, thereby compressing the collateral vessels feeding the esophageal varices (2) compression tamponade by a triple-lumen double-balloon tube which compresses the esophageal varices by inflation of the esophageal balloon. The first technique will control most hemorrhages as long as traction can be maintained and tolerated by the patient, but in most instances this technique must be supplemented by early ligation of the varices through transthoracic esophagotomy, or through transabdominal gastrotomy. With the second technique, most hemorrhages can be controlled by compression of the esophageal varices by the balloon alone. If bleeding continues, this is indicative of a gastric varix or some other source of bleeding. In these rare cases, gastrotomy and ligation of the bleeding point are indicated. Of the two techniques, esophageal balloon tamponade is favored since it usually averts operative intervention at a time when operative stress can be seriously detrimental to the liver. This argument, of course does not hold in the instance of portal hypertension secondary to a block in the portal vein, since in this instance the liver is normal. On the other hand, in practice, the esophageal balloon will control the hemorrhage and operation can be avoided. This may be of particu

lar importance to a toddler who may have several years to achieve sufficient growth prior to a definitive shunting procedure. Esophagotomy and suture of varices afford only temporary protection from bleeding. This limited period of protection may be sufficient to prepare a poor risk cirrhotic patient for operation but it is insufficient to protect a toddler where years may be needed before a splenorenal shunt can be performed.

Constructive approaches to the long term control of varices have taken two general forms (1) decompression of the portal hypertension by a portal vein to vena cava or splenic vein to renal vein shunt (Fig. 14) (2) obliteration by interruption of the blood supply to the varices by ligating the varices. The latter has limited value since vascular connections are re-established usually within a relatively brief period of months. Partial esophagogastrectomy and a jejunal limb interposition enjoys a greater success but is limited in that associated postoperative nutritional problems may make it inadvisable in the individual with cirrhosis. This is a point yet to be established.

At this time the portacaval shunt in either the form of a splenorenal or a portal vein to vena cava shunt is generally accepted as the best available method of reducing portal hypertension. The portacaval shunt was introduced experimentally over seventy years ago but only since 1943 has it been per

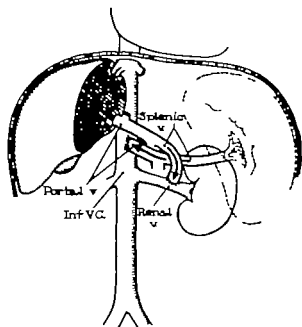


Figure 14 A drawing portraying two commonly used portal to systemic venous shunts. Note the acute angle the portal vein makes with the vena cava to increase the efficiency of this shunt.

Table 3 Differentiation between Intrahepatic and Extrahepatic Block

	INTRAHEPATIC BLOCK (CIRRHOSIS)	EXTRAHEPATIC BLOCK
Esophageal varices	Present	Present
Hepatomegaly	Usually present	Absent
Splenomegaly	Usually present	Present
Ascites	May be present	Rare
Peripheral edema	May be present	Absent
Thrombocytopenia	Usually present	Present
Leukopenia	May be present	Present
Anemia	Usually present	Absent
Jaundice	Usually absent	Absent
Liver function	Impaired	Not impaired

abdominal pain, progressive enlargement and tenderness of the liver, rapid production of ascites and, finally, varices with perhaps hemorrhage. On the other hand, in cirrhosis the progression of symptoms although similar is stretched out over months or years.

The differentiation between the extrahepatic and intrahepatic block is usually difficult (Table 3).

As previously mentioned, the presenting symptom of portal hypertension may be upper gastrointestinal hemorrhage frequently severe. Practically speaking, if this occurs in a child with splenomegaly the statistics overwhelmingly favor a diagnosis of bleeding esophageal varices. Unfortunately if little is known medically about the child prior to hemorrhage, splenomegaly may go undetected during the first few hours of hospitalization until blood volume is restored and the spleen re-expands to its pre-hemorrhagic size. In the adult, early differential diagnosis may be very difficult, but it is of great importance to attempt to make a differentiation since the two most common causes of massive hemorrhage, peptic ulcer and esophageal varices, require different therapeutic approaches. To complicate the differential diagnosis, it has been found that perhaps one out of every five adult individuals with cirrhosis and varices also has a peptic ulcer and bleeding can occur from either. It is strongly urged that an esophageal balloon (Fig. 13) be passed as a diagnostic procedure during an acute hemorrhage in any individual in whom there is the slightest suspicion of underlying liver disease even if there is a coincident history suggestive of peptic ulcer. If the balloon brings the hemorrhage under prompt control, the bleeding is probably from esophageal varices. If on the other hand, bleeding is not brought under control, the source is usually gastric varices

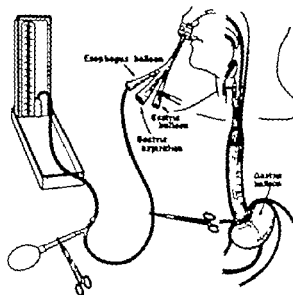


Figure 13. Diagram of triple lumen double-balloon tube used to tamponade esophageal varices. Note the method of connecting with a blood pressure manometer.

or an ulcer. In this latter instance, assuming that the individual is suspected of having cirrhosis, early gastrotomy is indicated.

Rarely but sufficiently often to warrant mention, massive upper gastrointestinal bleeding can occur from hemorrhagic gastritis which may be an isolated phenomenon or part of a generalized bleeding diathesis associated with a terminal phase of cirrhosis.

Commonly we associate varices with massive sudden hemorrhage; however they can seep blood slowly over periods of days or weeks and in these instances gastric neoplasms must be considered in the differential diagnoses.

Finally but rarely massive bleeding from a hiatus hernia must be differentiated from a bleeding esophageal varix. If bleeding occurs in the presence of both, differentiation and control come only by gastrotomy. Balloon tamponade is not effective in the presence of a hiatus hernia, in fact, it is contraindicated since it may impede blood flow in the midportion of the esophagus and fail to compress varices in the lower portion of the esophagus.

Treatment. Bleeding from esophageal varices is without predictability and may occur at any time during the disease process. There is no definite correlation between the cause of the hypertension and the time of onset, frequency or severity of bleeding. Neither does the size of the varices correlate with any of these. For example, massive fatal hemorrhage may occur from radio-

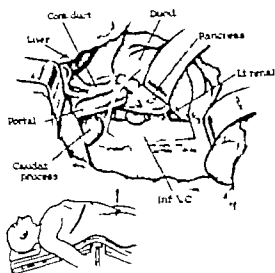


Figure 16. Anatomic relation of the portal vein to the vena cava as seen through a right thoracoabdominal incision. (From Madden: *Atlas of Techniques in Surgery* 1958. New York, Appleton-Century-Crofts, Inc.)

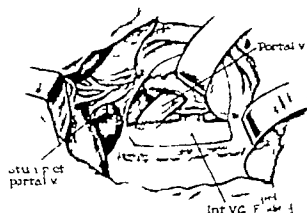


Figure 17. Complete end-to-side portal vein to vena cava shunt. (From Madden: *Atlas of Techniques in Surgery* 1958. New York, Appleton-Century-Crofts, Inc.)

slowly over a one- to two-year period and that the procedure can be performed only in cases of prehepatic or intrahepatic obstruction. Furthermore, it has the theoretical disadvantage of disrupting part of the afferent flow into the liver.

Prognosis. Preparing an accurate statistical evaluation of the natural history of portal hypertension is a virtual impossibility. It is a rare condition and patients who reach the larger medical centers where statistical evaluation is possible have already undergone a certain amount of selection. It is a condition that is usually superimposed on a disease, cirrhosis of the liver which exerts a dominant force on the evaluation of any therapeutic measure for portal hypertension. For example, portal hypertension may occur at any time in the cirrhotic process and may be associated with good or bad liver function, esophageal varices may appear at any time after portal hypertension has developed, and, finally bleeding from varices can occur at any time after they become demonstrable. An obvious need for endless qualifications of every statistic has been recognized by the students in this field. In order to avoid broad comparisons of disease modifications which are, at best, laborious and frequently impossible, these statistical qualifications are seemingly ignored for a more simplified comparison of whether this or that form of tamponage stops acute hemorrhage, or this or that operation stops episodes of subsequent bleeding. Granting their inaccuracies, reported statistics do give some idea of magnitude. It is

estimated that if supportive therapy alone is available, nearly all cases of extrahepatic and one-half of all cases of intrahepatic portal block will survive the initial major hemorrhage from varices. With the addition of esophageal tamponage three-quarters of the intrahepatic group will survive the initial hemorrhage. Without additional treatment, one-half of the patients who survive the initial hemorrhage will live five years or more without additional hemorrhage. Additional therapy in the form of a technically satisfactory end-to-side portal vein to vena cava shunt will result in only one in forty patients dying as a result of operation and one in twenty having a subsequent hemorrhage. These recent statistics represent a marked improvement over the past, but five years will be needed to determine alteration in longevity.

TUMORS OF THE LIVER

Differential diagnosis of benign, primary malignant and metastatic tumors of the liver is usually resolved only by biopsy. Clinical manifestations are not characteristic and include increasing anemia, sensation of fullness in the hypochondrium, weight loss and occasionally fever when a rapidly growing neoplasm undergoes central ischemic necrosis. Late stigmata of progressive invasion and compression of bile ducts and blood vessels include obstructive jaundice and signs of progressive cirrhosis with enlarging liver ascites and abdominal pain.

Benign Tumors. Benign tumors of the liver are generally pathologic curiosities rarely diagnosed, and are usually encountered at autopsy or operation for other disease. Lipoma, fibroma, adenoma, hamartoma

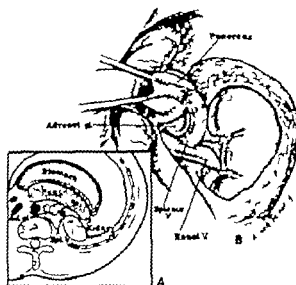


Figure 15 A completed end-to-side splenoportal shunt. (From Madden Atlas of Techniques in Surgery 1958 New York, Appleton-Century-Crofts Inc.)

formed in the human with any significant frequency. Since then the operative technique and the preoperative and postoperative management of the patient have undergone considerable refinement and deserve brief summaries.

Patient selection Recommendation for operation depends on evidence of portal hypertension (varices, splenomegaly, abdominal wall collateral circulation, ascites), a history of active upper gastrointestinal tract bleeding when causes other than varices have been excluded, and a demonstration on the part of the patient that liver function can be improved with a conservative medical regimen. In the latter point, some elaboration is indicated. In the past, considerable importance has been placed on tests of liver function as a basis for patient selection. Although this is still valid to a degree, selection on the basis of an individual's capacity to improve liver function during a period of conservative medical therapy seems to be of greater value and serves as a method of estimating reserve liver capacity. If a patient shows continued deterioration of liver function despite conservative therapy, the end is near and operation will only serve to hasten it. A word of caution should be sounded, however, in any instance following a period of massive gastrointestinal tract hemorrhage, liver function studies can be noted to be progressively depressed despite a good medical regimen. This depression, frequently transient, discourages any emergency operation. It clears in three to four weeks after the hemorrhage and does not carry the same

ominous note as a similar pattern of deterioration in a patient who has not recently bled.

The preoperative and postoperative care of these patients is essentially the same as in the accepted medical regimen of cirrhosis, i.e., bed rest, nutritious diet rich in calories, proteins and vitamins, correction of ascites, if present, by salt restriction, diuretics and paracentesis and correction of anemia by whole blood transfusion. Recently by virtue of recognition the problem of "ammonia intoxication" has been seen more often. Occasionally this is evident preoperatively but more frequently postoperatively. The syndrome of ammonia intoxication is presently believed to be a result of an abnormal accumulation of ammonia in the body occurring when the liver is unable to utilize ammonia in the ornithine cycle, when there is shunting of portal blood around the liver and, finally, when there is greater production of ammonia from high protein diets. Usually transient, occasionally chronic, the syndrome can be controlled by reducing the protein in the diet or by reducing the capacity of bacteria to produce ammonia. Of the two methods, the latter is preferable and can be accomplished by giving small amounts of neomycin and bacitracin orally.

Surgical procedure Practically speaking, the portal and splenic veins are the only two veins in the portal system of sufficient size to warrant use in a shunting procedure. Rarely the superior mesenteric can be used. The use of each vein has certain individual advantages and disadvantages. In splenoportal shunt (Fig. 15) in which the splenic vein is anastomosed end to side to the renal vein, the actual and theoretical advantages are immediate control of hypersplenism by virtue of splenectomy and less distortion of the afferent blood flow to the liver. The disadvantages are that a technically satisfactory shunt can be difficult to achieve. Portal pressure reductions are usually not as great as those achieved by the portal vein to vena cava shunts and follow-up studies would suggest a higher incidence of closure of the shunt. In portal vein to vena cava shunt (Figs. 16 and 17) in which the portal vein is anastomosed end to side to the vena cava, the actual advantages are usually satisfactory reductions in portal pressure by a shunt which is technically easier to achieve and an extremely small incidence of shunt closure as suggested by follow-up studies. The disadvantages are that hypersplenism is not improved immediately but improves

will arouse the suspicion of the surgeon but a definite diagnosis cannot be made unless biopsy of the liver is performed during surgical exploration or through a needle. The needle biopsy technique may obviate the necessity of a surgical exploration if the specimen is positive for tumor cells. However a negative report does not exclude the possibility of metastatic liver disease.

The clinical significance of suspected metastatic disease in the liver on physical examination, or of documented disease in the liver on biopsy is that it presents, in our present state of cancer therapy, a point of no return as far as curative resection of the primary lesion is concerned. Even though this represents a convenient clinical rule the occasional patient in whom a solitary neoplastic focus or local direct invasion can be removed in an en bloc resection should not be overlooked.

One of the most striking features of metastatic liver disease is the frequent advanced degree of liver destruction unassociated with real clinical evidence to suggest liver function impairment.

PYOGENIC ABSCESS OF THE LIVER

The advent and subsequent widespread use of antibiotics have decreased the incidence of pyogenic abscess of the liver. This now rare and subtle disease process however should remain an important diagnostic consideration for the clinician can often prevent an otherwise fatal outcome by early recognition and treatment.

Pathogenesis. Frequently bacteria reach the liver via the portal vein, the hepatic artery and the lymphatic system. In the normal individual, these microorganisms are destroyed by the liver. Occasionally however this protective mechanism may be impaired by primary liver disease or by overwhelming generalized septicemia, which can disrupt the favorable balance normally existing between pathogenic bacteria and host resistance. This imbalance can be directly reflected in the formation of solitary or more commonly multiple liver abscesses.

Although liver abscesses can follow septicemia from any primary focus they are most commonly a sequela of primary intra-peritoneal infections, particularly those which have venous drainage via the portal system. Direct extension into the liver can occur from suppurative disease in the gall bladder and in the subphrenic space and from peptic ulcer penetration. The sequence of acute appendicitis with cellulitis of the

mesoappendix and mesentery pylephlebitis and septic emboli to the liver is the recognized common primary mechanism however in over half of the collected cases there has been no recognized primary focus of infection.

Liver abscesses secondary to infection arising primarily from the gastrointestinal tract will usually yield cultures of *Escherichia coli* nonhemolytic streptococcus or *Clostridium perfringens*. Those arising from primary infection elsewhere will usually yield cultures of *Staphylococcus pyogenes* var *aureus* or hemolytic streptococcus. Many other organisms have on rare occasion been found in liver abscesses they include gonococci, spirochetes, *Klebsiella pneumoniae* and *Salmonella typhi*.

Symptoms. The symptoms of pyogenic abscess of the liver are usually subtle in the beginning, develop insidiously and can become masked by the symptoms arising from the primary focus of infection. When masking has not occurred, malaise, anorexia and low grade fever associated with vague right upper quadrant pain may be the only suggestive symptoms at the onset. With progression of the disease, the syndrome will change to a general toxicity reaction of spiking temperature, in the 105° to 106° range associated with shaking chills, profuse sweating and the local manifestations of severe right upper quadrant pain with frequent radiation to the right shoulder and hypochondrium.

Local signs depend in part upon which portion of the liver has undergone abscess formation. Direct shock tenderness over the lower rib cage and tenderness by palpation of an enlarged liver are usually constant findings. A variable amount of clinical and laboratory icterus may be present, progressive jaundice is a poor prognostic sign. Increase in the number of leukocytes to 20,000 to 25,000 with a predominant polymorphonuclear shift, is common and can be used as a differential point in ruling out the possibility of an amebic abscess. Elevation of the right diaphragm, as seen on fluoroscopic examination, is an occasional finding in abscesses of the right lobe. Also associated with right lobe expansion can be compression of the inferior vena cava, measurable as an increase in femoral vein pressure.

Treatment. The principles of therapy are the same as those of therapy of pyogenic abscess elsewhere. Incision and drainage must be first accomplished. An appropriate antibiotic proved to be effective against the

and myoma have been reported, but the most common is cavernous hemangioma. Varying greatly in size, these tumors usually present beneath the liver capsule as purple, raised, multinodular compressible masses. Except for large hemangiomas which may be ruptured by trauma, excision is usually unnecessary since progressive enlargement is the exception.

Malignant Tumors. Primary carcinomas of the liver are rare have a geographical incidence which parallels that of cirrhosis of the liver and comprise up to 0.3 per cent of all autopsy findings and up to 2.5 per cent of all cancers. Up to 1835 approximately 2500 histologically proved cases had been recorded in the world literature. Two peak periods of age incidence occur in infancy and childhood and in the fifth and sixth decades, the tumors in the former period frequently being of the embryonal type with organoid features and those in the latter of the parenchymal hepatic cell type. A third histologic type is derived from intrahepatic bile duct cells and is occasionally associated with hepatoma cells as a mixed tumor. Predominance of involvement of males and of patients with cirrhosis has been well documented.

In the Department of Surgical Pathology at Columbia Presbyterian Medical Center in New York, twenty-one patients with proved primary liver carcinoma were seen from 1930 to 1955. In the Babies Hospital in New York, from 1922 to 1955 seven patients were observed.

The etiologic basis of liver carcinoma is at present unknown, but the relationship to cirrhosis with fibrosis and regeneration of new liver cells, is receiving special attention. The gross pathologic process varies from single large irregular and nodular masses to multicentric nodules involving the entire liver. A third, less common, sclerous form has also been observed. The tumors are usually deep seated and do not present the umbilicated superficial masses seen with metastatic lesions to the liver.

Clinically there is little to support differential diagnosis between cell types though liver cell carcinoma is generally the most rapidly progressive. Extrahepatic metastasis occurs in all parts of the body, the most frequent sites being the lung, lymph nodes and hepatic vein. Survival with either type beyond one year after diagnosis has been made by laparotomy or punch biopsy is a rarity.

At the present time, radical partial hepa-

tectomy offers the only hope of cure. Resection of the gross tumor and surrounding liver appears to prolong life in some patients and several cures have been reported. The operative mortality is high, up to 25 per cent, and survival from one and one-half to seven years has been reported in 42.2 per cent of forty-five patients. Of the twenty-eight patients seen at the Columbia Presbyterian Medical Center only one has survived, this patient had partial hepatectomy at the age of thirteen years and has been well for six years. Isolated case reports continue to appear but few patients survive for five years following operation. With progress in earlier detection and technical facility these results can only improve.

Metastatic carcinoma of the liver is as common a finding in the family of neoplastic disease as primary carcinoma is rare. The reason for this is twofold. One, the liver filters the blood and lymph return from an organ complex which has a high intrinsic incidence of neoplastic disease and, two, the liver provides a superb medium in which a tumor embolus will grow. Consequently liver metastases from malignant tumors arising from the gastrointestinal tract, the extrahepatic biliary tract and the pancreas are common. The routes of spread are essentially three in number.

Tumor emboli gain access to the portal system either through venous invasion of primary tumors arising in organs drained by the portal system or by direct invasion of the portal system by tumors arising in, for instance, the genitourinary system. Tumor emboli which gain access to the liver via the hepatic artery are frequently derived from metastatic or primary malignant tumors in the lung.

Tumor cell transport via the portal lymphatics in a pure state is rare. However the combination of venous and lymphatic transport is common.

Tumor cell invasion by direct extension into the liver is uncommon since the capsule of the liver seems to offer an effective barrier. Even though this mode of spread is rare, collective evidence suggests that direct invasion when seen is usually from neoplasms arising in the gallbladder or stomach and rarely from the kidney, adrenal gland, colon, esophagus or pancreas.

The diagnosis of a metastatic tumor in the liver must be suspected in every patient under investigation for malignant disease elsewhere in the body. An enlarged liver with or without one or more firm masses,

ated fluid may occur Leukocytosis is usually minimal and the percentage of polymorphonuclear cells rarely exceeds 80 Liver function tests rarely show significant alteration

Treatment The major objectives are decompression of the abscess cavity prevention of local bacterial invasion and riddance of viable amebas from the individual as a whole.

The drug of choice in amebic hepatitis and amebic liver abscess is chloroquine in recommended dosages of 0.6 gm by mouth daily for two days, followed by 0.3 gm. daily for two to three weeks

Abscess cavity decompression should be accomplished by trocar puncture and aspiration. Great care should be exercised to introduce the trocar through the extraperitoneal subdiaphragmatic route and to prevent any bacterial contamination. If it seems justified, the cavity may be irrigated with a suitable broad spectrum antibiotic and chloroquine.

Finally the primary infection must be treated by a course of any of the accepted antiamebic chemotherapeutic agents of which Diiodoquin is perhaps most common. Properly treated amebic hepatitis and hepatic abscess have, on the whole a good prognosis.

READING REFERENCES

- Abel, A. L. Primary Carcinoma of Liver with Report of a Case Successfully Treated by Partial Hepatectomy. *Brit. J. Surg.* 21: 684, 1933.
- Anderson, H. H., Bostick, W. L., and Johnstone, H. G. *Amebiasis: Pathology, Diagnosis, Chemotherapy* Springfield, Ill. Charles C. Thomas, 1953.
- Baker, B. M., Jr., and Carter, E. P. Three Instances of Unusual Gonococcal Infection. *Bull. Johns Hopkins Hosp.* 50: 57, 1932.
- Blakemore, A. H. Portacaval Shunting for Portal Hypertension. *Surg. Gynec. & Obst.* 94: 443, 1952.
- Blakemore, A. H. Treatment of Bleeding Esophageal Varices with Balloon Tamponade. *New York J. Med.* 54: 2037, 1954.
- Blakemore, A. H., and Voorhees, A. B. Jr. End to Side Portacaval Shunt. *Arch. Surg.* 74: 678, 1957.
- Blakemore, A. H. and Voorhees, A. B. Jr., Nutrition Requirements and Management in Patients with Cirrhosis of the Liver Pre- and Postoperative. *Ann. Surg.* 147: 875, 1958.
- Child, C. G., III. *Hepatic Circulation and Portal Hypertension*. Philadelphia, W. B. Saunders Company, 1954, 444 pp.
- Ecker, E. E., and Lynch, J. Spirochetal Abscess of the Liver. *Arch. Path.* 20: 253, 1935.
- Edmondson, H. A., and Steiner, P. E. Primary Carcinoma of the Liver: A Study of 100 Cases Among 48,000 Necropsies. *Cancer* 7: 462, 1954.
- Eisenmenger, W. J., Ahrens, E. H., Jr., Blondheim, S. H., and Kunkel, H. G. The Effect of High Sodium in the Formation and Control of Ascites in Patients with Cirrhosis. *Ann. Int. Med.* 37: 261, 1952.
- Falkoon, W. W., Eckhardt, R. D., Cooper, A. M., and Davidson, C. S. The Effect of Human Serum Albumin, Mercurial Diuretics and a Low Sodium Diet on Sodium Excretion in Patients with Cirrhosis of the Liver. *J. Clin. Invest.* 23: 595, 1949.
- Farnsworth, E. B., and Krakus, J. S. Electrolyte Partition in Patients with Edema of Various Origins, Qualitative and Quantitative Definition of Cations and Anions in Hepatic Cirrhosis. *J. Lab. & Clin. Med.* 33: 1545, 1948.
- Faust, E. C. *Amebiasis*. Springfield, Ill., Charles C. Thomas, 1954.
- Ferris, E. B., Jr. and Blankenhorn, M. A. Obstruction of the Vena Cava Inferior in Liver Abscess, New Diagnostic Sign. *Internat. Clin.* 1: 1, 1941.
- Habib, D. V., Randall, H. T., and Soroff, H. S. Management of Cirrhosis of Liver and Ascites with Particular Reference to Portacaval Shunt Operation. *Surgery* 34: 580, 1953.
- Hunt, A. H. *Portal Hypertension*. London, E. & S. Livingston, 1958.
- Kinney, T. D., and Ginsberg, H. S. Pyogenic Liver Abscesses Due to *Klebsiella pneumoniae*. *New England J. Med.* 288: 145, 1943.
- Layne, J. A., and Schemm, F. R. The Use of a High Fluid Intake and Low Sodium Acid-Ash Diet in the Management of Portal Cirrhosis with Ascites. *Gastroenterology* 9: 705, 1947.
- Lee, H. L. *Salmonella enteritidis* in Liver Abscess, Report of Case. *Chinese M. J.* 49: 577, 1935.
- Linton, R. R., and Warren, R. Emergency Treatment of Massive Bleeding from Esophageal Varices by Transesophageal Suture of These Vessels at Time of Acute Hemorrhage. *Surgery* 33: 243, 1953.
- Madden, J. L., and Lore, J. M., Jr. Surgical Anatomy of the Portal System. *S. Forum*, 2: 133, 1953.
- McDermott, W. V., Jr., Adams, R. D., and Riddell, A. G. Ammonia Metabolism in Man. *Ann. Surg.* 140: 539, 1954.
- Ochsner, A., DeBakey, M., and Murray, S. Pyogenic Abscess of the Liver. *Am. J. Surg.* 40: 293, 1938.
- Popper, H., and Schaffner, F. *Liver: Structure and Function*. New York, Blakiston Company, 1957.
- Rosenberg, D., and Ochsner, A. Primary Carcinoma of Liver: Analysis of 55 Autopsied Cases, Record of Case with Resection, and Review of Recent Literature. *Surgery* 24: 1036, 1948.
- Von Ebert, E. M. Abscess of the Liver Occurring in Association with or Following Typhoid Fever. *Am. J. M. Sc.* 141: 803, 1911.
- Wallace, R. H. Resection of Liver for Hepatoma. *Arch. Surg.* 43: 14, 1941.
- Whipple, A. O. Problem of Portal Hypertension in Relation to Hepatosplenopathies. *Ann. Surg.* 122: 419, 1945.
- Yeomans, F. C. Primary Carcinoma of the Liver: Operation for Recurrence over Seven Years after Primary Operation. *J.A.M.A.* 64: 1301, 1915.

bacteria on in vitro culture is then administered systemically and locally into the abscess cavity.

Incision and drainage should be carried out in the solitary but not the multiple, abscess group for adequate drainage of the latter can seldom be achieved and in any such extensive attempt at hepatic drainage the danger of hemorrhage would be formidable. The course of the incision and subsequent drainage tract should never traverse the free pleural or peritoneal cavities because of possible spread of the infection into these free spaces. If the abscess cannot be drained via the infradiaphragmatic retroperitoneal route the operative procedure should be staged, during the first stage the peritoneal or pleural avenue of communication is obliterated either by suture or packing, at the second stage following inflammatory walling off of the area, the abscess is entered and drained. Diagnostic needle aspiration should be reserved for exploration under direct vision at the time of operation. Percutaneous needle aspiration should be avoided because of uncontrolled pleural or peritoneal contamination and should be reserved for the rare cutaneous pointing of an abscess.

AMEBIC ABSCESS OF THE LIVER

Amebiasis is recognized as a common disease in the Tropics and a less common but nevertheless significant, disease in the Temperate Zone. Since its identification by Loesch in 1875 studies have established the probable incidence in the United States as 10 per cent of the population. This figure probably high for the northern sections of the country and low for the southern areas establishes an incidence which makes the disease of local importance.

The frequency with which amebic abscess of the liver occurs following primary colon infestation is unknown, however in 1945 Payne reported a greater than 50 per cent incidence of hepatitis and a 3 per cent incidence of hepatic abscess in 1000 patients with amebiasis. Amebic abscess has an unexplained predilection for young male adults.

Pathogenesis. *Entamoeba histolytica* traverses the upper gastrointestinal tract following ingestion in the cystic form. In the distal small bowel, the ameba will emerge from the cyst form and, following division, arrive in the cecum as the invasive metacystic trophozoite. During the invasion of the colon wall the venous branches may be invaded and the trophozoite will be transported to

the liver through the portal system. There, the described pathologic process is that of thrombotic occlusion of the smaller venules by fibrin-encased amebas. Subsequent ischemia of the vein wall plus the proteolytic enzyme elaborated by the ameba, will destroy the vein wall and give the ameba free access to invade and destroy the liver lobule. This early and frequently multifocal invasion of the liver gives rise to the clinical diagnosis of amebic hepatitis. With further invasion and proliferation of the intrahepatic colonies focal areas of complete necrosis will form and give rise to the liver abscess. The abscess is usually located in the dome of the right lobe probably as a result of the predominance of right colon blood return to the right lobe and the usual infestation of the cecum and ascending colon. The abscess wall thickness usually represents the rapidity of destruction, with the thicker more fibrous wall confining the slower destructive process. The abscess cavity is filled with a semifluid material made up usually of cellular debris and old blood. In the large abscesses, free floating amebas are rare and a positive diagnosis must be obtained from wall scrapings. In the smaller cavities, amebas can frequently be seen in the "chocolate pus." Cultures for bacteria are usually negative.

The natural course of an abscess, if allowed to progress untreated, is expansion, destruction and rupture into the adjacent anatomic areas. Invasion through the diaphragm into the lung, with occasional subsequent evacuation through a bronchus, is a common complication.

Symptoms. As previously indicated, the acute hepatitis stage if uninterrupted by therapy may merge imperceptibly into the abscess formation, and, as expected, there are symptoms common to both.

The patient usually complains of anorexia, malaise, weakness and a progression of right upper quadrant discomfort ranging from vague heaviness to severe pain. The pain may radiate to the shoulder but this is not a prominent feature. There may be no previous history of dysentery.

Weight loss, fever and profuse sweating are common early findings. Jaundice is infrequent. The liver may be slightly enlarged and slightly tender on pressure. Perhaps one of the most important findings is that of an elevated diaphragm with either reduced mobility or fixation. This may be associated with an actual bulge if an abscess is present. Localized pleurisy with or without associ-

Two ductal systems usually lead the external secretion of the pancreas to the duodenum, although variations are frequent. The major duct, or duct of Wirsung, courses through the tail and body neck and part of the head of the gland to enter the duodenum at the ampulla of Vater in close connection with the orifice of the common bile duct. The major pancreatic duct and the bile duct join and share a common opening into the duodenum in approximately 70 per cent of human beings. In many of these the two lumens are separated by a septum to within a few millimeters of the common opening. In other individuals the openings are separate although close together. The minor pancreatic duct, the duct of Santorini, arises in the head of the pancreas and usually enters the duodenum separately proximal to the ampulla of Vater. Variations in the minor duct are common, it may connect freely with the duct of Wirsung within the pancreas, it may be absent or it may be of such prominence that it appears to be draining most of the pancreatic juice. The distal part of the common bile duct may lie between the pancreas and duodenum, or actually may pass through a portion of the head of the pancreas. In any case it is so closely applied to the pancreas that tumors and

occasionally inflammation of this portion of the gland may block it and cause obstructive jaundice.

Microscopically the pancreas consists of glandular alveoli distributed in a racemose fashion about ductules. The islets of Langerhans are scattered throughout, they occur in greater concentration in the body and tail of the pancreas which probably accounts for the fact that about 75 per cent of islet cell tumors are in this region.

The pancreas is both an exocrine and an endocrine gland. Its exocrine secretion pancreatic juice enters the duodenum via the pancreatic ducts and provides enzymes for digestion of foodstuffs as well as alkali to aid in neutralization of acid chyme. Its best known internal secretion, insulin, is important in the regulation of carbohydrate metabolism.

Pancreatic juice is a colorless, odorless, alkaline fluid with a *pH* of 8.0 to 8.3. Its specific gravity varies between 1.007 and 1.042, being increased after stimulation that results in secretion of juice containing nitrogen and, hence, protein and enzymes in great concentration. It is decreased after stimulation that results in juice containing little nitrogen. Its alkalinity is due to its content of large amounts of bicarbonate, the

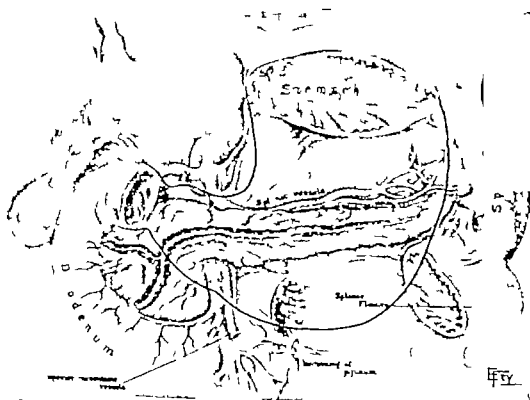


Figure 1. Anatomy of the pancreas, relating to surrounding structures (Walters, W., and Clagett, O. T.: *Surgery of the Pancreas*. In *Piercel, G. M.: The Cyclopedic of Medicine, Surgery and Specialties*, vol. 10. F. A. Davis Co.)

THE PANCREAS

By JOHN M. WAUGH, M.D. and
GEORGE A. HALLENBECK, M.D.

JOHN McMASTER WAUGH, the son of a doctor, is a Missourian who went from Tarkio College, situated in the city of his birth, to Rush Medical College. He received his surgical training at the Mayo Clinic, where he remained to become the Chief of a surgical section.

GEORGE AARON HALLENBECK, also the son of a physician, returned to his birthplace after receiving his liberal arts and medical education at Northwestern University. He chose to do graduate work in physiology to prepare himself for his surgical training.

The pancreas is an elongated gland situated retroperitoneally and lying transversely behind the stomach. Its length varies from 12.5 to 15 cm., its breadth from 3.4 to 4 cm. and its thickness from 1.5 to 2.5 cm. Its weight ranges from 70 to 115 gm. For descriptive purposes it can be divided into three parts, namely the head, the neck, and the body and tail. The head conforms to the concavity formed by the duodenum, with which it is in contact. The neck is a slight narrowing of the gland where it lies anterior to the superior mesenteric vessels. The body tapers toward the left to form the tail.

The relationship of the pancreas to surrounding structures is clearly shown in Figure 1. When the abdomen is first opened, the pancreas is hidden from view by the stomach and the transverse colon and mesocolon. It can be exposed by division of the gastrocolic omentum followed by retraction of the transverse colon downward and retraction of the stomach upward. The neck, together with part of the body and head of the pancreas, also can be exposed by division

of the gastrohepatic omentum and retraction of the stomach downward. The tail of the pancreas touches, or nearly touches, the spleen in a sufficient number of instances that care must be taken to avoid injuring it during splenectomy.

The space bounded by the pancreas posteriorly, the gastrohepatic omentum, stomach and gastrocolic omentum anteriorly and the transverse colon and mesocolon inferiorly is the omental bursa, or lesser peritoneal cavity. It communicates normally with the greater peritoneal cavity by the foramen of Winslow, which lies behind the portal vein, common bile duct and hepatic artery. Arterial blood is supplied to the pancreas by branches from the vascular anastomosis of the superior pancreaticoduodenal artery from above and the inferior pancreaticoduodenal artery from below. The latter courses between the head of the pancreas and the duodenum and branches from the splenic artery, which courses from right to left along the superior border of the body and tail of the gland.

Circumstantial evidence suggests that it may be formed by alpha cells in islets of Langerhans. It has been theorized that the reason for the surprisingly low requirement for insulin after pancreatectomy is that the hyperglycemic factor glucagon, has been eliminated along with endogenous insulin.

Dogs maintained by use of insulin after pancreatectomy do poorly and show fatty infiltration of the liver. This disorder of the metabolism of fats is prevented by the feeding of raw pancreas and, to some extent at least, by feeding pancreatic juice or such lipotropic agents as choline and methionine. Although controversy exists as to the mechanism involved and the importance of the postulated pancreatic hormone (lipocatic) in this regard, it is clear that the pancreas in some way participates in regulation of the metabolism of fat. Evidence has not yet been found that a similar fatty infiltration of the liver develops in human beings after pancreatectomy.

ANOMALIES OF THE PANCREAS

Annular Pancreas. The ventral anlage of the pancreas on rare occasions fails to fuse with the larger dorsal anlage. It then develops independently and comes to surround the second portion of the duodenum as a circular and, sometimes, constricting ring. This abnormally placed pancreatic tissue has been reported to be the site of pancreatitis and to cause obstruction of the common bile duct, but the most frequent way in which it produces symptoms is by causing obstruction of the duodenum. Duodenal obstruction may be evident in the first days of life, but, more commonly patients with this disorder reach adult life before requiring treatment. The lesion appears to be asymptomatic in many persons. Obstruction of the duodenum can be corrected easily in some patients by careful division of the constricting ring through its lateral side, followed by suture of the cut surfaces of ectopic pancreas. In other patients, the condition can be better treated by gastroenterostomy or duodenojejunostomy. Of these last two operations, duodenojejunostomy performed retrocolic ally probably is preferable because it is more likely to avoid the complication of peptic stomal ulceration, which can follow gastroenterostomy and because it should provide better drainage of the proximal portion of the duodenum while not interfering with the function of the stomach.

Pancreatic Heterotopia. This is the presence of pancreatic tissue outside its usual



Figure 2 Nodule of heterotopic pancreatic tissue, measuring 1.5 by 1 by 0.8 cm. in the anterior wall of duodenum immediately below the pylorus. The mass looked like a fibroma grossly at operation (Vaugh, J. M.: *Surgical Diseases of the Pancreas*. In Walters, W.: *Lewis Practice of Surgery* vol. 7 W. F. Prior Co.)

location and without continuity with the pancreas proper. A total of 471 instances had been reported in the literature up to 1944. Heterotopic pancreatic tissue may occur in the walls of the duodenum (Fig. 2) stomach or jejunum, less frequently it is seen in the gallbladder, splenic capsule or ileum or in Meckel's diverticulum. The tissue usually presents itself as a single, firm, yellow or white, opaque, lobulated, round or irregular node with a granular surface. Typically this misplaced tissue may range from 1 cm. to 4 cm. in diameter. Vague digestive symptoms, or symptoms suggestive of gastric or duodenal ulcer or of cholecystic disease, may be associated with heterotopic pancreatic tissue and apparently have often been relieved by removal of the tissue.

TRAUMATIC LESIONS OF THE PANCREAS AND PANCREATIC FISTULAE

Injuries of the Pancreas. The pancreas is injured but rarely because of its anatomic situation. It is protected posteriorly by the spinal column, the lower ribs offer it a certain degree of shelter anteriorly and it is shielded by the stomach and other viscera. Trauma severe enough to injure the pancreas usually produces injury to other organs of

exact concentration of which depends on the rate at which juice is being secreted. The sum of the concentrations of chloride and bicarbonate ions is constant and nearly the same as the total base in plasma. At extremely decreased rates of secretion the bicarbonate may exist in the same concentrations that it does in plasma, whereas it may reach more than 140 mEq per liter at faster rates of secretion. Sodium and potassium occur in concentrations equivalent to those in plasma. The juice contains a considerable amount of protein, with which the enzymes are associated.

Pancreatic juice contains proteolytic, amylolytic and lipolytic enzymes. Trypsin, probably a combination of two or more enzymes, is secreted in the inactive form of trypsinogen which is activated by contact with enterokinase found in the small intestine. This trypsin group of enzymes can reduce proteins to peptides. Pancreatic lipase splits fats to glycerin and fatty acids in the presence of bile salts furnished by the liver. Pancreatic amylase splits carbohydrates to maltose. The optimal pH for activity of all three enzymes is more than 7.0. As little as 10 per cent of the normal quantity of pancreatic juice suffices to permit normal digestion and absorption of food. Complete removal of the gland in dogs and man is usually but inexplicably not always followed by impaired digestion and absorption, particularly of fat and protein. The reason for the lack of this deficit in some patients after pancreatectomy is not understood. Steatorrhea resulting from deficiency of pancreatic juice often can be controlled by oral administration of pancreatic extracts (pancreatin).

Pancreatic juice also contributes to the neutralization of chyme entering the duodenum from the stomach, sharing this function with bile and mucus entericus. This is of great importance for two reasons: the enzymes in intestinal and pancreatic juice function best in an alkaline environment and peptic ulceration of the duodenum, a likelihood when such neutralization is prevented, is controlled if neutralization is effective.

Experiments carried out principally on dogs indicate that secretion of pancreatic juice can be stimulated by sham feeding (cephalic phase) and by the presence of foodstuffs or acid in the intestine (intestinal phase). A gastric phase of pancreatic secretion has not been demonstrated, that is, food limited to the stomach does not stimulate pancreatic secretion. Vagotomy abolishes the

cephalic phase of pancreatic secretion as it does the same mechanism for gastric secretion. Pancreatic juice secreted in response to sham feeding or stimulation of the vagus nerves is sparse, viscid and rich in enzymes. Acid placed in the duodenum calls forth profuse secretion of pancreatic juice which is relatively poor in enzymes but of considerable volume; it contains bicarbonate in great concentration.

In 1902, Bayliss and Starling extracted a substance from the mucosa of the small intestine which when given intravenously stimulated secretion of watery pancreatic juice poor in enzymes. This was the first known hormone and was named secretin. The validity of the concept that it is a hormone has been amply proved since. One adequate stimulus for release of secretin is a decrease of the pH of duodenal contents. In the dog, the pH must reach 4.0 before a very active response is obtained. Products of digestion of protein, fats, fatty acids and soaps when introduced into the small intestine stimulate flow of a pancreatic juice rich in enzymes. Products of digestion of carbohydrate are less active in this regard.

In 1943 Harper and Raper isolated from duodenal mucosa a second substance which they named pancreozymin. This calls forth the secretion of pancreatic juice rich in enzymes when it is given intravenously. Evidence now favors the concept that pancreozymin is important in the pancreatic secretory response to food. The presence or relative importance of nervous reflexes between the small intestine and pancreas in stimulation of pancreatic secretion by food cannot be assessed with certainty at present.

Total pancreatectomy is followed by fatal diabetes unless insulin is given. The relationship between the pancreas, the adrenal cortex and the anterior pituitary gland in the regulation of carbohydrate metabolism and in spontaneously occurring diabetes is of great importance. Suffice it to say that insulin, formed by the beta cells in the islets of Langerhans in the pancreas, controls the hyperglycemia of ordinary diabetes and of the diabetes which follows pancreatectomy. Paradoxically, as little as 15 to 30 units of insulin daily usually controls the diabetes after pancreatectomy in man. These quantities are much less than those often required to control spontaneously occurring diabetes. A hyperglycemic glycogenolytic factor called glucagon also can be extracted from pancreatic tissue. Its status as a hormone and its physiologic importance remain uncertain.

plied equally to the pancreatic duct in a way that should eliminate any differential pressure between the two systems. Recent evidence indicates that bile in the canine pancreatic duct at physiologic pressures does not cause pancreatitis. Obstruction of the pancreatic duct in fasted animals produces edema of the pancreas and prompt increase in values for serum amylase but does not produce the severe pancreatitis seen clinically.

If dogs are fed shortly before the pancreatic ducts are ligated, severe pancreatitis usually develops. Obstruction of the flow of pancreatic juice in the presence of an actively secreting gland apparently can cause pancreatitis. Although ligation of pancreatic arteries or veins alone does not cause acute pancreatitis, such alteration of the pancreatic blood vessels, when combined with obstruction to pancreatic ducts results in severer acute pancreatitis than that caused by obstruction of the ducts alone. It seems likely that vascular factors can be important in the progression of acute pancreatitis. Cases of pancreatitis occurring in the presence of obstruction of a common channel by a stone at the ampulla of Vater have been reported, but absence of such obvious mechanical obstruction is commoner. Edema and spasm of the sphincter of Oddi have been postulated. Ample evidence in human beings and in dogs shows that such spasm can occur in response to a variety of drugs. Alcohol, commonly linked with pancreatitis in clinical cases, is especially active in this regard.

Once the disease is established, pancreatic enzymes often exude from the gland into the peritoneal cavity. Lipase hydrolyzes near-by fat, producing necrosis of the fat, which is grossly visible as whitish spots consisting of salts of fatty acids.

Acute edematous pancreatitis. This form of acute pancreatitis typically is manifested as an attack of severe epigastric pain usually referred through to the back and often felt in the scapular regions and lower part of the thorax. As already mentioned, many of these patients have gallstones as well and previously may have experienced the indigestion and colic characteristic of this disorder. Jaundice, if it occurs, is usually transient and seldom severe. Nausea and vomiting are common and epigastric tenderness is virtually always present. Leukocytosis and a moderate degree of fever occur. Shock, a typical feature of acute hemorrhagic pancreatitis, is not seen in the acute edematous variety of the disease. Although penetrating

duodenal or gastric ulcer and coronary occlusion may be included in the differential diagnosis the differentiation between this disease and simple acute cholecystitis which so often accompanies it, is usually the most pressing problem.

The presence of acute pancreatitis must be suspected in any patient who has sudden onset of epigastric pain often suggestive of biliary colic or acute cholecystitis, which persists for more than twenty four hours. The presence of this type of pancreatitis formerly became known with certainty only after the swollen, tense pancreas and, often, regions of necrosis of fat were seen at laparotomy. Now the presence or absence of acute pancreatitis can be learned with considerable certainty by a determination of serum amylase, the value for which will be increased when the disease is present. The activity of serum lipase requires longer to measure, increases more slowly but remains increased longer than does the activity of serum amylase. Measurement of serum amylase is an extremely important test that can be done quickly and should be performed whenever acute pancreatitis enters the differential diagnosis. The test is not absolutely specific. Moderately elevated values can occur for example, with acutely perforated duodenal ulcer.

Surgical treatment has been tried during the acute phase of edematous pancreatitis. It usually has consisted of procedures such as cholecystostomy, cholecystectomy and perhaps drainage of the common bile duct and the placing of drains in the region of the pancreas. Data indicate that a much lower mortality rate is obtained by avoiding laparotomy at this stage of the disease and planning later study of the biliary tract, with subsequent surgical correction of such lesions as cholelithiasis, choledocholithiasis and cholecystitis after the acute process subsides. Thus, current opinion favors medical treatment of acute edematous pancreatitis, a fact that renders accurate diagnosis doubly important.

Specific measures that have been found useful include (1) For relief of pain, the use of nitroglycerin in a dose of $\frac{1}{400}$ or $\frac{1}{200}$ grain is worth a trial. However opiates are usually needed. Bilateral blocking of the splanchnic nerves with procaine hydrochloride is recommended by some physicians, both to relieve pain and in the hope that somehow it will relieve the spasm in the ducts and blood vessels which they assume to occur. (2) Nothing is given by

such severity as to endanger the patient's life. A number of penetrating wounds, caused chiefly by gunshot or stabbing, have been reported. The pancreas can be injured by nonpenetrating wounds such as those caused in auto accidents by compression against the steering wheel. In several of these victims, the pancreas has been sutured and recoveries have been reported. It is seldom that injuries can be diagnosed with certainty by symptoms manifested by the patient. Evidence of shock or intra abdominal hemorrhage is the usual indication for abdominal exploration in these patients. In an exploration of the upper portion of the abdomen after any type of injury one always ascertains that the retroperitoneal portion of the duodenum and the pancreas have not been injured. After suture of the gland, drains should be left to allow escape of the pancreatic fluid from the injured region.

If the main duct has been severed, it may be possible to reconstruct it over a T tube the adjacent tissue being approximated around the T tube with interrupted silk sutures. The T tube should be left in place for about three weeks. In some instances of injury to the body and tail of the pancreas, resection of this part of the gland will offer the safest solution to the problem.

Pancreatic Fistulae. Most pancreatic fistulae follow the drainage of pseudocysts, stab wounds or gunshot wounds of the upper part of the abdomen or surgical trauma to the pancreas. Partial pancreatic fistulae generally close spontaneously although this may require months. Conservative measures should be tried unless the patient is losing an alarming amount of pancreatic juice. The juice often can be collected by a catheter placed into the fistulous tract and attached to a bottle or a rubber bag. Once the fistula is well established, the pancreatic juice often becomes clear and sufficiently inoffensive so that the patient can drink it, thus avoiding the sequelae of constant loss of fluid and electrolytes. Surgical treatment occasionally becomes necessary when conservative methods fail. If the fistula is from the tail or body this portion of the pancreas can be removed readily. This is the simplest way of correcting the fistula. On the other hand, if there is an excessive inflammatory reaction about the fistula, or if the latter is in the region of the head of the pancreas internal drainage, such as to the duodenum or jejunum, usually proves satisfactory.

INFLAMMATION OF THE PANCREAS

Acute Pancreatitis. Acute pancreatitis, apart from the specific type which may occur with mumps may be divided into two varieties namely acute edematous pancreatitis and acute hemorrhagic pancreatitis. The latter and less common type is a fulminating process which frequently progresses rapidly to necrosis of the pancreas and death of the patient. The former and more common variety often is accompanied by cholelithic disease and has a lower mortality rate. Acute pancreatitis has been observed at all ages from two to seventy-seven years, although most patients are between thirty and fifty years of age. As a rule it occurs spontaneously but it can follow trauma to the abdomen or surgical operations usually those in which dissection was performed in the region of the pancreas.

Factors suggested as possible causes of acute pancreatitis are numerous and include infection obstruction of the pancreatic duct, chemical irritation due to reflux of bile or duodenal contents into the pancreatic ductal system, autolysis due to activation and escape of enzymes into the gland, local vascular disturbances and allergic phenomena. At present, it is fair to state that the exact cause of pancreatitis is not known. The commonest theories predicate obstruction of the pancreatic duct and passage of bile into the ductal system. Usually the major pancreatic duct and the common bile duct join shortly before they enter the duodenum and share a common channel of variable length. On anatomic grounds, edema, spasm or the presence of a small stone at the papilla of Vater could obstruct both ducts and leave a channel through which bile could enter the pancreatic duct in 30 to 70 per cent of persons. The former figure was obtained by dissection and the latter by tests in which the papilla was occluded and fluid injected into the bile duct by several investigators.

Injection of bile and numerous other irritants into the dog's pancreatic duct at pressures sufficient to rupture tiny ductules produces severe pancreatitis. It is difficult to imagine how a differential pressure great enough to force bile into the pancreatic duct could occur under normal circumstances. In the dog, at least, pressure in the common bile duct is usually a little less than that in the pancreatic duct. The extremely high pressures that can occur with retching or vomiting are derived from an increase in general intra-abdominal pressure and are ap-

nausea, vomiting, constipation and abdominal distention are common. Although some episodes may last only a few hours, attacks often continue for as long as ten days. Opiates are required for relief. Occasionally the pathologic picture of destructive chronic pancreatitis is seen in patients who do not give a history of pain.

Significant physical findings consist of abdominal tenderness without the boardlike rigidity seen in acute hemorrhagic pancreatitis, often decreased bowel sounds associated with distention of the abdomen and occasionally enough jaundice to be detected grossly.

The most valuable laboratory finding is increased values for serum amylase and lipase during acute attacks of pain, although it must be remembered that this phenomenon may not be seen because so much destruction of pancreatic tissue may have occurred late in the course of the disease. If edema in the head of the pancreas is sufficient to produce jaundice, the concentration of serum bilirubin may increase to 4 or 5 mg. per 100 ml., the pigment is principally of the direct-reacting variety. The concentration of serum calcium may decrease, as described previously and transient glycosuria may occur. Diagnosis in some patients is simplified when a roentgenogram of the abdomen discloses calcific deposits in the region of the pancreas (Fig. 3). When ileus is present, one may see dilatation of some loops of small intestine. If the gallbladder is still present, cholecystography may reveal evidence of cholecystic disease.

The progressive destruction of the pancreas and repeated attacks of severe pain lead to numerous sequelae, including diabetes mellitus, steatorrhea, malnutrition, pancreatic calcification, abscesses and pseudocysts, and addiction to opiates.

Specific medical treatment for the disease is unknown. The measures used during acute attacks are those for the treatment of acute pancreatitis. Between attacks patients may be virtually symptomless unless sequelae of the disease have occurred. Tests should be made at intervals to determine the presence or absence of diabetes. Steatorrhea, when present, usually will be improved if a diet low in content of fats is combined with oral administration of pancreatin. Alcohol definitely provokes attacks in certain patients and should be avoided. Every care must be taken to prevent addiction to opiates, which occurs all too easily in this situation and renders treatment most difficult.

Surgical treatment often offers these sufferers much relief and there are many indications for its use.

Operations on the biliary tract. About half the patients who have relapsing pancreatitis have associated disease of the biliary tract. Surgical treatment of the associated condition often is followed by subsidence of the pancreatitis. A diseased gallbladder should be removed. The common bile duct should be explored to remove any stones that may be present. Drainage of the common duct by a T tube for six months to a year results in subsidence of the associated pancreatitis in many patients when the biliary tract has been diseased and in about half of the patients who do not have associated disease of the biliary tract. Obstructive jaundice persists occasionally and requires a sidetracking operation such as choledochoduodenostomy.

Operation to improve drainage of pancreatic juice into the duodenum. Many surgeons, stimulated by the good results reported by Doubilet and Mulholland, have employed sphincterotomy a procedure in which the sphincter of Oddi is severed, usually through a transduodenal approach, after the papilla of Vater has been identified by passing an instrument through it via the common bile duct. Most surgeons agree that although good results often are obtained, failure is not uncommon. It is understandable that sectioning the sphincter of Oddi



Figure 3. Cholecystogram of patient with relapsing pancreatitis with calcification. The gallbladder functioned normally and contained no stones.

mouth during the acute phase, an injunction that ordinarily is enforced automatically by the anorexia which is present. (3) Continuous suction on a tube placed into the stomach helps control vomiting, may minimize stimulation of pancreatic secretion by acid reaching the duodenum and prevents distention of the intestines as a result of the ileus that sometimes develops. (4) Fluid and electrolytic balances are maintained by appropriate intravenous therapy. (5) Use of anticholinergic drugs has been suggested on the grounds that they can diminish pancreatic secretion. (6) Calcium gluconate given parenterally may be needed, since there is often an appreciable loss of calcium into the regions of fatty necrosis. The amount needed depends on the concentration of calcium in serum, which should be checked periodically. (7) It is well to keep in mind the fact that diabetes sometimes becomes manifest and may require treatment. (8) Some evidence exists that antibiotics are beneficial in reducing the incidence of problems related to secondary infection.

This plan of treatment, although logical, is somewhat empirical. Because acute edematous pancreatitis usually runs a self-limited course and gradually subsides over a period of four to seven days, it is not easy to assess accurately the relative importance of the measures that have been suggested. Definitive surgical treatment of disease of the biliary tract, when this is present, carried out when the pancreatitis is quiescent, is an integral part of treatment.

Acute hemorrhagic pancreatitis. This form of acute pancreatitis may appear to result from progression of the edematous form of the disease or it may occur with dramatic suddenness without prior evidence of milder pancreatitis. The clinical picture is an intensification of that seen in edematous pancreatitis with more rapid progression of symptoms and with the added feature of profound shock. The mortality rate is extremely high and many patients die within twenty-four hours of onset of the disease. If death occurs early, hemorrhage into the pancreas and a hemorrhagic exudate in the peritoneal cavity may be the principal pathologic findings. If the patient lives a few days longer, necrosis and gangrene of the pancreas, even to a point of complete destruction of the gland, are paramount. Those patients surviving a few weeks or more are prone to have abscesses or pseudocysts in the region of the pancreas which may com-

municate with adjacent viscera. The serum amylase is increased.

Treatment of shock demands immediate attention. Correction of the decreased blood volume by transfusion of liberal amounts of whole blood is most important. Although some surgeons favor immediate laparotomy with drainage of the region of the pancreas, the majority favor nonoperative management. Many persons are in such poor condition when first seen that laparotomy would appear to carry a prohibitive risk to the patient. Abscesses or pseudocysts in surviving patients should be drained at once. The principles of nonoperative treatment are the same as those described for acute edematous pancreatitis, with the added need for measures to counteract shock.

Relapsing Pancreatitis. In some unfortunate persons attacks of pancreatitis are recurrent over a period of many years and produce a clinicopathologic picture described by the term "relapsing pancreatitis" or "recurrent pancreatitis." Animal experimentation indicates that extremely mild episodes of acute edematous pancreatitis can be followed by complete functional and morphologic recovery of the pancreas, whereas episodes of ordinary severity produce varying degrees of destruction of pancreatic tissue with subsequent atrophy and fibrosis. Repeated attacks of inflammation, edema and even necrosis of pancreatic tissue progressively destroy functioning pancreatic parenchyma, leaving a fibrosed gland which sometimes shows zones of calcification, abscesses, pseudocysts or even enough destruction of the islets of Langerhans to cause diabetes. Males are affected about six times as often as are females.

The cause of relapsing pancreatitis, like that of acute pancreatitis, cannot be stated with certainty and the same factors are considered to be contributory. Alcoholic excess is frequently associated with relapsing pancreatitis and cholelithic disease has been noted in about 50 per cent of the subjects.

The outstanding symptom during exacerbations of relapsing pancreatitis is pain in the abdomen. It may be mild but is usually extremely severe. It may be felt in any part of the abdomen, but it is characteristically worse on the left and typically extends through to the left upper lumbar region. It is variously described as colicky, cutting, piercing or boring and its severity may lead the patient to seek relief vainly by assuming unusual postures, doubling up in a chair or flexing himself around a pillow. Anorexia,

tum, displacing the stomach superiorly. The contents of pseudocysts are typically thick and dark or reddish brown.

Retention Cysts. True cysts of the pancreas are characterized by a lining of cuboidal or low columnar epithelium and by the presence of dilated pancreatic ducts within or adjacent to the wall (Fig. 5). Thirteen cases, approximately a fourth of Thigpen's series of pancreatic cysts, fell into this category. None of the patients presented a history of trauma and gallstones or evidence of cholecystitis was present in less than a third at the time of operation. Evidence of pancreatitis was present in a third of the subjects, but it is difficult to say whether this was a cause or a result of the disease. Ten retention cysts were unilocular and three were multilocular. They varied from 4 to more than 20 cm. in diameter. Fluid in these cysts is usually yellow.

Cystadenomas. These are benign neoplasms in which the cysts are multilocular and, of course, lined with epithelium (Fig. 6). Seven occurred in Thigpen's series.

Cystadenocarcinomas. Four such lesions were found among the fifty-five patients with pancreatic cysts; one of these was associated with hepatic metastasis at the time of exploration. The cysts associated with this type of malignant tumor varied in size from that of a pear to 15 cm. in diameter. Microscopically the growths were predominantly papillary cystadenocarcinomas although mixed in one were regions of squamous cell epithelioma. Needless to say this variety of

carcinoma of the pancreas is extremely rare and is not to be confused with the common adenocarcinoma of the pancreas.

Other Cystic Lesions of the Pancreas. Thigpen's series of fifty-five cystic lesions of the pancreas was completed by two cystic hemangioendotheliomas and one of each of the following: cystic adenocarcinoma of the islets of Langerhans, cavernous hemangioma, dermoid cyst, epidermoid cyst and a cyst of accessory pancreatic tissue.

Symptoms and Diagnosis of Cyst of Pancreas. A pancreatic cyst can become extremely large so gradually that the patient is unaware of it until he or his physician happens to palpate the tumor which usually presents as a smooth, firm, rounded epigastric mass. Although symptoms may be denied in some instances, there is usually a feeling of tightness and pressure in the upper part of the abdomen or actual pain. An antecedent history of trauma, or of symptoms suggestive of pancreatitis or cholecystitis is likely to be elicited in the case of pseudocysts, and symptoms of the last-named two conditions, when present, may overshadow the clinical picture. Anorexia and consequent loss of weight are common and chills and fever sometimes occur with pseudocysts. Jaundice is present in 5 to 10 per cent of patients who have pancreatic cysts. Steatorrhea appears to be present rarely.

The mass is firm and rounded. It feels cystic, lies in the upper part of the abdomen, is too tense to allow demonstration of a fluid wave, is typically not tender or only slightly

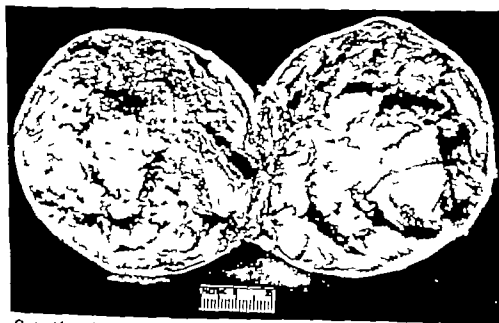


Figure 4. Cyst with walls of fibrous connective tissue (pseudocysts) (Waugh, J. M.: Surgical Diseases of the Pancreas. In Walters, W.: Lewis Practice of Surgery vol. 7 W. F. Prior Co.)

cannot influence obstruction of pancreatic ducts distant from the sphincter

Operations on the pancreas In recent years increasingly more patients who have chronic relapsing pancreatitis have been treated by resection of a portion of the tail of the pancreas and implantation of the cut surface of the gland into a loop of jejunum made with the Roux en Y technique. The purpose of this operation is to permit retrograde drainage of pancreatic juice into the intestine. In many cases there will be several obstructions or strictures of the main pancreatic duct as it courses through the body and tail of the gland. To facilitate drainage of as much of the gland as possible this part of the duct can be opened throughout a considerable length. The resulting pancreaticojejunostomy then becomes almost side to side (Puertow and Gillesby). Encouraging results have been reported to follow the use of this procedure in advanced cases of pancreatitis. Abscesses may require drainage. Pseudocysts may need external or internal drainage. If the disease is limited to the tail and body of the pancreas, where considerable degrees of calcification may occur resection of this portion of the gland should be considered. Total pancreatectomy has been used for diffuse calcification and unbearable pain. The operation is formidable and is attended by late complications which rarely make it advisable.

Operations to decrease external pancreatic secretion. Partial gastrectomy, which results in diminution of the volume of pancreatic secretion, has been suggested as treatment for chronic relapsing pancreatitis. At present, data regarding its effectiveness are few and do not indicate that the procedure has much promise.

Operation to relieve pain. Splanchnicectomy has been performed to relieve pain and good results have been reported by some observers. Our results with it have been discouraging.

PANCREATIC CALCULI

Quite different from the calcification which sometimes occurs in the parenchyma of the pancreas in association with relapsing pancreatitis are calculi that sometimes develop in the pancreatic ducts. These stones are composed of calcium carbonate and calcium phosphate and in many respects they are similar to calculi of the salivary glands. They are grayish white, hard and irregularly shaped. They may vary in size

from 1 mm. to 7 cm. They may be single or may be multiple to the extent of several hundred.

Patients of all age groups are affected, but pancreatic calculi occur more commonly in patients who are between the ages of forty and sixty years. Males are affected three or four times as frequently as females. The etiologic factors are unknown.

Although the incidence at necropsy was thought to be 1 in 1500 to 1 in 3000, it is probably greater according to Snell and Comfort.

The symptoms may be minimal or entirely absent but usually follow the pattern described under relapsing pancreatitis. Snell and Comfort list the following symptoms in order of frequency: abdominal colicky pain, steatorrhea, diabetes mellitus and, less frequently jaundice. The diagnosis is confirmed by a roentgenogram of the abdomen. The treatment is surgical and consists of exposure and incision of the duct over the stone, with removal of the latter. Careful exploration of the ducts often reveals multiple stones. After removal of the stones, a small T tube should be left in the duct for two or three weeks. There is rarely any drainage of pancreatic juice after removal of the tube. If parenchymal calcification is associated, it may be advisable to excise a portion of the gland.

CYSTIC LESIONS OF THE PANCREAS

Pseudocysts. These lesions of the pancreas are defined as cysts having walls composed of fibrous tissue without any epithelial lining (Fig 4). They accounted for twenty four of the fifty five cysts of the pancreas studied by Thigpen. Etiologic factors are believed to be pancreatitis, disease of the biliary tract and trauma. A history of trauma was obtained in four of the twenty four subjects and disease of the gallbladder pancreatitis or a suggestive history of such was present in nineteen of the remaining twenty patients.

In other series, trauma has been reported to be a cause more frequently than would be indicated by these figures. Development of a pseudocyst after trauma to the abdomen may be an extremely slow process and months or even a year or more may pass before the cyst is recognized.

Pseudocysts may range in size from a few centimeters to as much as 35 cm. in diameter and are almost always unilocular. Such cysts may present themselves through the gastrohepatic omentum, displacing the stomach inferiorly or through the gastrocolic omen-

stomach or a loop of small intestine. In recent years, the procedure of internal drainage of pancreatic cysts, first done in 1923 has been practiced increasingly and has given good results in the hands of many surgeons. It is especially useful in true cysts of the pancreas. Most pseudocysts respond well to prolonged drainage with or without marsupialization.

NEOPLASMS OF THE PANCREAS

Benign Tumors. Benign tumors of the ampulla of Vater are rare. They consist of polypoid masses of tissue which may not produce symptoms but which can cause biliary obstruction and jaundice. The surgeon dealing with presumed malignant neoplasms of the ampulla of Vater should keep in mind the possibility of the presence of these small benign polyps and avoid radical pancreatectomy for a benign lesion.

Sarcoma Sarcoma of the pancreas occurs with extreme rarity results of excision, when this has been possible, have been poor.

Carcinoma. Carcinoma of the pancreas is the commonest neoplasm of this organ, it constitutes 1 to 2 per cent of all carcinomas. Approximately 75 per cent of carcinomas of the pancreas originate in the head of the organ and only 25 per cent in the body or tail. Symptoms and surgical management differ with the location of the lesion so that carcinoma of the head and that of the remainder of the pancreas can be profitably considered separately. Furthermore, the clinical features of carcinoma of the head of the pancreas and of carcinoma originating in the region of the ampulla of Vater have so much in common that these two lesions are best discussed together.

Carcinoma of the head of the pancreas and carcinoma of the ampulla of Vater. Carcinoma of the head of the pancreas and carcinoma of the ampulla of Vater occur more frequently in males than in females and are commonest in the sixth decade of life.

Both these tumors are adenocarcinomas, but carcinomas originating from the ampulla or the papilla of Vater as well as from the adjacent bile ducts are more likely to have a papillary arrangement of cells and to be more differentiated histologically than are carcinomas of the pancreas itself. Metastasis to regional lymph nodes apparently occurs earlier in carcinoma of the head of the pancreas than it does in ampullary tumors and invasion of the perineural lymphatic vessels

is more common in growths originating in the pancreas. Ampullary carcinomas are often soft, whereas carcinoma of the head of the pancreas is typically hard. Carcinomas of the head of the pancreas usually are accompanied by intense fibrosis of the surrounding pancreas which may if the ducts are blocked, involve the entire organ. Biopsy often shows only fibrosis even when carcinoma is present, an important point to remember during operations for this disease.

Obstructive jaundice becomes the most striking feature of both carcinoma of the head of the pancreas and carcinoma of the ampulla of Vater and actually may be the first detectable sign of disease in lesions of the ampulla. In carcinoma of the head of the pancreas, jaundice is usually preceded by a decline in health over a period as long as six months during which time the patient may experience anorexia with associated loss of weight and progressive weakness and epigastric pain often referred to the back. The pain of pancreatic carcinoma is constant, it often is described as nagging or gnawing and appears to be intensified at night. Pain may be absent in ampullary carcinoma and when it occurs it is often episodic at first.

Jaundice usually occurs sooner or later in association with carcinoma in either region and, at the time surgical intervention is being debated, the problem often has resolved itself into the differential diagnosis of jaundice. The point of practical importance is to separate the patients who have intrahepatic jaundice, due usually to hepatitis, and who should not undergo operation, from those who have obstructive jaundice, due to choledochal stone or neoplastic obstruction, and who should have surgical treatment.

Points in favor of hepatitis include a history of exposure to the epidemic type of the disease or of prior blood transfusion, the absence of a palpable gallbladder, lack of significant pain, a predominance of indirect reacting bilirubin in the serum early in the disease, positive results of cephalin-cholesterol flocculation tests and other similar tests which indicate parenchymatous disease of the liver and the presence of some bile in the duodenum on duodenal drainage.

Patients who have obstruction of the common bile duct due to stone often give a prior history suggestive of cholecystitis or cholelithiasis. They usually have colicky intermittent pain associated with chills and fever that is different from the boring pain which



Figure 5. Retention cyst (true cyst) (Wang, J. M. *Surgical Diseases of the Pancreas*. In *Walter, W. Lewis Practice of Surgery* vol. 7 W. F. Prior Co.)

so and usually does not move with respiration. Unless the patient is experiencing an attack of acute pancreatitis, the values for serum amylase and lipase are usually within normal limits. Roentgenologic examination of the stomach using barium reveals displacement of the organ by the extrinsic mass. When the head of the pancreas is involved, widening of the curve of the duodenum often can be demonstrated by the same method. Calcium in the wall of a pancreatic

cyst occasionally may outline it well on plain roentgenograms of the abdomen. Other cystic enlargements which enter the differential diagnosis are cysts of the omentum, liver, ovary, kidney and spleen. Excretory urograms and sometimes even retrograde pyelography may be needed to exclude hydronephrosis.

Treatment of Pancreatic Cysts. The treatment of choice for any cyst of the pancreas is total excision, with preservation of as much of the normal pancreas as possible. If this proves to be impossible or to involve unwarranted risk to the patient, the alternatives are marsupialization or internal drainage by anastomosis of the cyst to the stomach or small intestine. Thirty-five per cent of the afore-mentioned fifty-five patients studied by Thigpen had cystic lesions of the pancreas that could be excised readily. Excision is particularly important whenever possible in the case of neoplastic cysts, since if the lesion is malignant, as some are, it represents the only hope of cure.

Pseudocysts respond to external drainage by marsupialization more readily than do true cysts. By marsupialization is meant evacuation of the cyst, exploration of it to rule out the possibility of a malignant lesion and suture of the wall of the cyst to the skin, leaving an opening about 2.5 cm. in diameter. A large catheter is sutured in place to insure free drainage and in most subjects the resulting fistula soon closes. Continued drainage of pancreatic juice may continue for years in a few patients if further steps are not taken. A chronic fistula usually can be corrected satisfactorily by an operation in which the fistulous tract is dissected out so that its end can be implanted into the



Figure 6. Multilocular cystadenoma of pancreas excised surgically (Wang, J. M. *Surgical Diseases of the Pancreas*. In *Walter, W. Lewis Practice of Surgery* vol. 7 W. F. Prior Co.)

pancreas, may provide a positive diagnosis. False negative results are common, however and the surgeon is often forced to rely on his own appraisal of the lesion based on gross inspection. Carcinomas of the ampulla may be small, soft and difficult to feel accurately and direct inspection of the papilla of Vater after the duodenum is opened may be necessary for certain diagnosis. If the neoplasm has spread, a diagnosis often can be made by biopsy of metastatic lesions in the liver, regional lymph nodes, omentum or elsewhere.

When the surgeon has satisfied himself that the patient has a carcinoma of the ampullary region or of the head of the pancreas, he must decide whether the disease is sufficiently localized to permit and warrant resection or whether he is forced to content himself with a palliative procedure designed to relieve jaundice and sometimes to forestall or correct obstruction of the duodenum. Since palliative procedures offer no hope of cure, resection is preferable. However since resection in the presence of metastatic lesions which cannot all be removed is followed by no greater comfort and no longer survival than are palliative procedures and, because the operative mortality and morbidity rates in these patients are greater than they are in those subjected to simpler palliative procedures, resection is to be avoided in these circumstances. This is in contrast to many malignant tumors of the stomach and colon, for example, in which palliative resections are often indicated when technically feasible. Resection of the head of the pancreas involves removal of the duodenum, because the blood supply of these two organs is inseparable, and of the distal portions of the common bile duct and stomach, followed by anastomosis of the biliary tract, stomach and sometimes the tail of the pancreas to the intestine. Because the first successful resection of this type was performed by Whipple, in 1935 it is generally referred to as the Whipple operation. The presence of metastatic tumors in the liver, omentum, root of the mesentery of the small intestine and the lymph nodes around the aorta and celiac axis, as well as more distant metastasis, is a contraindication to performance of the operation.

In the absence of these findings, the surgeon must decide whether fixation of the lesion itself by local extension precludes its removal on technical grounds. Sometimes firm fixation posteriorly or malignant involvement surrounding the superior mesenteric

vessels makes it apparent that resection is impossible. On other occasions, the local resectability of the lesion can be determined only after preliminary dissection has been started, with incision of the peritoneum lateral to the duodenum and elevation of the duodenum and pancreatic head to find out whether the growth has involved the vena cava, portal vein or superior mesenteric vein. Extension of carcinoma around these veins is the commonest factor preventing resection when other factors appear favorable. A two-stage procedure applicable to this situation in which the portal vein is first ligated with the plan of removing it along with the growth ten days or two weeks later has been described by Childs. Ligation of the portal vein under these circumstances has been tolerated satisfactorily but data are not yet available to prove that this extensive procedure produces results good enough to justify it.

Approximately 30 per cent of malignant lesions causing jaundice are found to be suitable for resection. Although resection in one stage is preferable, a few patients remain in whom resection appears warranted and technically feasible but whose general condition is so poor that the risk appears too great. In these, a two-stage procedure is applicable, the surgeon corrects the jaundice in the first stage and plans to perform the resection later if and when, the patient's condition can be improved. Many different anatomic arrangements have been used to re-establish gastrointestinal continuity, to join the biliary tract to the intestine and to dispose of the remainder of the pancreas. The tail of the pancreas if uninvaded by the malignant process, should be preserved to prevent the diabetes which would follow its removal. Some surgeons simply have tied its duct, oversewn its end and left it isolated from the intestinal tract, but in most instances digestion is improved if the cut end of the gland is implanted into the intestine. It is rational to arrange the parts so that bile and pancreatic juice enter the intestine before gastric contents do so they can neutralize chyme at the anastomosis and so minimize the chance of formation of gastrojejunal ulcer; at the same time it is well to isolate the biliary and pancreatic systems from the main stream of food (Fig. 7).

The most satisfactory procedure in our hands has been end-to-end pancreaticojejunostomy with end-to-side choledochojejunostomy and end-to-side postcolic or antecolic antiperistaltic gastrojejunojejunostomy. Suc

may be described by some patients with carcinoma of the pancreas. However it should be remembered that the presence, absence or quality of pain is not an unfailing guide in the differential diagnosis of obstructive jaundice because some patients with jaundice from obstruction of the common bile duct by stone have no pain. The jaundice associated with stone in the common bile duct tends to fluctuate and is typically moderate in degree, because more often than not the obstruction is not complete. Early in the course of the disease results of liver function tests indicate obstruction as opposed to intrahepatic jaundice although later these results may not be clear cut. Because of cholelithiasis and cholecystitis the gallbladder is usually thickened sufficiently so that it does not dilate to an extent whereby it becomes palpable.

In the presence of neoplastic obstruction of the bile ducts, the gallbladder is usually normal and can dilate hugely to become palpable. The generalization that in the presence of obstructive jaundice a palpable gallbladder indicates a malignant lesion and a nonpalpable gallbladder indicates stone is an old rule of thumb in medicine that constitutes Courvoisier's law. Although not infallible, this rule is extremely useful. The jaundice of neoplastic obstruction of the bile ducts is progressive, intense and unrelenting. This type of jaundice is often thought of as painless and this is frequently so especially when the lesion is of the ampulla. However it should be clear that many patients with carcinoma of the pancreas have pain. Although they occasionally are palpable these tumors cannot be felt in most patients in the early phase of the disease. Results of liver function tests carried out early in the course of the jaundice are suggestive of obstruction rather than of intrahepatic disease. Bile is not recoverable from the duodenum on duodenal drainage in the majority of patients who have carcinoma of the head of the pancreas and in about half of those with ampullary neoplasms. Gross blood is obtained in the duodenal contents in about 25 per cent of patients who have ampullary lesions. Deformities in the region of the ampulla can be seen in about a fourth of the patients with neoplasms in this region. Values for serum amylase and lipase are occasionally but not consistently increased.

With acute diagnosis, a few patients who have carcinoma of the head of the pancreas may be operated on before the onset of jaundice. Much more commonly however

the preoperative diagnosis is that of obstructive jaundice the differentiation between obstruction due to stone and that due to neoplasm is sometimes almost certain and sometimes in considerable doubt. Adequate preoperative preparation is most important and may require a few days to as much as two weeks. Anemia should be corrected by transfusion of whole blood. A diet high in carbohydrate and protein and low in fat is given. It may be supplemented by intravenous administration of solutions of glucose and protein hydrolysate. Particularly important is administration of vitamin K to restore the often-depleted prothrombin to normal levels and prevent the bleeding that formerly contributed so much to the hazards of operations on jaundiced patients. If an increased prothrombin time does not return to normal after administration of vitamin K, considerable hepatic damage is present and the risk of operation is increased.

Once the abdomen has been opened through a right upper rectus or upper transverse incision, the surgeon's first task is to determine the nature of the lesion and, if it is malignant, whether or not it can and should be resected.

When biliary obstruction is caused by a stone impacted at the ampulla of Vater, cholecystitis and cholelithiasis usually are present, the gallbladder usually is not greatly dilated the common bile duct is usually enlarged and may or may not contain palpable stones. There is no true tumor palpable in the region of the head of the pancreas, although the region of the ampulla may feel thickened, and there is of course, no metastasis in the liver or elsewhere. The situation calls for exploration of the common bile duct, removal of the stone or stones, drainage of the duct with a T tube and cholecystectomy.

When obstructive jaundice is caused by neoplasm, the gallbladder is typically thin walled and distended, except for the unusual instances in which previous disease has rendered it inelastic or in which metastatic lesions blocking the hepatic or cystic ducts prevent bile from reaching it. Although a strategically placed carcinoma of the head of the pancreas may obstruct the common bile duct while the tumor is still small, the presence of large, firm, nodular easily palpable tumors associated with extensive fibrosis of the gland is the rule. Immediate examination by the frozen section technique of tissue for biopsy taken with the Silverman needle or by removal of a wedge-shaped piece of

per cent, whereas for patients with carcinoma of the head of the pancreas it was 28 per cent. In carcinoma of the pancreatic head, three (12.5 per cent) of twenty-four potential survivors lived three years or more and three (16 per cent) of nineteen possible survivors lived five years or more. In carcinoma of the ampulla, nine (47 per cent) of nineteen potential survivors lived three years and five (38 per cent) of thirteen possible survivors lived five or more years after leaving the hospital. Patients who have this operation and die of recurrent carcinoma often live no longer than do others who have only palliative procedures. Patients who live three years have an excellent chance of living five or more years. Results of radical extirpation of carcinoma of the ampullary region leave no doubt that it is a worth while undertaking (Fig. 8). Results of treatment of carcinoma of the head of the pancreas are discouraging, but since there is at present no other procedure that offers any hope of cure, radical resection is justifiable in properly selected cases.

Carcinoma of the body and tail of the pancreas. Approximately 25 per cent of carcinomas of the pancreas originate in the body and tail. It is not known why the incidence in the head is three times that in the body and tail. The sex and age of patients compare with those of patients having carcinoma of the head and the pathologic aspects are essentially the same. There is a great incidence of local invasion of the surrounding structures. The mesenteric vessels, celiac axis, aorta and vena cava frequently are surrounded and invaded before laparotomy is performed. Because initial symp-

toms are so often obscure, local invasion usually has occurred already at the time of exploration and accounts for the fact that resection is frequently not possible. Metastasis arises early and the first spread is usually to the liver with the lungs next in order.

The symptoms differ somewhat from those seen in carcinoma of the head, although pain which is the main symptom, is usually constant and unrelenting and more noticeable at night. Frequently it extends to the back and left side. Anorexia and loss of weight are frequent complaints and can be extreme even in patients who have had a tumor of the pancreas for only a short time. Jaundice is a late complication and usually indicates such an extensive tumor that resection is impossible. Nausea and vomiting are not infrequent and diarrhea and steatorrhea may be seen occasionally.

The laboratory data are typically not helpful in diagnosis. Some degree of anemia usually is present. The patient occasionally may have increased serum amylase. Roentgenographic findings are not particularly diagnostic, but distortion of the stomach, the colon or the upper portion of the small intestine may be noted.

Physical examination usually reveals nothing abnormal except a mass which is often in the midportion of the epigastrium or a little to the left. It may be rather indistinct, but at times it may be large. Venous thrombosis is seen frequently in patients with advanced carcinoma of the body and tail of the pancreas and usually heralds a hopeless situation. As a rule, diagnosis is not made and confirmed except at laparotomy.



Figure 8. Carcinomatous papilla of Vater removed by radical pancreaticoduodenectomy (probe through papilla) with survival for nine and one-half years after operation (Vaugh, J. M. *Surgical Diseases of the Pancreas*. In Walters, W.: *Lewis Practice of Surgery* vol. 7 W. F. Prior Co.)

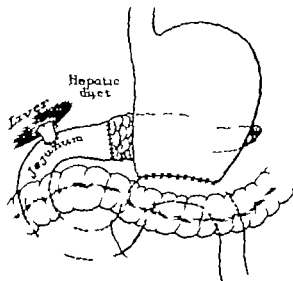


Figure 7 A satisfactory method of re-establishing pancreatic, biliary and gastrointestinal continuity after radical pancreaticoduodenectomy (Waugh, J M. *Surgical Diseases of the Pancreas*. In Walters, W. Lewis Practice of Surgery vol. 7 W F Prior Co.)

tion is applied constantly after operation to a Levin tube placed in the proximal limb of the jejunum for about three days while fluid and electrolytic balance is maintained by appropriate intravenous therapy. Vitamins K, B and C are given daily and antibiotics should be administered to combat infection. The wound is freely drained by Penrose drains and often by Chaffin sump drains.

Should a biliary or pancreatic fistula develop it is treated by suction applied through a catheter with protection of the surrounding skin by ointments such as lanolin or aluminum paste. Such fistulae usually close spontaneously. The patient should be examined frequently for diabetes during the postoperative period, since transient hyperglycemia occasionally occurs requiring use of insulin. Although reimplantation of the pancreas into the jejunum greatly reduces the loss of fat in the feces, as compared with the loss in early techniques in which the pancreatic stump was simply closed, some patients require pancreatin to control steatorrhea. The dosage varies with the patient but may be as much as 30 tablets a day in extreme instances.

If the lesion is inoperable, the surgeon is justified in relieving the jaundice by making some type of anastomosis between the biliary and intestinal tracts. Gastroenterostomy should be performed as well when there is evidence of actual or impending obstruction of the duodenum. Relief of jaundice eliminates the pruritus that is so often a problem and improves the patient's sense of well

being. Various surgeons advocate such procedures as cholecystogastrostomy, cholecystoduodenostomy, cholecystojejunostomy, or side-to-side choledochoduodenostomy for relief of jaundice. We know of no data to establish unequivocally one method as superior to the others and choledochoduodenostomy and choledochojejunostomy have given satisfactory results in our hands.

Bilateral block of the splanchnic nerve with alcohol often helps control the pain of pancreatic carcinoma.

Transduodenal excision of ampullary carcinoma frequently was used before the Whipple operation was developed. Results indicate that cure is so unusual after it that the procedure is best classed as palliative and is seldom, if ever indicated at present.

The operative mortality rate for palliative operations performed because of carcinoma of the head of the pancreas and ampullary carcinoma has been extremely high in the past, figures of 50 per cent and even more being reported in the earlier literature. Since 1939 a pronounced decrease has occurred in the mortality rate, probably because of the preoperative and postoperative use of vitamin K and improvement in general supportive therapy. During the five-year period from 1949 to 1953 inclusive, the operative mortality rate in 213 operations performed at the Mayo Clinic for relief of jaundice due to obstruction of the bile ducts by malignant lesions of the pancreas or bile ducts was 7.5 per cent. Survivors almost always are relieved of jaundice. Survival after palliative operations is usually short, averaging about six months in carcinoma of the pancreas and approximately a year in ampullary carcinoma.

The operative mortality rate for the Whipple operation which was 30 to 40 per cent at first, now appears to be approximately 20 per cent when all cases are considered. The higher rates obtained early in the development of the operation probably were associated with its employment in some patients who now would be considered inoperable. With better selection of cases, values considerably less than 20 per cent have been reported by some surgeons. Operative mortality and survival rates are better when the operation is performed for ampullary carcinoma than they are when the procedure is done for carcinoma of the pancreatic head. In a series of eighty-five cases from the Mayo Clinic, the operative mortality rate for patients undergoing this radical operation for carcinoma of the ampulla was 13

islet cell tumor can be considered: symptoms of insulin shock must appear when the patient fasts the blood sugar at this time must decrease to 50 mg. or less per 100 ml. symptoms must be promptly relieved after ingestion of glucose.

When these conditions are satisfied, a diagnosis of spontaneous hypoglycemia can be made. Causes of hypoglycemia other than islet cell tumors should be sought before operation is carried out. The following diseases occasionally may be associated with varying degrees of hypoglycemia: hepatic disease, including cholangitis, cirrhosis, hepatitis, carcinomatosis or von Gierke's disease; endocrine disturbances, such as hypofunction of the pituitary or adrenal glands, hypothyroidism or hyperthyroidism; lesions of the central nervous system, and some neuroses. It may be difficult to decide whether mild symptoms in neurotic patients are functional or perhaps due to islet cell tumors. A few patients have been encountered who secretly administered insulin to themselves to produce hypoglycemia. This bizarre form of malingering has been detected in some instances only by searching the patient's hospital room and finding hidden syringes and insulin.

Once the diagnosis of islet cell tumor is made, surgical exploration is indicated. Delay should be avoided since it can permit mental deterioration to occur as a result of repeated attacks of hypoglycemia, a phenomenon that can become irreversible. Immediately before operation, intravenous admini-

stration of 1000 ml of a 5 or 10 per cent solution of glucose should be started and continued throughout the operation to avoid hypoglycemia. An upper transverse incision gives adequate exposure of the pancreas after the gastrosplenic omentum has been divided. In some patients a tumor may be readily visible, but in many others a careful search is needed. The tail and body are first mobilized so that the gland can be inspected and palpated on both its posterior and anterior surfaces. If no tumor is found here, the head of the pancreas is examined on both surfaces if necessary by reflection of the second part of the duodenum to the left and anteriorly after incision of the peritoneum lateral to it (Kocher's maneuver). A search should be made for accessory pancreatic tissue in such locations as the stomach, duodenum and gastrosplenic omentum, since heterotopic tissue sometimes can be the site of islet cell tumors.

If a tumor is found, it is excised. If a tumor is not found after a most careful search of the whole organ, the portion including the tail and body of the pancreas is removed, together with the spleen. Careful dissection of this specimen often reveals a tumor. If not, the resection may be continued, with removal of the remainder of the pancreas and the duodenum (Fig 9) assuming a tumor to be in the pancreatic head. However in most instances it is wiser to conclude the operation as a partial pancreatectomy in order to learn with certainty by the absence or continuation of symptoms whether sub-

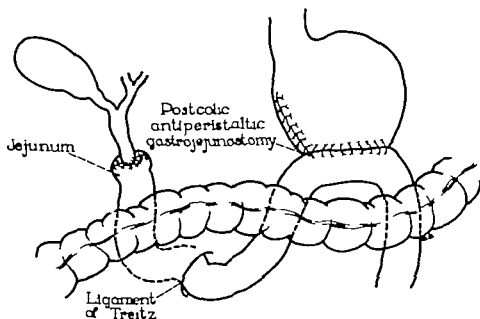


Figure 9 A method of reconstruction after total pancreatectomy and duodenectomy (Wang, J M; Surgical Diseases of the Pancreas. In Walters, W Lewis Practice of Surgery vol. 7 W F Prior Co.)

Although carcinoma of the body and tail of the pancreas should be most readily resected, rarely is it technically feasible because of the local extension. For example, resection was possible in only eight of 158 patients having adenocarcinoma of the body and tail of the pancreas operated on at the Mayo Clinic. The patient who survived the longest had recurrent adenocarcinoma two years later. In a group of ninety-eight patients followed after exploratory laparotomy and biopsy of the pancreas alone for this lesion, the average survival period after operation was three and a half months and the average survival from onset of symptoms to death was eleven months. We have been unable to find any mention in the literature of a patient alive and well without recurrence more than five years after resection of an unquestioned carcinoma of the body or tail of the pancreas.

Tumors of the Islets of Langerhans. The islets of Langerhans more numerous in the tail and body than in the head of the gland, are made up of cells, some of which elaborate insulin. Neoplasms originating from this tissue may be histologically benign, may be malignant and metastasizing or may appear malignant cytologically but demonstrate no tendency to metastasize. Furthermore, tumors in each of these groups may or may not be "functioning tumors" associated with hyperinsulinism.

Howard and his associates in 1950, presented a comprehensive review of hyperinsulinism and islet cell tumors of the pancreas and collected 398 tumors. Of the 224 tumors removed at operation, 211 were functioning and ten were nonfunctioning. In three instances, the status was unclear. Twenty four of the tumors found at operation proved to be malignant. Their study showed that 174 of these tumors were found at necropsy of which fifty three were functioning and ninety two were nonfunctioning, this point was undetermined in the remaining twenty nine. At necropsy thirteen proved to be malignant. It is understandable that the incidence of nonfunctioning tumors should be greater in lesions obtained at necropsy than in those obtained surgically since it is usually hypoglycemia which leads to the suspicion that such a tumor is present and prompts operation for it. About 5 per cent of functioning adenomas are multiple although these are usually in the same part of the gland, they can be widely separated. As mentioned earlier about 75 per cent of islet cell tumors originate in the body and

tail of the pancreas and 25 per cent are found in the head of the pancreas.

Nonfunctioning islet cell tumors and benign functioning adenomas are usually firm, grayish pink and fairly well delineated from normal pancreas, if not actually encapsulated. Adenomas which show cytologic evidence of malignancy usually are poorly encapsulated but otherwise are grossly similar to frankly benign growths. Malignant islet cell tumors are soft, brownish red, vascular, not encapsulated and may present metastasis. The usual size of the benign adenoma associated with hyperinsulinism is 1 cm. to 2 cm. Tumors smaller than 5 mm. in diameter rarely produce hyperinsulinism, and growths as large as 15 by 13 by 10 cm. have been reported. Whether benign or malignant, these tumors tend to present a histologic pattern somewhat resembling that of normal islets frequently presenting arrangements of cells in the form of islands, acini, cords, strands or ribbons according to Lopez Kruger and Dockerty. Functioning benign adenomas usually are associated with a longer history of symptoms than are functioning malignant tumors.

With the exception of the few cases of nonfunctioning malignant islet cell tumors in which the clinical picture is that of a metastasizing adenocarcinoma, the symptoms of islet cell tumors are those of hypoglycemia resulting from an excess of endogenous insulin. The symptoms of hypoglycemia are many and varied. Mild fatigue and apprehension associated with pronounced hunger sometimes are predominant. Frequently symptoms referable to the central nervous system are especially prominent. Kepler and Moersch have stated that any one of the following states may dominate the clinical picture: apathy, irritability, restlessness, fatigue, anxiety, incoordinability, negativism, automatic behavior, somnambulism, confusion, excitement, disorientation, drunken behavior, states of fugue, attacks of unconsciousness, delirium, mania, stupor and coma. It is not surprising that epilepsy, alcoholism or brain tumor frequently is suspected erroneously of being present. Because these patients often learn to ingest large amounts of carbohydrate for temporary relief of symptoms, they tend to become obese.

The diagnosis of hypoglycemia is made by determining the value for blood sugar at a time when symptoms suggestive of this disorder are present. Whipple has presented the following triad of findings which must be present before a diagnosis of functioning

- Comfort, M. W.: Serum Lipase: Its Diagnostic Value. *Am. J. Digest. Dis.* 3:817 1937.
- Comfort, M. W., Gambell, E. E., and Baggenstoss, A. H.: Chronic Relapsing Pancreatitis. A Study of Twenty-nine Cases without Associated Disease of the Biliary or Gastro-Intestinal Tract. *Gastroenterology* 6:239 376, 1918.
- Doubilet, H., and Mulholland, J. H.: Recurrent Acute Pancreatitis: Observations on Etiology and Surgical Treatment. *Ann. Surg.* 128:600, 1918.
- Dragstedt, L. R.: Some Physiologic Problems in Surgery of the Pancreas. *Ann. Surg.* 118:576 1913.
- Edmondson, H. A., and Berne, C. J.: Calcium Changes in Acute Pancreatic Necrosis. *Surg. Gynec. & Obst.* 79:240 1911.
- Ellison, E. H.: The Ulcerogenic Tumor of the Pancreas. *Surgery* 40:147 1958.
- Elman, R.: Variations of Blood Amylase during Acute Transient Disease of the Pancreas. *Ann. Surg.* 105:379, 1937.
- Gambell, E. E., Comfort, M. W., and Baggenstoss, A. H.: Chronic Relapsing Pancreatitis: Analysis of 27 Cases Associated with Disease of Biliary Tract. *Gastroenterology* 11:1 1948.
- Harper, A. A., and Raper, H. S.: Pancreozymin, a Stimulant of the Secretion of Pancreatic Enzymes in Extracts of the Small Intestine. *J. Physiol.* 102:115, 1943.
- Howard, J. M., Moss, N. H., and Rhoads, J. E.: Hyperinsulinism and Islet Cell Tumors of the Pancreas; with 398 Recorded Tumors. *Internat. Abstr. Surg.* 90:417 1950.
- Kepler, E. J., and Moench, F. P.: The Psychiatric Manifestations of Hypoglycemia. *Ann. J. Psychiat.* 94:99 1937.
- Kibler, C. E., and Bernatz, P. E.: Operative Experience with Carcinoma of the Body and Tail of the Pancreas. *Proc. Staff Meet. Mayo Clin.* 33:247 1958.
- Lopez Kruger, R., and Dockerty, M. B.: Tumors of the Islets of Langerhans. *Surg. Gynec. & Obst.* 85:495, 1947.
- McGaughey, R. B., Hallenbeck, G. A., Bollman, J. L., and Grindlay, J. H.: Ductal and Vascular Factors in Etiology of Experimentally Induced Acute Pancreatitis. *Arch. Surg.* 74:881 1957.
- Puestow, C. B., and Gillesby, W. J.: Retrograde Surgical Drainage of Pancreas for Chronic Relapsing Pancreatitis. *Arch. Surg.* 78:808, 1958.
- Snell, A. M., and Comfort, M. W.: The Incidence and Diagnosis of Pancreatic Lithiasis: Review of Eighteen Cases. *Am. J. Digest. Dis.* 8:237 1941.
- Thigpen, F. M.: A Pathologic Study of Cysts of the Pancreas. Thesis, Mayo Foundation, Graduate School, University of Minnesota, 1940.
- Thomas, J. E.: The External Secretion of the Pancreas. Springfield, Ill., Charles C Thomas, 1930 149 pp.
- Waugh, J. M., and Giberson, R. G.: Radical Resection of the Head of the Pancreas and of the Duodenum for Malignant Lesions: Some Factors in Operative Technique and Preoperative and Postoperative Care with an Analysis of 85 Cases. *S. Clin. North America*, August, 1957 pp. 965-979.
- Whipple, A. O., Parsons, W. B., and Mullins, C. R.: Treatment of Carcinoma of the Ampulla of Vater. *Ann. Surg.* 102:763, 1935.
- Wollaefer, E. E., Comfort, M. W., Clagett, O. T., and Osterberg, A. E.: Efficiency of Gastrointestinal Tract after Resection of Head of Pancreas. *J.A.M.A.* 137:838, 1948.
- Zimmermann, B.: Endocrine Functions of the Pancreas. Springfield, Ill., Charles C Thomas, 1962, 82 pp.
- Zollinger, R. M., and Ellison, E. H.: Primary Peptic Ulcerations of the Jejunum Associated with Islet Cell Tumors of the Pancreas. *Ann. Surg.* 142:709 1955.

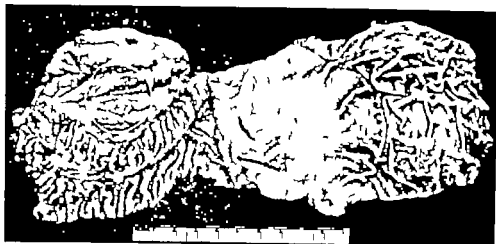


Figure 10 Small islet cell tumor of the head of the pancreas necessitating total pancreatectomy. The patient in satisfactory condition ten years after operation (Waugh, J. M. *Surgical Diseases of the Pancreas*. In Walters, W.; Lewis' *Practice of Surgery* vol. 7. W. F. Prior Co.)

sequent radical total pancreatectomy (Fig. 10) is necessary. Many patients in whom no tumor is demonstrated (46 per cent of fifty-six such patients in a series reported by Howard and associates) have satisfactory results after partial pancreatectomy. Symptoms are regularly relieved when adenomas are removed.

Islet Cell Tumors Associated with Peptic Ulcer. In 1955 Zollinger and Ellison described two patients with such intractable peptic ulcers that each underwent a series of operations culminating in total gastrectomy. In each case, tumors of the islets of Langerhans which did not cause hypoglycemia were finally found. The following year Ellison summarized the cases of twenty-four such patients collected from his own experience and from the literature. Of twenty-one patients whose sex was known, thirteen were women and eight were men. Their ages varied from nineteen to seventy-eight years. In nineteen cases the tumors were malignant and in five they were benign. In fourteen cases they were single and in ten they were multiple. In eleven of the patients with malignant disease, the tumors had metastasized from the pancreas at the time of recognition. Five patients had tumors of other endocrine glands as well. Hypoglycemia was present at some stage of the disease in only three patients.

In general, the ulcer diathesis accompanying these tumors was a fulminating one accompanied by excessive gastric hypersecretion not controlled by the usual medical or surgical measures. Ulcers often were located atypically in the esophagus, in the distal part of the duodenum or even in the jejunum. The tumors were discovered only at nec-

ropsy in nine patients who had undergone twenty-three operations for peptic ulcer and had finally died of some complication of an ulcer after an average of seventeen months. Tumors were removed at operation in eleven patients. Of these, three died soon thereafter of ulcer and were found still to have islet tumors. Three more died later of metastatic carcinoma, five were living at the time the report was made.

The assumption has been made that these islet cell tumors are in some way ulcerogenic. It has been postulated that they elaborate a humoral agent capable of stimulating gastric hypersecretion. An early suggestion that glucagon might be this agent remains unproved. In Ellison's words,

It seems reasonable, therefore, that the surgeon faced with the problem of controlling recurrent marginal ulceration or an atypically located primary ulcer in patients with known hypersecretion should not overlook the possibility that an islet-cell tumor of the pancreas may be the causative factor. In these instances, a careful search for individual tumors or even resection of the body and tail of the pancreas might best be considered before subjecting the patient to total gastrectomy as a final, heroic means of controlling the acid factor.

Parenthetically the intense fibrous tissue reaction which frequently surrounds gastrojejunal ulcers often lies in the region of the pancreas and can make accurate palpation of the pancreas for small tumors very difficult.

READING REFERENCES

- Beyliss, W. M., and Starling, E. H. *The Mechanism of Pancreatic Secretion*. J. Physiol. 28:323, 1902.
Cattell, R. B., and Warren, L. W. *Surgery of the Pancreas*. Philadelphia, W. B. Saunders Company 1963, 374 pp.

derived from the phrenic artery the renal artery and the aorta. The lymphatics drain eventually into thoracic lymph nodes. Only the medulla receives a nerve supply derived primarily from the splanchnic plexus of medullated preganglionic fibers.

The cortex exhibits from the periphery inward three distinct areas: the zona glomerulosa, the zona fasciculata and the zona reticularis. There is evidence that these divisions of the cortex may be functional as well as structural. The glomerulosa may be considered as concerned with electrolyte exchange, the fasciculata with organic metabolism and the reticularis with androgen and estrogen effects. The medulla is structurally uniform and functionally elaborates two hormones: epinephrine and norepinephrine. The former comprises normally 75 to 80 per cent of the secretion and causes an increase of cardiac output, whereas the latter exerts a pressor effect by increasing peripheral resistance by vasoconstriction.

During the fourth week of embryonal life mesothelial buds are found near the upper mesonephron which ultimately coalesce to form the adrenal cortex. Cortical buds not joining the main cellular mass may survive and exist as accessory adrenal cortical tissue in the region of the adrenal, kidney, spermatic cord, testis, broad ligament or ovary. The ectodermal medulla is derived from cells of the sympathetic ganglia which develop into chromaphil cells from pheochromoblasts. These sympathochromaffin cells become surrounded by the adrenal cortical mass to form the medulla at about the seventh week of fetal life. Sympathetic ganglia along the aorta, at the aortic bifurcation and the carotid bifurcation are derived from sympathoblasts which may give rise to tumors similar to those of the adrenal medulla.

By the fourth month of intrauterine life, the adrenals exceed the kidneys in size and at birth they are one-third the size of the kidneys. During the first three weeks of postnatal life, the adrenal loses one-half of its size owing to degeneration of the fetal cortex and little change occurs thereafter until puberty. In adult life the usual ratio of adrenal to kidney is about 1:30. Although it has been recently demonstrated that accessory adrenal cortical bodies occur quite frequently in infants, and may persist in adults, they seldom are of clinical significance. These accessory bodies seldom produce tumors and usually cannot be detected by ACTH stimulation after total adrenalectomy.

The anomalies of the adrenal of impor-

tance in surgery are unilateral agenesis, heterotopia (beneath renal capsule), accessory adrenal tissue, benign adrenal cysts and congenital bilateral adrenal cortical hyperplasia giving rise to abnormal sexual development.

The adrenal glands bilaterally share a common inaccessibility to surgical approach. Each gland is more independent of the kidney than has been commonly emphasized, though each lies in a superior extension of the perinephric fascia. Each lies superior and medial to the upper pole of the corresponding kidney with multiple fascial and vascular attachments to surrounding adipose tissue. The basal surface is often concave and a superior convexity extends upward along the aorta on the left and the vena cava on the right. The approach is more difficult in obese and heavily muscled subjects. Exposure is always easier in the female patient than in the male because of her less well developed musculoskeletal structures.

The arterial blood supply is derived from the renal artery inferiorly from the aorta medially and one or more branches from the phrenic artery superiorly. Blood from the cortex passes into capillary networks which empty into the well-developed venous system of the medulla. The veins correspond in general to the arterial supply. The most difficult vessel to secure is the large but short vein leaving the midportion of the gland medially and directly entering the vena cava on the right, on the left it is longer and easier to expose. The important vessels are secured by clamp and long ligatures, preferably of silk because of the depth of the wound and its low coefficient of friction.

Four different surgical approaches to the adrenals have been used and the route chosen is determined according to the problem and physical characteristics of the individual patient.

Cahill has advocated the transabdominal approach because it has the advantage of simultaneous bilateral exploration through a single incision. It has the disadvantage of requiring transperitoneal dissection and displacement of viscera and of being a more distant as well as less direct approach to the adrenal than is the transcostal approach. It is better adapted to thin patients (especially those with pheochromocytoma) and offers good opportunity for exploration for ectopic adrenal tissue as well as adnexal exploration in women.

Excellent exposure is accomplished by the transthoracic approach, but this carries the

THE ADRENAL GLANDS

By J HARTWELL HARRISON M D., and
ROBERT E DESAUTELS M D

JOHN HARTWELL HARRISON was educated at The University, Virginia and is not modest about the identification of The University Harvard Medical School and the Peter Bent Brigham Hospital provided the broad surgical background upon which his urologic interests rest

ROBERT EUGENE DESAUTELS, a Vermont Yankee was educated at Harvard College and Medical School. A musician by avocation, he is actively engaged in laboratory investigations in the field of urology

This subject in recent years has attained an increased importance which may be largely attributed to the work of Reichstein and of Kendall respectively each of whom separately isolated, identified and synthesized crystalline adrenal cortical steroids more than a decade ago. Since these advances many intensive studies of adrenal cortical physiology have been made and directly resulted in the development of replacement therapy adequate to support life even in the total absence of the adrenal glands. This adequate substitution therapy has greatly enlarged the scope of adrenal surgery and has altered the prognosis of patients having adrenal cortical insufficiency who require surgical intervention of any sort. The physician today can approach the problems of diagnosis and treatment of disorders of the adrenal glands with a confidence not possible ten years ago.

SURGICAL ANATOMY AND EMBRYOLOGY OF THE ADRENAL GLANDS

The adrenal glands are normally somewhat darker yellow in color and of firmer consistency than is the surrounding peri-

nephric fat. They lie in a superior extension of the perinephric fascia. Their size varies considerably and is influenced by the physiologic state of the individual. Adrenal weight in previously healthy adults dying of accidental causes ranged from 5.8 to 11.3 gm. (per pair) with an average value of 8.3 gm. whereas the weight of normal adrenals removed surgically from patients having hypertension or cancer averages 5 to 7 gm. each (10 to 14 gm. per pair) which shows an expected contrast to the post-mortem weight largely due to the great vascularity of these important endocrines. In shape each has a somewhat conical center having ribbon-like superior and inferior extensions which vary in thickness according to the state of activity and circulation at the time of observation.

These glands are divided anatomically and physiologically into cortex and medulla. In contrast to the yellow cortex, the medulla is brown and develops a very dark brown upon staining with chromates. It is completely surrounded by the cortex. Selve has estimated that the flow of blood through the adrenals is at the rate of 6 to 7 cc. per gm. of tissue per minute. The blood is de-

as a possible cause of the condition since the secretion of ACTH is subject to neuro-hormonal stimuli from the hypothalamus.

The excessive production of glucocorticoids by the adrenal cortex results in a protein catabolic effect which leads to protein depletion and diminution of the muscular mass of the individual. The thin delicate skin and capillary fragility which is manifested by easy bruisability as well as striae are characteristic. Osteoporosis with loss of strength of bones and pathologic fractures is frequent. The accelerated protein breakdown results in increased gluconeogenesis and diminished carbohydrate tolerance. Insulin-resistant diabetes results from these processes. Accompanying the excessive protein breakdown is an excessive deposition of fat with abnormal distribution involving especially the face, back and trunk. The cervicodorsal fat pad has been characterized as a buffalo hump.

The anterior pituitary gland may show cytologic abnormalities characterized by a basophilic cytoplasmic hyalinization, vacu-

olization and degranulation. A basophilic tumor is present in one-third of the subjects. Less frequently other types of pituitary tumor are encountered. The question is still unanswered in many cases as to whether the changes in the pituitary are primary or secondary to those in the adrenal cortex. In about 30 per cent of the patients, an adrenocortical tumor is present and at least one-half of these neoplasms prove to be malignant. Unilateral hyperfunctioning tumors are often associated with diminished size and function of the contralateral gland. A small percentage of patients exhibit no structural abnormality of the adrenal glands grossly or histologically. However at operation the glands always appear enlarged and physiologically hyperactive with an excessive degree of vascularity. When the lesion is due to adrenocortical hyperplasia, the glands at operation are found to be enlarged and to have a dark brown color instead of the normal canary yellow color of the adrenal. Small adenomas may be found in the cortex associated with these changes. Histologically

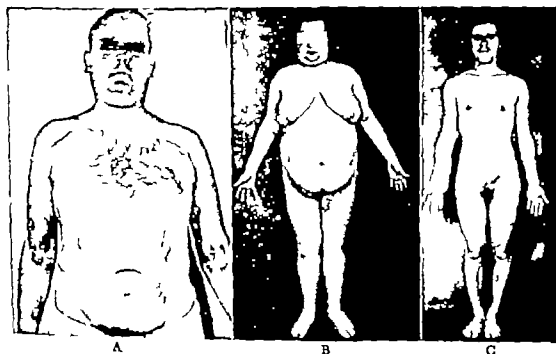


Figure 1 A, Pure Cushing's syndrome in a man, thirty years of age, exhibiting obesity, striae, bruisability, moon face, osteoporosis, weakness, impotence and loss of muscle due to hyperplasia of the adrenal cortex and corrected by bilateral adrenalectomy.

B, Example of intergrade between Cushing's syndrome and virilism in a woman, aged thirty years, having hirsutism, amenorrhea, hypertension, great muscularity, hypertrophy of clitoris, diabetes and high excretion of androsterone (courtesy of Dr. Harry Friedgood) due to carcinoma of the adrenal cortex.

C, The ultimate extreme of virilism in a female, aged twenty-two years, shown by bodily configuration, muscular development and hirsutism. Amenorrhea, hypertrophy of the clitoris and a markedly elevated 17-ketosteroid excretion characterized this patient's state of virilism caused by adrenal cortical hyperplasia involving primarily the zona reticularis (Harrison, J. H., and Laidlaw, J. C., S. Forum, vol. 4).

limitation of a unilateral operation at that session. It is indicated only in the case of very large tumors.

The bilateral posterior transcostal route with the patient flexed at the trunk in the prone position permits exploration of each gland simultaneously with minimal disturbance of other viscera. Subperiosteal resection of the eleventh and twelfth ribs is necessary on the right for adequate exposure and also is often desirable on the left. The approach is extrapleural and subdiaphragmatic; the kidney on each side being displaced inferiorly. This approach is of particular value when an adrenal tumor is suspected but has not been localized preoperatively, and when bilateral adrenalectomy is being carried out, especially in a thin patient. The posterior approach, though most direct, has the limitation of a small operative field with restricted visualization demanding two to three retractors in the wound.

The posterolateral approach affords a wide field of operation, with direct access to the suprarenal region on one side at a time and is extremely useful when an adrenal tumor has been accurately localized prior to operation or when the patient is too large or obese for an approach via the posterior route.

The anatomic relations are the guiding features in surgery of the adrenal when it is being performed for hyperplasia, neoplasm, cyst or for removal of the normal gland. Variations in blood supply must be expected, especially when a tumor is present, and increased vascularity as well when hyperplasia is present.

CLINICAL PATTERNS OF HYPERADRENOCORTICISM

The pathologic states apparently mediated by hyperfunction of the adrenal cortex present a wide variety of conditions which have been classified clinically into Cushing's syndrome at one extreme and the adrenogenital syndrome at the other extreme. Between these two extremes are seen varying examples of the mixed syndrome exhibiting features of both, in which one or the other predominates according to whether the glucocorticoids (zona fasciculata) or the sex hormones (zona reticularis) are being produced in excess, but in which both are participating to an abnormal degree (Fig. 1).

Cushing's Syndrome. Cushing's syndrome, properly defined, refers to the clinical picture described in 1932 by Harvey Cushing and known as pituitary basophilism. The exact cause of this condition is unknown.

However the clinical manifestations are those of hyperadrenocorticism. An antecedent history of prolonged periods of stress, worry and physical hardship has been noted in some of these subjects. It has been the impression of some physicians that this disease is on the increase. The disorder has been comparatively rare; it occurs most frequently in young adults and is three to five times more common in females than in males. Cushing attributed the disease to the basophil adenoma of the anterior pituitary gland. He also noted the frequent association of basophil tumors of the pituitary and either hyperplasia or tumor of the adrenal cortex.

It has been established that adrenocorticotrophic hormone secreted by the anterior pituitary gland controls the function and structure of the normal adrenal cortex. It is believed by some that ACTH is secreted by basophilic cells which indicates that perhaps abnormalities of the basophil function are involved in initiating disorders of the adrenal cortex. It is significant that certain patients with Cushing's syndrome have a remission following pituitary irradiation and the remission has been sustained in approximately one-sixth of these.

In one instance three years after total adrenalectomy for hyperplasia causing Cushing's syndrome a huge chromophobe adenoma of the pituitary became apparent and was surgically removed. High blood levels of ACTH became normal after hypophysectomy and deep tanning of the skin disappeared. This case concretely illustrates pituitary-adrenal relations in Cushing's syndrome.

Kepler and others have suggested that adrenocortical hyperfunction is the initial event. This is supported by the numerous instances in which adrenocortical hyperplasia or tumor occurs without a demonstrable basophilic adenoma. The recent clinical and pathologic studies of Heinbockel furnish further evidence that the primary disorder arises in the adrenal cortex and may cause the changes in the pituitary gland described by Cushing. There is no doubt that the clinical picture is mediated by the adrenal cortex, since removal of this hyperfunctioning tissue has usually resulted in a reversal of the clinical picture. The intensive administration of 11-17 oxygenated cortical steroids does reproduce the symptoms and signs exhibited by patients with Cushing's syndrome as well as induce pituitary changes. The hypothalamus has been under suspicion

as a possible cause of the condition since the secretion of ACTH is subject to neuro-hormonal stimuli from the hypothalamus.

The excessive production of glucocorticoids by the adrenal cortex results in a protein catabolic effect which leads to protein depletion and diminution of the muscular mass of the individual. The thin delicate skin and capillary fragility which is manifested by easy bruisability as well as striae are characteristic. Osteoporosis with loss of strength of bones and pathologic fractures is frequent. The accelerated protein breakdown results in increased gluconeogenesis and diminished carbohydrate tolerance. Insulin-resistant diabetes results from these processes. Accompanying the excessive protein breakdown is an excessive deposition of fat with abnormal distribution involving especially the face, back and trunk. The cervicodorsal fat pad has been characterized as a buffalo hump.

The anterior pituitary gland may show cytologic abnormalities characterized by a basophilic cytoplasmic hyalinization vacu-

olization and degranulation. A basophilic tumor is present in one third of the subjects. Less frequently other types of pituitary tumor are encountered. The question is still unanswered in many cases as to whether the changes in the pituitary are primary or secondary to those in the adrenal cortex. In about 30 per cent of the patients, an adrenocortical tumor is present and at least one-half of these neoplasms prove to be malignant. Unilateral hyperfunctioning tumors are often associated with diminished size and function of the contralateral gland. A small percent age of patients exhibit no structural abnormality of the adrenal glands grossly or histologically. However at operation the glands always appear enlarged and physiologically hyperactive with an excessive degree of vascularity. When the lesion is due to adrenocortical hyperplasia, the glands at operation are found to be enlarged and to have a dark brown color instead of the normal canary yellow color of the adrenal. Small adenomas may be found in the cortex associated with these changes. Histologically

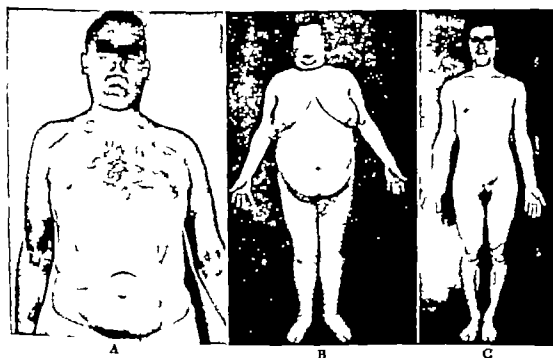


Figure 1 A, Pure Cushing's syndrome in a man, thirty years of age, exhibiting obesity, striae, bruisability, moon face, osteoporosis, weakness, impotence and loss of muscle due to hyperplasia of the adrenal cortex and corrected by bilateral adrenalectomy.

B, Example of intergrade between Cushing's syndrome and virilism in a woman, aged thirty years, having virilism, amenorrhea, hypertension, great muscularity, hypertrophy of clitoris, diabetes and high excretion of androsteroes (courtesy of Dr. Harry Friedgood) due to carcinoma of the adrenal cortex.

C, The ultimate extreme of virilism in a female, aged twenty-two years, shown by bodily configuration, muscular development and hirsutism. Amenorrhea, hypertrophy of the clitoris and a markedly elevated 17-ketosteroid excretion characterized this patient's state of virilism caused by adrenal cortical hyperplasia involving primarily the zona reticularis (Harrison, J. H., and Laidlaw, J. C. S. Forum, vol. 4)

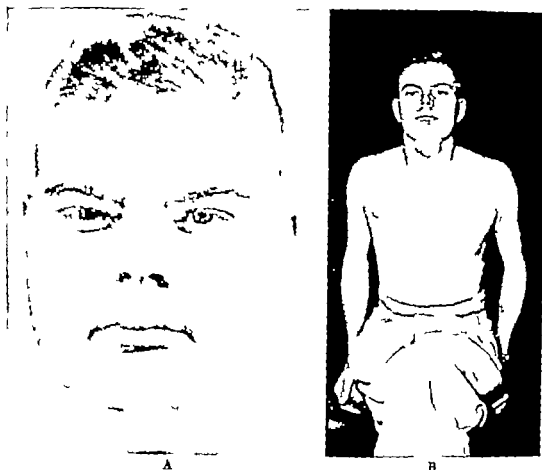


Figure 2. A, A man, aged twenty-two years, having a moon face, obesity striae, hypertension and weakness due to adrenocortical hyperplasia which was treated by bilateral total adrenalectomy. B, This shows the striking change and improvement in the patient five months after adrenalectomy (Courtesy of Dr G. W. Thorn.)

usually one finds hyperplasia of the zona fasciculata when the clinical condition is pure Cushing's syndrome. Unilateral adrenocortical hyperplasia is rarely seen.

Other associated changes which have been reported are carcinoma of the pancreas, cysts of the pancreas, pancreatitis, infarcts of the pancreas, fatty infiltration of the liver, nephrosclerosis and renal calcinosis, and 80 per cent of the patients have a marked degree of osteoporosis. Generalized arteriosclerosis and cardiac hypertrophy are also found. Atrophy of the ovaries in the female patients and diminution in size and softening of the testes in men are common.

Obesity is a common feature of the disease in both sexes. It is truncal in distribution and the cervical dorsal fat pad, or buffalo hump, is characteristic. Thinning of the skin with multiple bruises as a result of increased capillary fragility is common. Abdominal and thoracic striae are common to both sexes. An intensely reddened countenance is frequently observed and hypertension, muscular weakness and easy fatigability are out-

standing symptoms (Fig. 2). Erythremia, glycosuria and osteoporosis are frequent concomitants. Polyphagia, polydipsia and polyuria are accompaniments of insulin-resistant diabetes. Edema of the lower extremities and albuminuria often are a reflection of an accompanying degree of nephritis. Amenorrhea in females and impotence in males are common. Hirsutism occurs in the majority of women affected, but true virilization does not occur with the primary hyperglucocorticoid state (Fig. 3). Profound personality changes occur with increased irritability in some progressing at times to a major psychosis usually of the depressive type. Acne is commonly found and patchy cutaneous pigmentation may occur. The skin is surprisingly delicate and easily injured. There is a greatly increased susceptibility to infections of all types. Wound healing has been thought to be delayed, but these wounds which are surgically performed seem to heal *per primum* when asepsis is strict.

The laboratory findings in Cushing's syndrome may be listed as follows: decreased

glucose tolerance, hyperglycemia and glycosuria, a hematologic picture characterized by eosinopenia, lymphopenia, leukocytosis and erythremia increased urinary corticosteroid excretion (17 hydroxycorticoids) normal or elevated 17 ketosteroid excretion decreased serum chloride and potassium concentration, increased serum carbon dioxide content osteoporosis demonstrated by roentgenograms of the skeleton, albuminuria. When the underlying pathologic lesion is carcinoma of the adrenal cortex, the 17 ketosteroids are usually elevated, especially the beta fraction.

Adrenogenital Syndrome (Virilism) This form of hyperadrenocorticism is found at the other extreme of the spectrum from the hyperglucocorticoid state of Cushing's syndrome. The condition is attributed to excessive production of androgens by the adrenal cortex. Between these two extremes are found various intergrades with varying clinical manifestations, according to the degree of involvement of the zona fasciculata and zona reticularis by the enhanced physiologic processes. As a consequence of the virilizing properties of the hormones elaborated, there may result female pseudohermaphroditism at birth, precocious puberty in the preadolescent male and heterosexual development in the preadolescent and adult female. In

addition to their androgenicity these hormones have a markedly anabolic effect on protein metabolism. Thus patients with pure Cushing's syndrome present evidence of protein depletion and loss of muscle mass, whereas those with a pure adrenogenital syndrome show signs of protein conservation and increased muscle mass.

According to Wilkins, postnatal adrenal virilism in females prior to puberty is almost without exception due to adrenal cortical tumor. Virilization may begin during the first few months of life and progresses rapidly. Pubic and axillary hair develops and there is hypertrophy of the clitoris. In the male, there is a picture of macrogenitosomia praecox. Rapid growth of muscle and skeleton is characteristic. Varying degrees of obesity may be seen along with the increased muscularity according to the amount of activity of the zona fasciculata. Very high levels of 17-ketosteroid excretion are seen. The differential diagnostic problem is with regard to whether or not there is congenital adrenal cortical hyperplasia or tumor. With the former there is a typical response to the administration of cortisone resulting in inhibition of the adrenocortical activity and an improvement in the clinical condition.

The adrenogenital syndrome usually be-



Figure 3. A, A woman, aged twenty-three years, six months after the development of moon face and other early signs of Cushing's syndrome due to an adrenocortical adenoma on the left side. B Photograph of patient six months after surgical removal of adenoma of the adrenal cortex.

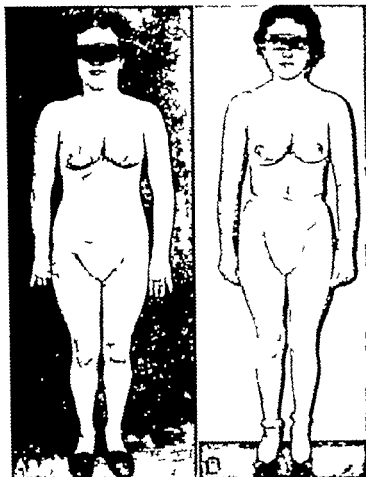


Figure 4 A, Virilism in a twenty-eight year-old female characterized by muscular development and hirsutism from the age of eleven years and amenorrhea from the age of eighteen, due to tumor of the adrenal cortex classified as carcinoma histologically and reacting as such physiologically by failure to respond to ACTH. B, Striking decrease in hirsutism and appearance of more feminine contours three months after removal of adrenal cortical carcinoma from this patient. Menses became regular and the patient continued to be well ten years later. A solitary left pulmonary metastasis was removed seven years after primary adrenal carcinoma was removed. This metastasis caused no endocrine or clinical disturbance though it showed the same histologic features as the original tumor.

gins at ages varying from adolescence to before the menopause. The majority of cases are probably due to tumor but adrenocortical hyperplasia, especially involving the zona reticularis, may be the cause (Fig. 1 C). The development of secondary masculine characteristics due to increased androgens and the suppression of feminine characteristics occur simultaneously. Hirsutism involves the face, extremities and trunk. The hair of the scalp may be thin and occasionally one sees not only temporal recession but actual baldness. Irregularity of the menses which become scanty and ultimate cessation of the menses occur in the majority of patients. Libido is diminished, hypertrophy of the clitoris and simultaneous diminution in the size of the breasts, ovaries and uterus occur. The breasts become more masculine in contour. Deepening of the voice, extreme degrees of muscular develop-

ment and male configuration are striking. The condition differs from Cushing's syndrome not only in the metabolic alterations observed, but also in the natural course of the disease which in the case of virilism may occur over a period of many years (Fig. 4). The changes in Cushing's syndrome usually come about within a period of about two to three years.

In the adrenogenital syndrome, the pituitary gland does not usually show the changes found in Cushing's syndrome. Also the pathologic changes in the liver, kidney, pancreas, muscle, bone and reticuloendothelial system are absent in pure virilism, but these changes may be present in the various intergrades between the two extremes of the spectrum.

Patients having the mixed syndrome exhibit the characteristics of virilism shown by masculinization which usually predominates in the picture, as well as the metabolic ab-

normalities of Cushing's syndrome. These mixed pictures may be produced by adrenocortical hyperplasia perhaps more often than by tumor though the latter may also be productive of this bizarre complex picture. Patients having the mixed syndrome will exhibit hirsutism, amenorrhea, hypertrophy of the clitoris, hypertension, obesity masculine extremities with increased muscular development, plethora, seborrheic dermatitis, glycosuria, hyperglycemia, cutaneous striae, erythremia and extremes of personality change. Patients with virilism show very high levels of urinary 17 ketosteroids, ranging from 25 to 90 mg per twenty four hours. When carcinoma is present, there is a high level of the beta fraction of 17 ketosteroids. There may be no elevation of 17 hydroxy corticoids but in those having the mixed syndrome this will be present. High levels of androgens measured biologically can be recovered in the urine of patients with virilism.

The various causes of virilization in women must be considered in the differential diagnosis. The ovarian lesions which may produce virilism are arrhenoblastoma, Leydig cell tumor, diffuse luteinization or hyperthecosis, and polycystic disease of the ovaries (Stein-Leventhal syndrome). Palpation of a pelvic mass directs attention to the ovary but, unfortunately the tumors are frequently too small to be so detected. Leiomyomas of the uterus which are incidental must also be differentiated. A small number of patients with so-called adrenal rest tumors of the ovary have been described. Young emphasized the necessity of considering ovarian tumors in the differential diagnosis of virilism and recommended that preliminary pelvic exploration be carried out prior to adrenal surgery when no localizing evidence of an adrenal lesion could be demonstrated. When the lesion is primary in the ovary an excessive response to ACTH is not to be expected in the steroids excreted in the urine. At pelvic exploration, careful examination of the adrenal area should be carried out. However not finding pathologic change under these circumstances is not necessarily significant because intra abdominal palpation of the adrenals may be unsatisfactory. Fairly large tumors may not be apparent when intra abdominal palpation via a pelvic laparotomy exposure is carried out. Definitive suprarenal exploration is necessary for accuracy.

Primary Aldosteronism. A new clinical syndrome has been recently described by Conn, designated primary aldosteronism,

which causes a biochemical state of hypokalemic alkalosis. Relatively few cases have been reported in the literature, but an increasing number of instances of hyperaldosteronism, both primary and secondary, are gradually being discovered.

Conn has described the clinical picture as being characterized by intermittent tetany, paresthesia, periodic severe muscular weakness and paralysis, polyuria, polydipsia, hypertension and no edema. In its fully developed state, the condition exhibits excessive amounts of a sodium-retaining corticoid in the urine, severe hypokalemia, hypernatremia, alkalosis and a renal tubular defect in the reabsorption of water. There is no increase of excretion of 17 ketosteroids or of 17 hydroxycorticoids. The relative lack of important symptomatology at extremely low levels of serum potassium has been emphasized.

Operation upon the first patient described as having this condition was performed by Baum who found a well-encapsulated adenoma of the adrenal cortex 4 cm. in diameter. The cells of this tumor were laden with lipid material and bioassays of the tumor tissue gave values for aldosterone seventy-five to one hundred times greater per gram than that found in beef adrenal. The opposite adrenal gland showed thinning of the cortex and the atrophy was confined to the zona fasciculata. There was no atrophy of the zona glomerulosa. Chemical analysis of muscle biopsy specimens showed a great excess of intracellular sodium and marked decrease in the amount of intracellular potassium. Renal biopsies showed severe arteriosclerosis in all sections and a diffuse vacuolar change in the tubular epithelium thought to be hydropic degeneration. Areas of necrosis and also renal calcinosis were observed. This patient was only thirty-four years of age and the severe arteriolar lesions were attributed to excessive activity of the sodium-retaining steroid aldosterone.

Within one day following removal of this adenoma, the urinary excretion of sodium increased greatly and a marked retention of potassium occurred. Hypernatremia and hypokalemia disappeared by the sixth postoperative day and serum values for sodium and potassium were normal thereafter. Proteinuria, polyuria and polydipsia disappeared in six days. Blood pressure fell from 170/100 to a level of 120/70 in eighteen days. Bioassays for urinary sodium-retaining corticoids after operation gave normal values. The patient became completely symptom free. It is of especial interest that though

this lesion seemed to cause atrophy of the contralateral adrenal in the zona fasciculata, it did not cause any rise in 17 hydroxycorticoids and 17 ketosteroids which were also normal both before and after operation.

This syndrome of primary aldosteronism has been clearly established by Conn. Within six weeks of his initial report, four additional cases typical of this condition were recognized by others and in each of the patients an adrenal cortical adenoma was found. In another patient, the finding of an adenoma was made at autopsy. Wyngaarden reported one patient in whom no adenoma was found at autopsy though clinically the individual had all the characteristics of this syndrome. This suggests that the syndrome can occur without adrenal tumor and that bilateral adrenalectomy should be considered as therapy in such subjects. Conn very appropriately suggests that, since the bioassay for urinary aldosterone is impractical for broad clinical use, serum bicarbonate and potassium levels be determined routinely as a screening procedure on all hypertensive patients. Hypokaliemic alkalosis, unexplained by other conditions, should be regarded as an indication for careful adrenocortical study and exploration.

DIFFERENTIAL DIAGNOSIS AS TO CAUSE OF HYPERADRENOCORTICISM

In the differential diagnosis of these states one is presented with a varying history and

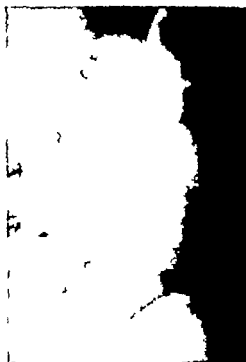


Figure 5. Plain film of the abdomen showing the shadow of a round tumor above the kidney in a man, aged forty years, having essential hypertension. At exploration this proved to be a large cyst of the adrenal gland with accompanying cortical atrophy on that side. Normal adrenocortical activity was found in both preoperative and postoperative studies.

findings, depending upon whether one is dealing with Cushing's syndrome virilism or the mixed syndrome with its bizarre manifestations. The consideration of major importance is whether the disease is being



Figure 6. Tomogram in conjunction with excretory urograms in obese women with virilism. The renal and adrenal areas are clearly demarcated showing no evidence of tumor and illustrating excellent detail without contrast instillation.

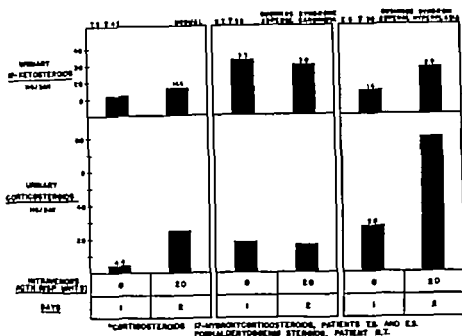


Figure 7 Urinary steroid response to the eight hour intravenous infusion of ACTH in the normal patient, in a patient with Cushing's syndrome due to adrenal cortical hyperplasia and in a patient with virilism due to adrenal carcinoma. The lack of response in the patient with carcinoma indicates the autonomous nature of the tumor. The hyperactive response in the patient with hyperplasia in contrast to the response in the normal patient and in the patient having carcinoma is significant in the differential diagnosis (Harrison, J. H. and Lakdaw J. C. S. Forum, vol. 4)

mediated by bilateral hyperplasia of the adrenal cortex, carcinoma of the adrenal cortex, adenoma of the adrenal cortex or a primary tumor of the ovary. A plain film of the abdomen (Fig. 5) and both excretory urography (Fig. 6) and retrograde pyelography may yield valuable information. Aortography is seldom necessary. Carbon dioxide injection into the retroperitoneal presacral area has replaced the use of oxygen because of its greater solubility and therefore lesser hazard of gas embolism. The limitations of the method must be recognized and it must be remembered that frequently masses of fat in the perinephric area have been mistaken for adrenal tumors. As high an error as 50 per cent has been reported with this latter technique alone. All of the diagnostic methods at one's disposal must be brought to bear in order to reach an accurate preoperative diagnosis.

The normal 17 ketosteroid excretion in the female ranges from 4 to 10 mg. per twenty four hours. The normal 17-hydroxycorticoid excretion ranges from 4 to 6 mg. per twenty four hours. With 25 clinical units of ACTH administered intravenously over a period of eight hours, the 17 ketosteroid excretion normally can be expected to rise to about 16 to 18 mg. and the 17 hydroxycorticoids can be expected to rise from the normal level up to 25 mg. (Fig. 7) When bilateral

adrenal cortical hyperplasia is present, resting levels of steroid excretion are high and an excessive rise in steroid excretion is expected as a result of ACTH stimulation. A level of 17 ketosteroid excretion may be from 30 to 60 mg. in twenty four hours, and the elevated excretion of 17 hydroxycorticoids, when hyperplasia is present, may be a resting level of 20 to 30. Following administration of 25 clinical units of ACTH, the 17 hydroxycorticoid level may rise to 70 or even over 100 mg. Likewise, when adrenal cortical hyperplasia is mediating the disease process under consideration, Wilkins has shown that by administering 100 mg. of cortisone acetate per day a decrease of the excessive levels of corticoids in the urine can be produced (Fig. 8) presumably as a result of an inhibitory controlling effect by the pituitary on the hyperplastic glands. When adrenal cortical carcinoma is producing the clinical picture one usually does not get a response as a result of stimulation with ACTH, and inhibition with cortisone administration does not occur. There have been a few exceptions to this, but, in general, these tests have served as a valuable differential diagnostic measure. When the clinical picture is due to adenoma of the adrenal cortex, a variable response may be obtained as a result of ACTH stimulation or cortisone inhibition. Failure to respond on the part of

ADRENAL INHIBITION WITH CORTISONE

TUMOR VS. HYPERPLASIA

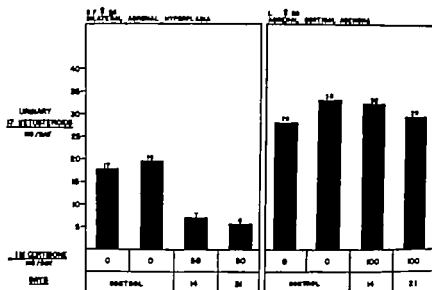


Figure 8 Adrenal cortical inhibition with cortisone acetate: tumor vs. hyperplasia (Thorn, G. W. et al. New England J. Med. vol. 248. Reprinted with permission of the New England Journal of Medicine)

an adenoma has been observed as has a definite response to stimulation and inhibition in other cases. Such findings would most likely depend on the degree of biologic autonomy of the individual tumor and perhaps to some extent on its degree of differentiation, both physiologic and histologic. When physiologic evidence of an adrenal cortical tumor is found by these tests increased efforts should be made in order to locate accurately its position. Adequate preparation of the patient and an excellent plain film are of great value. In addition, serial tomography has been helpful without, and in conjunction with, carbon dioxide instillation via the presacral route. The final certain method of localization is bilateral adrenal exploration. However before this is carried out, in all women showing a lack of hyperactive response to physiologic stimulation of the adrenals and showing signs of virilism, there should be the relatively simple procedure of laparotomy and pelvic exploration in order absolutely to rule out a possible responsible ovarian lesion. Adrenal exploration is carried out subsequently when a tumor is not found in the ovary. At the time of pelvic laparotomy palpatory exploration of the adrenals may reveal the presence of a tumor.

PATHOLOGY OF ADRENOCORTICAL TUMORS

Continued efforts are being made to correlate the histologic findings and the patho-

logic physiology produced by lesions of the adrenal cortex. Hyperplasia of the adrenal cortex may involve predominantly any one of the three zones. In Cushing's syndrome, hyperplasia of the zona fasciculata is usually predominant. Hyperplasia of the zona glomerulosa is sometimes seen in individuals with hypertensive vascular disease, especially in those who have been on a salt restricted diet. Hyperplasia of the zona reticularis may be found in individuals having extreme degrees of virilism. These functional aspects of the different zones of the adrenal cortex must be borne in mind when tumors of the adrenal cortex are being considered. The following simple classification of tumors of the adrenal cortex is suggested:

- 1 Tumors without apparent function
 - a. Cysts of the adrenal cortex
 - b. Adenoma of the adrenal cortex
 - c. Carcinoma of the adrenal cortex
- 2 Tumors with hormonal function
 - a. Adenoma
 - b. Carcinoma

Benign asymptomatic adenoma of the adrenal cortex occurs quite frequently and may be considered to be not pathologic. However unknown factors may stimulate growth of these tumors to sufficient size to cause symptoms and, in almost every patient in whom they do enlarge, abnormal function is manifested and the neoplasms tend to become malignant. Goldzeiter reported that 35 per cent of persons ex post-mortem

have small adrenal cortical adenomas. It is to be remembered also that adrenal cortical adenomas may occur in ectopic adrenal tissue and also in heterotopic adrenal tissue beneath the capsule of the kidney. Cysts of the adrenal cortex are considered as congenital lesions and may attain considerable size which may result in pressure atrophy of that adrenal gland. Only a few of these have been reported which had attained sufficient size to cause symptoms. Displacement of the kidney may be noted on palpation and on roentgenographic investigation as an incidental finding. Clinically they cannot be differentiated from a solid tumor except by surgical exploration or needle aspiration.

The nonendocrine carcinoma of the adrenal cortex may be confused clinically with renal cell carcinoma (Fig. 9) and may invade the kidney (Fig. 10) causing hematuria as the initial symptom. The tumor may be discovered as a result of its mass, and displacement of the kidney may be seen in the roentgenograms, either plain or pyelographic. The cell may have a clear to granular cytoplasm and, in contrast to the adenoma, it grows through its capsule and exhibits many mitotic figures with invasion of veins. Occasionally such neoplasms may develop bilaterally and they may also be found in aberrant adrenal cortical tissue. Metastases occur by hematogenous routes to the lungs and liver. Retroperitoneal nodes may be involved by lymphatic extension and direct growth. These tumors are usually found in adults from the third to the seventh decade of life.

The functional tumors of the adrenal cortex, whether adenomas or carcinomas, may produce the adrenogenital syndrome, Cushing's syndrome, the mixed syndrome, feminin-



Figure 9 A right retrograde pyelogram showing deformity of the upper portion of the renal pelvis and absence of filling of the upper calices in a twenty-eight year-old physician brought to the hospital in shock as a result of a severe renal hemorrhage. The hemorrhage and pyelographic findings were due to adrenal cortical carcinoma invading the kidney

ization syndrome, primary aldosteronism and miscellaneous changes such as gynecomastia, urinary excretion of chorionic gonadotropin or the recurrent hypoglycemic episodes described by Thannhauser. Histologic characteristics do not differentiate the functional from the nonfunctional tumors of the adrenal cortex. It is possible that histochemical studies ultimately may discern detectable differences. The age distribution is quite different from that in nonfunctioning tumors



Figure 10. Photomicrograph of the kidney and invading adrenal cortical carcinoma from the patient whose right pyelogram is shown in Figure 9. The adrenal cortex lies beneath the intrinsic capsule of the kidney and its cells are invading the renal tubules.

insofar as more than half of them occur in individuals younger than fifteen years of age. There is often disagreement among pathologists as to whether a tumor of the adrenal cortex is an adenoma or a carcinoma. In view through the capsule and the presence of many mitotic figures are indubitable evidence of the fact that malignant disease is present. Abnormal physiologic changes may be present for years before the tumor is discovered, which suggests slow growth initially. Rappaport found that one-fourth of these patients showed some degree of cortical atrophy on the opposite side. All patients having an adrenal cortical tumor should be protected by cortisone or hydrocortisone supportive therapy during and following the removal of the tumor. Tumors producing virilism are less likely to cause atrophy of the opposite adrenal gland.

Both adenoma and carcinoma of the adrenal cortex may produce Cushing's syndrome. The incidence of malignancy has been reported as being higher when the tumor causes the mixed syndrome with elements of both virilism and Cushing's syndrome. The highest incidence seems to be in persons in the third and fourth decades of life. The majority of these tumors are found in females and it is to be remembered that Cushing's syndrome due to hyperplasia of the adrenal cortex, is also much more common in women than in men. The physiologic autonomy of the tumor may be studied by efforts at inhibition with cortisone therapy or by efforts at stimulation with ACTH. These tests may be helpful with reference to prognosis.

Feminization in males is extremely uncommon, especially in adult males. It is characterized by gynecomastia, testicular atrophy and impotence. Wilkins reported a series of six instances all of which resulted from adrenal cortical tumors. In such cases, clinically it would be important to study if possible the urinary estrogen, 17 ketosteroid and androgen excretion.

MANAGEMENT OF HYPERADRENOCORTICISM

Medical therapy of Cushing's syndrome has been directed at adrenocortical inhibition. This has been most successfully carried out with cortisone acetate, but the improvements are incomplete and temporary. The administration of testosterone propionate has been of some help in improving protein metabolism. Pituitary irradiation in the treatment of Cushing's syndrome has been re-

viewed by Sosman. About 50 per cent of the patients treated underwent at least a partial remission, but in only one-sixth of those treated did the remission persist. Pituitary irradiation should not be used unless all physiologic tests indicate that the disease is due to adrenocortical hyperplasia, for in one-fourth of the patients having Cushing's syndrome malignant tumors are present. The therapeutic procedure of choice in Cushing's syndrome regardless of the adrenocortical lesion present, is the surgical removal of hyperfunctioning adrenal cortical tissue, which consists of resection of unilateral, rarely bilateral, tumors and total or subtotal adrenalectomy in patients with bilateral adrenal cortical hyperplasia. The following points in the surgical management of hyperadrenocorticism deserve emphasis.

Recently adrenocortical surgery has been made possible by the availability of adrenocortical hormones. It is necessary that all patients subjected to the removal of adrenocortical tissue be adequately prepared preoperatively and supported postoperatively by careful observation and adequate hormonal replacement therapy.

Cortisone acetate 100 mg. given intramuscularly should be given twelve hours and two hours before surgery. Hydrocortisone should be administered intravenously continuously at a rate of about 100 mg. per four hours during the operation and immediately thereafter. Hydrocortisone may be continued postoperatively by constant intravenous drip at a rate of 100 mg. per eight hours. An alternative schedule which may be used would consist of cortisone acetate, 100 mg., given intramuscularly every eight hours, the day of operation and the first postoperative day.

The dosage of cortisone acetate is gradually diminished 25 mg. per dose after the third postoperative day. This reduction must be carried out slowly and carefully according to the state of the patient who is recovering from the hyperadrenocortical state. Careful study of electrolyte balance, weight, carbohydrate metabolism and kidney function must be pursued throughout the immediate postoperative period.

The most expert medical opinion must be obtained with reference to the use of the salt retaining hormones such as fluorohydrocortisone and desoxycorticosterone acetate. A profound increase in excretion of sodium chloride may occur within the first ten days after bilateral total adrenalectomy and must be combated with adequate amounts of salt.

as well as the salt retaining hormones. Adrenocortical insufficiency is manifested by the onset of headache, weakness, malaise lassitude, nausea, vomiting and hypotension.

When an adrenal cortical tumor has been found and located preoperatively unilateral adrenalectomy is performed. When a tumor has not been located by preoperative study the patient is prepared for bilateral exploration. Since the surgical hazards are less on the left than on the right, exploration of the left adrenal gland is usually undertaken first. Simultaneous exploration of both sides can be carried out on patients who are not tremendously obese. Exploration of the opposite side is always desirable irrespective of the gross characteristics of the visualized gland. If obvious adrenal cortical atrophy is present, a hyperfunctioning tumor on the opposite side is very likely.

If the adrenal cortices appear hyperplastic or physiologically hyperactive, bilateral adrenalectomy is indicated. Poutasse and Higgins reported the finding of bilateral adrenal cortical atrophy and the presence of an ectopic hyperfunctioning adrenal cortical lesion.

Bilateral adrenalectomy is the procedure of choice for adrenal cortical hyperplasia. If a tumor is found, the tumor and the entire adrenal gland on that side are removed. In the majority of patients, a single-stage operation with exploration of both sides can be carried out. However if at the time of exploration it is apparent that the patient is not able to undergo a bilateral exploration, the performance of a unilateral adrenalectomy is the procedure of choice if the gland is abnormal.

Every effort is directed preoperatively toward the gaining of maximal renal and cardiorespiratory function. A plastic cannula is placed in the antecubital or internal malleolar vein preoperatively for the continuous administration of fluids, blood, hormones and pressor substances as necessary. The intravenous catheter should be removed within forty-eight hours because of its tendency to cause thrombophlebitis. Pentothal sodium induction followed by intratracheal nitrous oxide-ether anesthesia has been quite satisfactory. However in very poor-risk patients, a continuous spinal anesthesia has been very efficacious. These patients must be watched continuously during the first twenty-four hours after operation. Every measure is instituted to forestall pulmonary and vascular complications turning every two hours, deep-breathing exercises dorsiflexion exercises of the feet, elevation of the foot

of the bed, use of elastic stockings prior to and after operation and utilization of ambulation, avoiding the sitting position, as soon as reasonable.

Priestley and Cahill have advocated subtotal adrenalectomy. In our experience, it seems that bilateral total adrenalectomy is a preferable procedure for bilateral adrenal cortical hyperplasia. Subtotal adrenalectomy carries certain possible disadvantages. Relapse may occur in approximately 10 to 20 per cent of patients because of recurrent hyperplasia when an adequate remnant has been left. The state of the adrenal cortical remnant is unknown and adrenal insufficiency has occurred in approximately 50 per cent of patients so treated when reliance has been placed on this to maintain survival. The possibility that stress may precipitate an adrenal crisis, because there is not sufficient adrenal tissue to meet the hormonal demands remains a real possibility. All of these patients have to be instructed with regard to meeting stress by an increased ingestion of hormones such as cortisone acetate.

The results of adrenalectomy for hyperplasia or tumor of the adrenal cortex are very dramatic, but each patient has to go through a period of many months of adjustment to the new hormonal status. Careful observation during this period is very necessary. Susceptibility to infection must be guarded against and the patient's activities carefully regulated. In patients having virilism, hirsutism is very slow to change. Hypertension usually subsides but may persist, according to whether the vascular changes are irreversible. Osteoporosis is characteristically slow to heal. A dramatic improvement in diabetes may occur but this also may be gradual in improving. Menses may be resumed within one month after removal of an adrenocortical tumor. Improvement in the skin, striae and hemorrhagic tendencies occurs within a few months. The mental status of the patient is usually markedly improved along with appetite and strength. The prognosis of patients having these disorders of the adrenal cortex has been tremendously changed as a result of the availability of adequate hormonal substitution therapy which has made possible surgical removal of the adrenal glands and cure of the disorder.

ADRENOCORTICAL RELATIONS TO HYPERTENSIVE VASCULAR DISEASE

It is well known that adrenal cortical insufficiency is associated with hypotension and that the development of Addison's disease in patients with pre-existing hyperten-

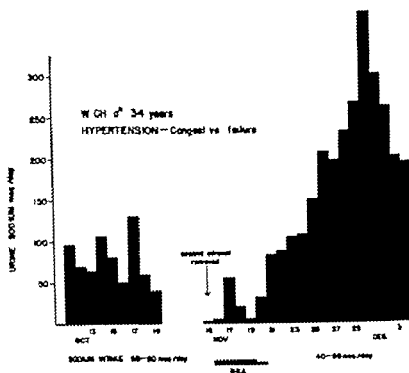


Figure 11 The urinary sodium excretion in a man, aged thirty four years, with malignant hypertension and intractable heart failure before and after bilateral total adrenalectomy. This sodium excretion has been a striking measurable physiologic benefit derived in certain patients having malignant hypertension pursuant to total adrenalectomy.

sion is usually accompanied by a fall in blood pressure to normal or subnormal levels. Hyperadrenocorticism and pheochromocytoma are each associated with elevation of the blood pressure. It is also known that ACTH is tolerated poorly by the hypertensive patient. Goldblatt has shown that experimental renal hypertension is not maintained in the absence of the adrenal glands. The administration of excessive amounts of desoxycorticosterone acetate to the Addisonian patient who has had pre-existing hypertension will result in the return of hypertension. The above evidence that the adrenal cortex is involved in the maintenance of hypertension has led, since the advent of cortisone to an investigation of the effects of adrenalectomy in patients with hypertensive vascular disease. The description of primary aldosteronism by Conn has further enhanced the interest in adrenocortical relations to hypertension.

Green found an improvement in patients with hypertension and those with diabetes after subtotal adrenalectomy. It was later shown by Thorn that the hypertensive patient not only could survive, but could lead an active existence, after total adrenalectomy. On the basis of experience gained in the last five years, the following conclusions

concerning the selection and maintenance of patients with severe hypertensive vascular disease may be made.

Complete and subtotal bilateral adrenalectomy are relatively safe procedures in properly selected patients. After total adrenalectomy patients can be safely maintained on adequate substitution therapy which includes cortisone and appropriate doses of desoxycorticosterone acetate, or its equivalent, with additional supplementary sodium chloride depending upon the individual demands.

Though the chronologic age of the patient is not a critical factor the physiologic age of the patient's vascular disease and the degree of reversibility of it are definite critical factors. Advanced coronary and cerebral arterial disease greatly enhances the hazard of adrenalectomy. Advanced renal vascular disease and renal insufficiency without exception are contraindications to adrenalectomy.

Adrenalectomy is ineffective in reducing arterial blood pressure unless some degree of adrenal cortical insufficiency is produced. However regression of advanced coronary renal or cerebrovascular arterial disease has not occurred, even though lowering of blood pressure has resulted.

The outstanding measurable physiologic benefit derived from total adrenalectomy in patients with hypertension has been the increased urinary excretion of sodium (Fig. 11) and water which occurs and significantly diminishes the total body sodium. This result has been of particular benefit in patients with intractable congestive heart failure (Fig. 12). Such a reduction in total body sodium is not a guarantee that blood pressure will fall and, in fact, a marked reduction in blood pressure in the presence of advanced vascular disease is not desirable. Patients having advanced irreversible vascular disease may suffer with cerebral anemia and diminution in renal function as a result of such reduction in blood pressure.

It seems that the simultaneous performance of sympathectomy may improve the effects of total or subtotal adrenalectomy in selected patients with hypertensive vascular disease.

On the basis of a series of patients with severe hypertensive vascular disease subjected to total adrenalectomy severe angina pectoris, electrocardiographic evidence of advanced coronary artery disease and severe renal insufficiency are contraindications to the operation. In fact, all patients having a blood urea nitrogen of more than 40 mg. per 100 cc. have been made worse by adrenalectomy.

Continuous spinal anesthesia has been the anesthesia of choice for this procedure in these patients. The prone position allowing

a simultaneous bilateral exposure of both adrenals is desirable and is utilized in all patients who are not too heavy or obese. Exposure of the left adrenal is obtained by resection of the twelfth rib and it is desirable on the right side to get a wider field of exposure by resecting the eleventh rib. The prone position has the advantage of not changing the patient's position during the operation. If the posterolateral approach is used, a gradual change in position is carried out as severe hypotension may be precipitated if this is performed suddenly. At the termination of the operation the patient is carefully turned on the back and remains on the operating table in the recovery room for at least two hours in order to insure circulatory equilibrium. Disregarding these precautions may precipitate a profound cardiovascular collapse and even cardiac arrest.

The best results have been obtained in those patients suffering from congestive failure which has not responded to all forms of medical therapy. At the same time these patients must have fairly good renal function. In a series of twenty patients with varying degrees of hypertensive vascular disease, more than 50 per cent obtained some improvement pursuant to total adrenalectomy. None of these patients had hypokalemic alkalosis or histologic evidence of adenoma causing Conn's syndrome of primary aldosteronism. This improvement was maintained for more than six months in ten patients and in six of these patients the im-

CHANGES IN HEART SIZE FOLLOWING BILATERAL ADRENALECTOMY IN HYPERTENSION

W.C. § 30

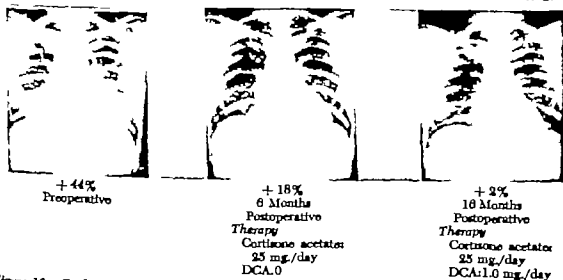


Figure 12. Cardiac roentgenograms of the patient described in Figure 11 showing reduction in size of cardiac silhouette following total adrenalectomy. Over a period of four and a half years clinical improvement has been commensurate with the marked changes herein illustrated.

provement was maintained for more than four years. The improvement for the most part seemed to be based on the increased urinary excretion of sodium and water. Mental equanimity and better adjustment to environment were manifestations of psychologic improvement. The causes of death in the fifteen patients who died in this group were myocardial insufficiency in two, uremia in four, congestive failure in one, cardiac arrest in one, adrenal cortical insufficiency in two, carcinoma of the prostate in one, rupture of an aneurysm in one and cerebrovascular accident in three.

THE ADRENAL CORTEX IN THE TREATMENT OF CANCER OF THE PROSTATE AND BREAST

The endocrine control of the growth of the prostate and breast has been known for many years. In 1896, Sir George Beatson appreciated that carcinoma of the breast could be influenced by the secretion of the ovaries and he obtained a limited remission of the disease by performing oophorectomy. This operation was replaced by irradiation sterilization and it was not until recent years that it was appreciated that cessation of the menses did not necessarily mean that the secretion of estrogens had stopped. Surgical sterilization is the only certain method of stopping ovarian estrogen production. In 1893 White suggested removal of the testes for prostatic enlargement and, in 1896, Cabot reported on this method of treatment. It remained for Huggins in 1941 to report his epoch making discovery of response of cancer of the prostate to castration and the administration of estrogens. In 1945 Huggins and Scott reported bilateral adrenalectomy for cancer of the prostate which had reactivated after previous response to castration and estrogens. Some improvement was noted in these patients, but they all succumbed to adrenal cortical insufficiency. When cortisone acetate later became available it was demonstrated that not only did the patient survive, but also he could lead an active existence after bilateral total adrenalectomy. An intensive study of the efficacy of this new attack on reactivated cancer of the prostate and breast has been carried out by Huggins and other investigators. At the same time the value of adrenocortical inhibition by means of orally administered cortisone acetate prior to adrenalectomy was utilized after it had been demonstrated that diminished function of the adrenal cortex could be accomplished in this manner. Improvement of

patients having carcinoma of the prostate and of patients having carcinoma of the breast has been observed following cortisone inhibition of the adrenal cortex by a number of observers. Further investigation of the effects of adrenocortical inhibition with cortisone acetate and with fluorohydrocortisone is being continued.

Hormonal studies before and after castration have shown a marked reduction as a result of this procedure in the excretion of androgens. This was measured biologically. However a significant excretion of androgen can be measured biologically after castration and this androgen excretion is further greatly diminished by total adrenalectomy. The administration of ACTH to patients after castration has shown a marked rise to occur in androgen excretion to much higher levels than were demonstrated in the resting state before orchiectomy. This concretely supports the assumption and studies of Huggins and Scott that the adrenal cortex is the source of extragonadal androgens which may play a role in the reactivation of cancer of the prostate.

At the Peter Bent Brigham Hospital a study of adrenalectomy for reactivated cancer of the prostate has been conducted during the past five years. The patients have all been in a terminal state from incurable cancer which has either failed to respond to other methods of therapy or has responded previously only to become reactivated at a later date. The patients chosen for treatment by adrenalectomy are now selected on the following bases: a previous response to castration and estrogen therapy has occurred, the local lesion or its metastases have subsequently reactivated, the patient having the neoplasm in its reactivated state should show a response to the administration of cortisone acetate over a period of weeks or months, and for the relief of unremitting pain.

If the response to castration and estrogen therapy lasted a year or longer the prognosis following adrenalectomy is better because a more susceptible tumor is probably present.

Some patients have shown improvement for one to two years with cortisone therapy after a relapse following orchiectomy and estrogen therapy.

The principles of management previously outlined before and after operation are followed. The patients being treated by adrenocortical inhibition with cortisone acetate prior to operation receive 100 mg. of this hormone daily for seven to ten days and the dose is gradually diminished to 50 mg. per

total adrenalectomy. With relief of pain, there is improvement of appetite, improved nutrition and a corresponding improvement in anemia. The patient showing a response to adrenalectomy is expected to have a decrease in calcium excretion and an early increase of alkaline phosphatase of the blood serum. Healing of pathologic fractures has been observed in two patients after total adrenalectomy. This form of therapy must be regarded as palliative at best and the results in the treatment of cancer of the prostate have not been as impressive as those obtained in the treatment of cancer of the breast. It must be emphasized at this time, that, on the basis of the histologic picture alone, the subsequent biologic behavior of the tumor either of breast or prostate, can not be accurately predicted. The highly undifferentiated anaplastic and especially sarcomatous types of carcinoma do not seem to respond as well to hormonal therapy as does adenocarcinoma.

RESULTS OF ADRENALECTOMY FOR CANCER OF THE BREAST

In adrenalectomy for cancer of the breast, the objective and subjective improvement may not necessarily be commensurate. As in cancer of the prostate subjective improvement may be very dramatic with relief of pain within twenty-four to forty-eight hours after the operation. The objective improvement includes the decrease or even disappearance of pulmonary metastases, the decrease of pleural effusion or the diminution in size of the primary lesion. Evaluation of the objective results shows that 25 per cent are excellent, 50 per cent of the subjects are improved and 25 per cent are unaffected. The subjective results are quite similar. It is not possible to select accurately those patients who will show an excellent response. However it seems that those patients who show a good response to adrenal cortical inhibition are more likely to get a good result pursuant to bilateral adrenalectomy than those who do not show such a response. The results have been better and more prolonged in the use of this therapy for cancer of the breast than for cancer of the prostate. The really significant fact is that both types of tumor have been affected by this method of hormonal therapy and it is indicated that further efforts be made to select more accurately those who will be benefited by such treatment. The striking diminution of calcium excretion following adrenalectomy has been emphasized by Pearson and serves

as a reliable index of improvement in the treatment of cancers of breast and prostate.

Prednisone (Meticorten) has been used effectively for adrenocortical suppression and has been accompanied by fewer side effects than cortisone acetate. Such medical therapy may give good results for periods up to two years in reactivated cancer of prostate and breast.

TUMORS OF THE ADRENAL MEDULLA

The adrenal medulla, though not essential for life, plays an important part in stress, owing to the liberation of epinephrine. It arises from primitive cells (sympathogonia) which migrate to and penetrate the adrenal cortex in about the seventh week of fetal life. Because of their common origin, both the sympathetic ganglia and the adrenal medulla are the seat of similar tumors. The most common of these is the neuroblastoma or sympathicoblastoma, less common are the pheochromocytoma, or chromaffinoma, and the benign ganglioneuroma.

Since the work of Cannon, an additional medullary hormone, norepinephrine, has been identified, existing with epinephrine in the normal gland in a ratio of 1:4. The presence of norepinephrine has been demonstrated in the adrenergic nerves of cattle as the probable chemical mediator of the sympathetic nerve impulse. The effects of medullary stimulation in the normal subject are due mainly to epinephrine; that is, increase in cardiac output with resulting hypertension, diminished peripheral resistance, increase in glycogenolysis, increase in oxygen consumption, increase in muscle and splanchnic blood flow and decrease in skin blood flow with resulting pallor. Norepinephrine, on the other hand, in equivalent doses produces hypertension due to increase in peripheral resistance without the other metabolic effects seen with epinephrine. With large doses the effects of the two drugs tend to overlap.

Pheochromocytoma. This tumor arises from chromaffin cells of the adrenal medulla, or other parts of the sympathetic system, and is the only secreting tumor of the medulla. About 10 per cent of these neoplasms are malignant and show a tendency toward early widespread metastases. About 10 per cent are bilateral and more than 15 per cent are extra adrenal in origin.

The syndrome produced by these tumors is characterized by sustained or paroxysmal hypertension. In Smithwick's series of 1000 cases of lumbar symp-

tomy for

hypertension there was a 0.5 per cent incidence of pheochromocytoma. Elevation of the basal metabolic rate and hyperglycemia may occur. About 25 per cent of the patients show acute paroxysms of hypertension with sudden, severe rises in blood pressure, sometimes to levels in the systolic pressure of 300 mm. of mercury. Headache, palpitation, anxiety, tachycardia, pallor and hyperhidrosis are of frequent occurrence and visual blurring, epigastric and precordial pain, nausea and vomiting may also occur. The attacks may last minutes or days and tend to become more frequent as time elapses. Paroxysms may be induced by pressure over the tumor or by emotional upsets. All of the complications of severe hypertensive vascular disease may occur. An attack may terminate in a fatal shocklike state of hyperpyrexia and hypotension or death may occur from pulmonary edema, ventricular fibrillation or cerebral hemorrhage. The persistent form of hypertension may be indistinguishable from essential hypertension on clinical grounds. In a few instances, hypermetabolism and hyperglycemia have been present without hypertension.

Since both epinephrine and norepinephrine are present in varying amounts, the quantitative chemical composition of the tumor determines the clinical picture, as a general rule. Though they differ markedly when used in small doses, the two hormones tend to produce similar effects when present in large amounts. Tumors which elaborate large amounts of norepinephrine or have a predominant amount of epinephrine, tend to produce hypertension, tachycardia, hyperhidrosis, hypermetabolism and frequently hyperglycemia. Tumors which are small and contain, for practical purposes, only norepinephrine closely mimic, in their effects, essential hypertensive vascular disease. In some patients, sustained hypertension has been observed in the absence of active secretion of medullary products in intervals between paroxysmal discharges of the medulla. Elevated blood pressure has also been noted to persist after removal of all hyperactive chromaffin tissues, apparently because of a process initiated by long-continued hypersecretion.

The plain film of the abdomen together with pyelography has been helpful in demonstrating the presence of a suprarenal mass in a number of instances. Presacral injection of carbon dioxide into the retroperitoneal spaces has been helpful in outlining tumors in the suprarenal area, but false positive results may be obtained and small tumors can

not be seen. In some patients a mass is palpable within the abdomen. Pharmacologic testing agents have been of great help in the diagnosis of these lesions. These are of two groups: the adrenergic blocking agents—benzodioxane and Regitine—and the provocative agents—histamine, Mecholyl and tetraethylammonium chloride. Of these, benzodioxane, Regitine and histamine have proved to be of the greatest value. Determination of urinary catecholamines, as a measure of adrenal medullary activity, appears to hold promise for a more accurate diagnosis of these tumors.

Surgical removal by the most direct route is the desired treatment. Because of the increased risk of cardiac arrhythmias in the presence of high circulating levels of epinephrine, cyclopropane and ethyl chloride should not be used as anesthetizing agents. Likewise, anoxia should be avoided because of its stimulating effect on the adrenal medulla. A general exploration of the abdominal cavity is of great value in view of the variability in location and the multiplicity of these tumors. A transperitoneal approach gives the opportunity for a general exploration and is especially useful when the location of the tumor is in doubt. Tumors arising outside of the abdomen are dealt with according to their particular location. When a well localized neoplasm is clinically diagnosed in the adrenal area, the transcostal approach provides for a direct attack on the tumor and its blood supply.

Paroxysmal rises in blood pressure during operation and profound hypotension postoperatively constitute the most serious complications. The former is combated by the use of adrenergic blocking agents and the latter by the employment of vasoconstrictive agents such as epinephrine, Neo-Synephrine and norepinephrine. The possible occurrence of adrenal insufficiency following surgery should be anticipated and the patient treated promptly.

Neuroblastoma Sympatheticum. This is the most common malignant tumor occurring in infancy and early childhood and, in spite of its highly malignant character and tendency toward early widespread metastases, much has been done to improve prognosis in these patients. About 57 per cent of the neoplasms are intra-abdominal in location.

In a few instances a spontaneous cure has been noted due either to hemorrhage and necrosis of the tumor or to conversion of the neoplasm into a well-differentiated, benign ganglioneuroma.

A large abdominal mass is often palpable.

Anemia, weakness, weight loss, evidence of metastases by x ray and physical examination, pain due to involvement of nerves or adjacent structures, and displacement of the kidney downward and laterally without invasion of the renal substance are all frequently seen. Metastases spread by way of lymphatics to regional lymph nodes and by way of the blood stream to bones, liver and other organs. Plain roentgenograms of the abdomen and either intravenous or retrograde pyelography along with survey films of the bones and chest, are of help in establishing the diagnosis.

A short, well-organized period of study and supportive therapy are the appropriate measures. A transabdominal or combined flank and abdominal approach with resection of the eleventh and twelfth ribs is used. If the tumor is not completely resectable as much as possible is removed and postoperative x ray therapy given. These tumors are generally very radiosensitive and the combination of surgery and x ray treatment started immediately following operation yields a cure rate which is much better than that previously obtained by surgery alone. In a few patients, roentgen ray therapy alone has been responsible for cure.

READING REFERENCES

- Albright, F. Cushing's Syndrome: Its Pathological Physiology, Its Relationship to the Adrenogenital Syndrome, and Its Connection with the Problem of the Reaction of the Body to Injurious Agents ("Alarm Reaction of Selye") Harvey Lect. 38:122, 1942-1943.
- Bosban, C. T. On Treatment of Inoperable Cases of Carcinoma of the Mamma. *Lancet* 2:104-107, 162-165, 1896.
- Cabot, A. T. The Question of Castration for Enlarged Prostate. *Ann. Surg.* 24:265, 1898.
- Cade, S. Adrenalectomy for Breast Cancer. *Brit. M. J.* 1:1, 1935.
- Cahill, C. F. Hormonal Tumors of the Adrenal. *Surgery* 16:233, 1944.
- Cahill, C. F., and Aronow H. Jr. Pheochromocytoma: Diagnosis and Treatment. *Ann. Int. Med.* 31:389, 1949.
- Cahill, C. F., Melicow M. M., and Darby H. H. Adrenal Cortical Tumors. *Surg. Gynec. & Obst.* 74:281, 1942.
- Cannon, W. B. The Emergency Function of the Adrenal Medulla in Pain and the Major Emotions. *Am. J. Physiol.* 32:356, 1914.
- Chambers, W. L. Adrenal Cortical Carcinoma in a Male with Excess Gonadotropin in Urine. *J. Clin. Endocrinol.* 9:451, 1949.
- Ciba Foundation Colloquia on Endocrinology, Vol. 8, The Human Adrenal Cortex. Boston, Little, Brown & Co., 1953.
- Conn, J. W. Primary Aldosteronism, a New Clinical Syndrome. *J. Lab. & Clin. Med.* 43:5, 1953.
- Conn, J. W. Primary Aldosteronism. *J. Lab. & Clin. Med.* 45:661, 1955.
- Crooke, A. C. Change in Basophil Cells of Pituitary Gland Common to Conditions Which Exhibit Syndrome Attributed to Basophil Adenoma. *J. Path. & Bact.* 41:339, 1935.
- Cushing, H. Pituitary Body Hypothalamus, and Parasympathetic Nervous System. Springfield, Ill., Charles C. Thomas, 1932.
- Emlet, J. R., Grimson, K. S. Bell, D. M. and Orgas, E. S. The Use of Pteripran and Regitine as Routine Tests in Patients with Hypertension. *J.A.M.A.* 146:1383, 1951.
- Gifford, R. W. Roth, G. M. and Jvale, W. F. Evaluation of New Adrenolytic Drug (Regitine) as Test for Pheochromocytoma. *J.A.M.A.* 149:1623, 1952.
- Goldblatt, H. Studies in Experimental Hypertension: Pathogenesis of Experimental Hypertension Due to Renal Ischemia. *Ann. Int. Med.* 11:69, 1937.
- Goldenberg, M. Adrenal Medullary Function. *Am. J. Med.* 10:627, 1951.
- Goldenberg, M., Aronow H. Jr., Smith, A. A., and Faber M. Pheochromocytoma and Essential Hypertensive Disease. *Arch. Int. Med.* 86:823, 1950.
- Goldenberg, M., Serlin, L., Edward, T., and Rappaport, M. M. Chemical Screening Methods for the Diagnosis of Pheochromocytoma. *Am. J. Med.* 16:310, 1954.
- Goldenberg, M., Snyder C. H., and Aronow M. Jr. A New Test for Hypertension Due to Circulating Epinephrine. *J.A.M.A.* 135:971, 1947.
- Goldenberg, M. and others. Hemodynamic Response of Man to Nor-epinephrine and Epinephrine and Its Relation to Problem of Hypertension. *Am. J. Med.* 5:792, 1948.
- Goldzieher M. A. The Adrenals. New York, The Macmillan Company, 1929.
- Green, D. M., Nelson, J. N. Dodds, G. A., and Smalley R. E. Bilateral Adrenalectomy in Malignant Hypertension and Diabetes. *J.A.M.A.* 114:436, 1950.
- Grimson, K. S. Emlet, J. R., and Hamblen, E. C. Diagnosis and Management of Tumors of Adrenal Gland. *Ann. Surg.* 134:451, 1951.
- Gross, R. E. The Surgery of Infancy and Childhood. Philadelphia, W. B. Saunders Company, 1953, pp. 606-625.
- Hall, P. F. The Functions of the Endocrine Glands. Philadelphia, W. B. Saunders Company, 1950.
- Harrison, J. H. and Jenkins, D. The Adrenals. In Urology edited by M. Campbell. Philadelphia, W. B. Saunders Company, 1954, pp. 2253-2256.
- Harrison, J. H. and Luskilaw J. C. Recent Studies of Hyperadrenocorticism. Its Pathologic Physiology and Management. *S. Forum* 4:559, 1954.
- Huggins, C., and Hodges, C. V. Studies on Prostate Cancer. I. Effect of Castration, of Estrogen, and of Androgen Injection in Serum Phosphatases in Metastatic Carcinoma of Prostate. *Cancer Res.* 1:293, 1941.
- Huggins, C. and Scott, W. W. Bilateral Adrenalectomy in Prostatic Cancer. *Ann. Surg.* 122:1031, 1945.
- Huggins, C., and Scott, W. W. Surgery of the Adrenals. *J.A.M.A.* 147:101, 1951.
- Jeffers, J. A., and others. The Clinical Course Following Adrenal Resection and Sympathectomy of 83 Patients with Severe Hypertension. *Ann. Int. Med.* 39:254, 1953.

- Kendall, E. C.: The Chemistry and Partial Synthesis of Adrenal Steroids. *Ann. New York Acad. Sc.* 59: 540 1949
- O'Neal, L. W., and Heinbecker P. The Adenohypophysis and Hypothalamus in Hyperadrenocorticism. *Ann. Surg.* 141: 1 1955
- Poutasse, E. F., and Higgins, C. C.: Surgery of the Adrenal Glands for Cushing's Syndrome. *J. Urol.* 70:129, 1953.
- Priestley, J. T., Randall, G. S., Walters, W., and Salama, R. M.: Subtotal Adrenalectomy for Cushing's Disease. *Ann. Surg.* 134: 464 1951
- Reichstein, T., and Shoppee, C. W. The Hormones of the Adrenal Cortex. *Vitamins & Hormones* 1 345, 1945.
- Selye, H. General Adaptation Syndrome and the Diseases of Adaptation. *J. Clin. Invest.* 6:117 1948.
- Selye, H.. Textbook of Endocrinology. Acta, Montreal, 1947
- Sosman, M. C. Cushing's Disease—Pituitary Basophilism. Caldwell Lecture, 1947. *Am. J. Roentgenol.* 62:1 1949
- Sprague, R. G., Kvale, W. F., and Priestley, J. T. Management of Certain Hyperfunctioning Lesions of the Adrenal Cortex and Medulla. *J.A.M.A.* 151: 699, 1953.
- Thorn, G. W., and others. Advances in the Diagnosis and Treatment of Adrenal Insufficiency. *Am. J. Med.* 10:595 1951
- Thorn, G. W., and others. Clinical Studies on Bilateral Complete Adrenalectomy in Patients with Severe Hypertensive Vascular Disease. *Ann. Int. Med.* 37:972, 1952.
- Thorn, G. W. and others: Medical Progress. Pharmacologic Aspects of Adrenocortical Steroids and ACTH in Man. *New England J. Med.* 249:232, 284 323, 399 414 588, 632, 1953.
- von Euler, U. S. and Strom, G.: Present Status of Diagnosis and Treatment of Pheochromocytoma. *Circulation* 15:5, 1957
- White, J. W. Present Position of Surgery of the Hypertrophied Prostate. *Ann. Surg.* 18:152, 1893.
- Williams, L.: The Diagnosis and Treatment of Endocrine Diseases in Childhood and Adolescence. Springfield, Ill., Charles C Thomas, 1950
- Wilkins, L., and others. Treatment of Congenital Adrenal Hyperplasia with Cortisone. I. Comparison of Oral and Intramuscular Administration with a Note of Compounds F and B. *J. Clin. Endocrinol.* 12:257 1952.
- Wilkins, L., and others: Treatment of Congenital Adrenal Hyperplasia with Cortisone. II. Effects on Sexual and Somatic Development. Hypothesis Concerning Mechanism of Feminization. *J. Clin. Endocrinol.* 12:277 1952.
- Wyngaarden, J. B., Keitel, H. G., and Iselbacher J. Potassium Depletion and Alkalosis. Their Association with Hypertension and Renal Insufficiency. *New England J. Med.* 250:597 1954.

THE SPLEEN

By ROBERT M. ZOLLINGER M.D.
and ROGER D. WILLIAMS M.D.

ROBERT MILTON ZOLLINGER was born in Ohio and was educated in The Ohio State University and its Medical School. After receiving his training in surgery in Boston and Cleveland, he returned to his own medical school as its Professor of Surgery. The interest of the Department of Surgery at The Ohio State University in diseases of the spleen has been a long-continued one.

ROGER DAVIS WILLIAMS is associated with the Department of Surgery at The Ohio State University as an Assistant Professor and received his education in medicine at Duke University. His surgical training was received at The Ohio State University where he also did graduate work in the Department of Pathology.

The spleen continues to be one of the most interesting yet least understood organs within the abdomen. Since the left upper quadrant of the abdomen is a relatively silent area, most physicians think of the spleen only in terms of splenomegaly or traumatic rupture. The spleen, however, is often involved in many generalized systemic disease processes and primary splenic abnormalities may produce widespread effects. Abnormalities of splenic physiology which have been more clearly delineated in recent years are not necessarily associated with splenomegaly. The unwary physician may be misled unless he is familiar with the many diseases of this organ.

The changing concepts of splenic function make difficult the classification of splenic diseases under categories wherein there may be indication or contraindication for splenectomy. It may be assumed that trauma usually presents the most common indication for splenectomy in the United States. Whereas some consider surgery of no value

in erythroblastic anemia and leukemia, many reports of the favorable results of splenectomy in certain cases tend to refute earlier impressions. Certainly no set rules regarding surgery can substitute for competent hematologic evaluation of patients with splenic disease.

SURGICAL ANATOMY

Lying beneath the left ninth, tenth and eleventh ribs, the spleen is rarely palpable unless enlarged. In the adult it averages approximately 11 by 7 by 4 cm. in size and weighs approximately 200 gm. It is usually shaped like a large coffee bean and often has a notch along its anteromedial border. The medial concave surface is bordered by the stomach to which the upper pole may be closely attached. The lateral convex surface lies against the left diaphragm and kidney.

The peritoneal attachments of the spleen are of great surgical importance. Three of these form ligaments: the

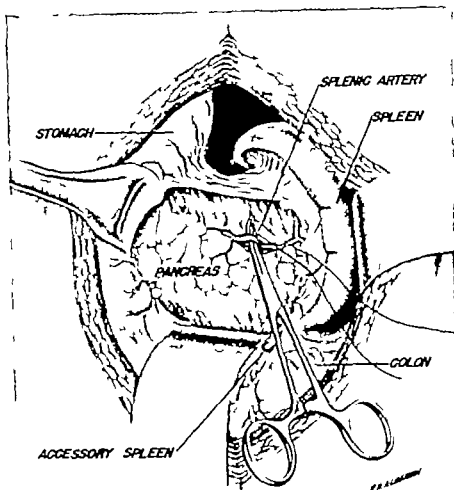


Figure 1 The spleen and its relations to the stomach, colon and pancreas. An opening has been made through the gastrosplenic ligament showing a suture being passed beneath the splenic artery proximal to the splenic pedicle.

which course the arteries and veins (Fig. 1) The *presplenic fold*, coursing from the lower pole and hilum of the spleen to join the gastrocolic ligament, is thin and rarely very vascular. The *gastrosplenic fold* extends as a double peritoneal layer from the splenic hilum to the greater curvature of the stomach. Through these layers course the short gastric arteries and veins which form large collaterals when there is impairment of flow in the splenic artery or vein. This ligament is greatly shortened as the superior hilum lies in close contact with the stomach. The *splenorenal ligament* another double peritoneal fold, stretches from the tail of the pancreas to the splenic hilum and contains the splenic artery and vein. Several smaller peritoneal attachments between the spleen and diaphragm and the spleen and colon may be prominent when the spleen is enlarged or there is portal venous hypertension.

The splenic artery the largest branch of the celiac axis, follows an irregular course over the superior border of the pancreas to

divide into several branches near the hilum. Of the four branches commonly noted, three supply the spleen and two supply the greater curvature of the stomach. The superior and inferior terminal arteries and the superior polar artery all give branches into the spleen and the latter in addition, gives off the short gastric vessels to the fundus of the stomach. The gastroepiploic artery branches off to the greater curvature of the stomach. The splenic vein follows a similar course to the artery except that its course is usually inferior and straighter than that of the artery being frequently partially embedded within the pancreas. The lymphatic drainage of the spleen follows the vessels along which there may be numerous lymph nodes.

INTERNAL ANATOMY

The internal anatomy is less readily defined. The larger arteries which enter at the hilum branch off into the trabeculae, the fibromuscular tissue framework of the spleen. Several trabeculae may form a small,

almost microscopic, lobule. The trabecular arteries branch off into a zone of lymphoid tissue the malpighian body. Continuing from this lymph follicle are the penicillate arteries which communicate either directly through capillaries, or indirectly through the splenic sinuses with the venules. Direct communications through multiple preformed channels have been noted. More recently studies well documented by photographs and movies strongly support a combined open and closed system where blood flows from the arteries into both pulp spaces and venous channels.

The splenic pulp is composed of sinuses and several types of cellular elements. The pulp contains lymphocytes, granulocytes and erythrocytes. There are numerous branching strands of lymphoid tissue known as the cords of Billroth. The sinuses occupy most of the pulp and are lined by a reticular network which becomes continuous with pulp partitions. Within this network are amebic phagocytic cells. In the congested spleen, the line between sinus and parenchyma may be poorly defined and changes which occur with certain splenic dysfunctions are somewhat inconstant.

PHYSIOLOGY

Normal physiologic functions of the spleen are of much less importance than splenic hyperfunction. The spleen is important, primarily as a large part of the reticuloendothelial system and, secondarily because of its position in the portal venous circulation. Since the spleen is not essential to life its functions being readily assumed by the remainder of the reticuloendothelial system, normal function has been difficult to evaluate.

Several functions may be ascribed to the spleen. It may act as a reservoir yielding increased quantities of blood during stress or exercise. Gradual changes in volume take place under normal situations. In the embryo the spleen takes part in blood formation and although this function ceases after birth, it may be taken up again under demand (extramedullary hematopoiesis). Lymphocytes continue to be formed throughout life. Blood destruction also occurs in the spleen, as in all reticuloendothelial tissue. Although phagocytosis is probably insignificant, fragmentation of red cells during periods of relative splenic congestion probably occurs. There is some evidence that the spleen has a regulatory action to increase red cell fragility. The spleen plays an active part in defense

mechanisms which include the formation of antibodies.

Several studies suggest that the spleen may have an endocrinologic function. The spleen has been considered a regulator of bone marrow controlling the emission of its various elements by a humoral mechanism. Some splenic extracts stimulate and others depress the bone marrow. Serum from patients with cytopenias considered due to hypersplenism or dyssplenism have produced similar cytopenias when injected into a normal individual. These functions are probably cytotoxicologic and often can be suppressed by adrenocorticotropin (ACTH) and cortisone. The release of a vasodilator substance, in combination with peripheral cytologic changes resulting from stress, has been considered due to a substance called "splenin" by Ungar. This response to stress is inhibited by splenectomy.

ANOMALIES

Except for accessory spleens anomalies of the spleen are rare. Congenital absence and marked lobulation of the spleen have been noted but are of no clinical importance.

Accessory Spleens. An accessory spleen consists of a separate encapsulated mass of splenic tissue which has probably been pinched off from the main splenic mass during embryologic development. It has been suggested that many occur in the embryo but disappear after birth unless perpetuated by increased demand in such diseases as congenital hemolytic icterus or thrombocytopenic purpura. They occur most commonly in the splenic hilum, the splenic pedicle or tail of the pancreas, the gastrosplenic ligament and the gastrocolic ligament. Rarely they are found over the left kidney, or even in the scrotum. Accessory spleens are reported in 18 to 30 per cent of patients undergoing splenectomy. They number from one to five or more and vary from a few millimeters to several centimeters in size. Although they have the same gross appearance as the spleen, they are readily confused with lymph nodes. Often the accessory spleen not only has its own small artery and vein but also a separate pedicle.

When splenectomy is performed for hypersplenism, it is important to remove all accessory spleens since they may be the cause for continuation or recurrence of the disease. In the patient with a relapse of hypersplenic hematologic changes suspicion of an overlooked accessory spleen may be confirmed by the injection of Thorotrast. diopague

substance which localizes in the spleen liver and other tissue of high reticuloendothelial content. The accessory spleen probably causes relapse of hypersplenism by its marked hypertrophy; however in many cases of thrombocytopenic purpura, accessory spleenectomy has not given great improvement.

ECTOPIC SPLEEN

Abnormal position of the spleen is quite uncommon and is due to either rare congenital elongation of the splenic pedicle or general abdominal wall relaxation with or without visceroptosis. This condition occurs most often in middle-aged women and the spleen is almost invariably enlarged.

The diagnosis of ectopic spleen may be extremely difficult, particularly when the organ becomes fixed in the new position. Symptoms consisting of nausea, vomiting, constipation or a tugging sensation within the abdomen are variable and may not be present at all in the freely movable spleen.

The treatment of ectopic spleen is splenectomy. This may be an emergency procedure if there is torsion of the splenic pedicle.

TRAUMA

Splenic injuries are common and if rupture of the spleen is kept in mind, its diagnosis is not usually difficult. Rupture or contusion of the spleen should be considered in most automobile accidents of severity particularly when the abdomen or left thorax is involved. Fractured ribs may direct attention to the underlying spleen, yet the association of rib fractures with splenic rupture occurs in only about one out of five cases. In children, rib fractures are even less common.

Classification of open or closed wounds serves little purpose. Most open wounds occur with war injuries and are usually obvious. Contusion or laceration of the spleen is more common, yet less readily diagnosed. Either contusion or rupture may follow a violent blow however rupture without obvious trauma may occur.

Two peculiarities of splenic rupture are often observed. The first of these is spontaneous rupture without a history of trauma, occurring usually in a soft, friable, diseased spleen. It has been reported to occur with malaria, typhoid fever, several of the lymphomas, Boeck's sarcoid and infectious mononucleosis. In addition, spontaneous rupture occurs even with a normal spleen although this is rare.

The second peculiarity of the spleen is its

tendency to secondary rupture at varying intervals following the episode of trauma. This phenomenon occurs in approximately one out of six cases, any time from one to thirty or more days after injury. Nearly 75 per cent take place during the first two weeks following trauma. The recurrence of rupture or hemorrhage may be due to continuous increase in a subcapsular hematoma or failure of the omentum or a clot to offer complete tamponade of the spleen.

The symptoms are those of blood loss into the peritoneal cavity. The signs may be minimal or marked. If the blood loss is great, weakness, abdominal pain and muscle spasm, either generalized or localized in the left upper quadrant, and depression of abdominal breathing are noted. Rebound tenderness is common and may be more noticeable than tenderness to palpation. Phrenic reference of pain to the left shoulder occurs in nearly three-fourths of the cases and is diagnostically significant. All symptoms may be overshadowed by shock and other bodily injuries. A palpable tender mass, fixed dullness in the left flank (Ballance's sign) or shifting dullness due to free blood in the peritoneal cavity is a less common finding.

Leukocytosis, with a white count of over 12,000 is of more diagnostic importance than is early anemia. With rapid hemorrhage, hemoconcentration may precede the lowering of hemoglobin or red blood cell count. Abdominal x ray study may be of value, both in suggesting splenic rupture and confirming the presence of other injuries. The findings include elevation of the diaphragm, depression of the colon, medial and anterior displacement of the stomach gas shadow occasionally with serration along its greater curvature, absence of the psoas shadow, absence of the left renal shadow and usually a diffuse haziness in the left upper quadrant without a normal splenic outline. Finally a peritoneal tap is readily performed utilizing a no. 20 or 18 needle and a 10-cc. syringe. If blood is encountered, the diagnosis of internal bleeding is confirmed and the possibility of splenic rupture strongly considered. A negative tap does not eliminate the diagnosis of splenic rupture.

The only safe treatment for splenic rupture is splenectomy which should be performed without hesitation. When the diagnosis is in doubt but there is strong evidence of intra abdominal injury exploratory laparotomy is justified. Suture of tears is not satisfactory. Whole blood should be made

readily available and usually one or more transfusions are required. Following splenectomy complete exploration of the entire abdomen is an absolute necessity in order that the possibility of other intra-abdominal injuries may be eliminated.

The results of splenectomy largely depend upon the extent of other injuries. Nearly half of the patients with ruptured spleen have other injuries to the head, chest or extremities which detract from the diagnosis of splenic injury and increase mortality. Approximately one-third of the patients have other intra-abdominal injuries to the kidney bladder or intestinal tract. Multiple injuries occur since the most common cause of splenic rupture is an automobile accident. If a ruptured spleen is the only significant injury the mortality is low when multiple injuries occur mortality will depend upon the time and adequacy of treatment of all injuries.

HYPERSPLENISM

Several clinical entities in which there is a depression of one or more of the formed elements of the blood are considered to be due to splenic overactivity. Whether the spleen acts directly to produce an increased breakdown of peripheral blood elements elaborates a humoral substance which affects the productivity of the bone marrow or produces antibodies which act on the cellular elements is subject to debate by hematologists. Regardless of the mechanisms of hypersplenism the resulting decrease in red cells, platelets white cells or all three of these elements produces well known clinical diseases. These diseases are classified as primary hypersplenism if congenital hereditary or of unknown cause, and as secondary

hypersplenism if they occur during the course of another chronic disease process.

Four clinical diseases comprise the syndrome of primary hypersplenism (Fig. 2). Depression of circulating blood platelets due to splenic overactivity results in *essential*, or primary thrombocytopenic purpura. The destruction of red blood cells produces congenital hemolytic anemia. Primary splenic neutropenia and panhematopenia are two more recently recognized diseases, in which there is a depression of leukocytes (granulocytes) or all three of the formed elements of the blood. In all four of these disease processes splenectomy effects excellent results.

Secondary hypersplenism produces identical cytopenias, but the splenic dysfunction develops secondary to another chronic disease. The primary chronic diseases include leukemia, Banti's disease, Gaucher's disease, Hodgkin's disease the other lymphomas, tuberculosis, Boeck's sarcoid and other chronic infections. Secondary hemolytic anemia, thrombocytopenia, neutropenia or panhematopenia may occur as a complication during any phase of one of these chronic disease processes. Although splenectomy may produce dramatic remission of the hypersplenism and thereby lengthen life, the primary disease continues its course.

An accurate diagnosis is the prerequisite to good results in the management of hypersplenism. Splenomegaly peripheral blood cytopenia and a hyperplastic bone marrow compose a triad which should cause the diagnosis to be considered. History and physical examination are important, toxic cytopenias are suggested by history. The spleen is usually but not always palpable; the larger spleens occur in patients with

PRIMARY HYPERSPLENISM

1 PLATELETS

2. RED CELLS

3 WHITE CELLS

4 ALL ELEMENTS



1 ESSENTIAL THROMBOCYTOPENIC PURPURA

2 CONGENITAL HEMOLYTIC ANEMIA

3 PRIMARY SPLENIC NEUTROPENIA

4 PRIMARY SPLENIC PANHEMATOPENIA

Figure 2. Several mechanisms may occur whereby a decrease of blood platelets, red cells, white cells or all three elements by a hyperactive spleen produce four clinical disease entities, depending upon which element or elements are decreased.

secondary hypersplenism. Routine blood studies may suggest hypersplenism, but more specific studies are required to confirm the diagnosis in most instances. Bone marrow studies must be made and interpreted by a competent hematologist. A bone marrow hyperplasia of the specific element or elements which are decreased in the peripheral blood should be found.

The epinephrine (Adrenalin) test is of value in the diagnosis of hypersplenism when interpreted in the light of other hematologic findings. After a fifteen to thirty minute base line period, two peripheral blood studies are made following which 0.5 to 1.0 cc. of 1:1000 epinephrine is given subcutaneously. Repeated blood studies at ten-minute intervals are made and correlated with the period of maximum blood pressure rise which coincides with maximum splenic contraction. A marked rise in peripheral blood elements is considered suggestive of hypersplenism, but the test must be interpreted with caution.

Primary (Idiopathic) Thrombocytopenic Purpura. Primary hypersplenism, associated with reduced blood platelets and abnormal bleeding, has been called essential thrombocytopenic purpura, idiopathic thrombocytopenic purpura and Werlhof's disease. Clinical manifestations, due to pathologic bleeding, are the result of a combination of abnormal capillary permeability and a lowered blood platelet level. With an onset early in life the disease may be continuous or intermittent.

One of three mechanisms responsible for the reduction of platelets is thought to be primary hypersequestration and lysis in the spleen. A second mechanism is responsible for abnormal platelet production in the bone marrow from a humoral substance liberated from the spleen. The production of thrombocytopenia in the normal individual by injection of serum from a patient with thrombocytopenic purpura, the occurrence of thrombocytopenic purpura in the newborn and the response of many patients with this disease to adrenocorticotropin (ACTH) and cortisone add weight to the contention of a number of recent investigators that immunologic factors are of great etiologic importance.

Recent reports show that idiopathic thrombocytopenic purpura occurs in two forms. One, an acute, usually self-limiting disease, occurring in infants and children, frequently develops spontaneous remission over a period of several weeks to a year.

Evidence for hereditary factors is inconclusive and there is nearly equal sex distribution. The other is a chronic disease, occasionally with temporary or incomplete remissions in young adults, but usually requiring splenectomy after failure of permanent responses to adrenocorticotrophic hormone (ACTH) or cortisone. Females are more commonly affected with this form.

Easy bruising, an otherwise asymptomatic rash, nosebleeds or menorrhagia may be the first symptoms. The signs and symptoms depend upon the severity of the thrombocytopenia. Although frequent ecchymoses may follow trauma in mild cases spontaneous skin mucous membrane, gastrointestinal or cerebral hemorrhages are usually associated with platelet levels well below 70,000. Menorrhagia is a common first symptom which probably is treated at its onset without diagnosis of thrombopenia since there are frequent associated gynecologic abnormalities. Intracranial hemorrhage presents a serious problem in approximately 10 per cent of cases.

The diagnosis is dependent upon the symptomatology and the hematologic findings. Splenomegaly is uncommon and when found suggests other diagnoses such as secondary hypersplenism due to leukemia, Hodgkin's disease or some chronic systemic infection. Purpura, or spontaneous hemorrhage, is found to be due to markedly lowered blood platelet levels. Bleeding time is prolonged, clotting time is normal and clot retraction delayed or absent. Demonstration of an abnormal coagulation mechanism can be made however by the finding of a defective prothrombin consumption. Reaction to the tourniquet test (Rumpel-Leede phenomenon) in which a tourniquet is applied to the arm for ten minutes at a pressure half way between systolic and diastolic blood pressure is positive, showing numerous petechiae of the skin. Differentiation can be made between purpura hemorrhagica and hemophilia by the history and a prolonged clotting time, normal clot retraction, a negative reaction to the tourniquet test and a normal platelet count in patients with hemophilia. Hyperplasia of the megakaryocytes of the bone marrow must be present. Erythroid hyperplasia is also usually present to a degree consistent with the anemia. This finding of marrow hyperplasia is of utmost value in differentiating essential thrombocytopenic purpura from other purpuras due to the effects of toxins, tumors or infections of the bone marrow.

The dramatic subsidence of bleeding and low mortality have made splenectomy the treatment of choice in most adult patients with this disease. Spontaneous remissions are common in childhood, however, and the results of splenectomy do not indicate an advantage over medical therapy. Adrenocorticotrophic hormone (ACTH) and cortisone decrease bleeding tendency by their effect on capillary permeability plus an elevation of platelet levels. They have been of great value in deferring surgery. In patients failing to respond to these drugs and those with chronic thrombocytopenia which relapses after drug therapy is stopped, splenectomy is indicated.

Surgery should not be delayed in the face of continuously lowered platelet levels because of the danger of intracranial hemorrhage. Preoperative fresh whole blood transfusions have been recommended, not only as a source of platelets but also to raise the lowered blood volume found consistently in these patients. In those patients with inadequate platelet response to splenectomy adrenocorticotropin (ACTH) or cortisone may be of value following removal of the spleen.

Primary (Congenital) Hemolytic Anemia. Of the several forms of hemolytic anemia, the spleen probably plays the most important role in congenital hemolytic anemia. This disease has been described under the terms congenital hemolytic jaundice, acholuric jaundice and spherocytic icterus, all of which point to the clinical features of anemia, jaundice without bile in the urine and the typical spherical characteristic of the red cells. Although spherocytosis is common with other forms of anemia, the familial tendency delineates this entity. Careful history and/or hematologic evaluation of several members of the family may be necessary to establish the diagnosis since mild cases may be entirely asymptomatic. Despite the persistence of spheroidal cells with increased susceptibility to hemolysis, splenectomy is usually curative.

The symptoms vary markedly with the severity of the disease. One member of the family may have severe anemia, splenomegaly and jaundice, while all others have only the characteristic spherocytosis without significant anemia. Latent forms of the disease may become active any time between infancy and the fifth decade of life. Remissions and exacerbations are then common. Mild jaundice, weakness and malaise are the most common symptoms. These patients are not

actually sick, except when hemolytic crisis and acute severe exacerbation of hemolytic activity occur. Dull pain or dragging discomfort in the left upper abdomen is occasionally found and splenic tenderness with splenomegaly increases during exacerbations of hemolysis. Nausea and vomiting, abdominal pain and a rapid increase in jaundice are noted with a hemolytic crisis.

As with all jaundiced patients, the diagnosis depends upon an accurate history plus certain laboratory data. Confirmation of the familial nature of the disease is important. The peripheral blood shows microcytic anemia with 2 to 4 million erythrocytes per cu mm and from 4 to 12 gm. of hemoglobin per 100 cc. Counts below 1 million occur with hemolytic crisis. Spherocytosis and a marked reticulocytosis (5 to 20 per cent) are invariably present. The spheroidal red cells show increased fragility with hemolysis in 0.7 to 0.5 per cent saline, as compared with 0.45 per cent for normal cells. The bone marrow shows an erythroid hyperplasia of a degree dependent upon the degree of anemia. Bone marrow studies are absolutely necessary to rule out other forms of anemia. In the indirect blood van den Bergh test, the value is elevated. There is an increase of urobilin in the urine and stool, but bile is absent in the urine (acholuric jaundice) while urobilin is increased. Splenomegaly is common, the spleen usually weighing 1000 to 1500 gm., but the spleen may not be palpable in patients having mild or asymptomatic anemia.

The only acceptable treatment is splenectomy. If the diagnosis is correctly established, the results of splenectomy are immediate and dramatic. Preoperative transfusions are contraindicated since the danger of producing hemolytic crisis is greater today than splenectomy in a moderately anemic patient. Once the splenic artery is ligated, blood replacement should follow. Although spherocytosis and increased red cell fragility are not changed, hemolysis ceases following splenectomy.

Primary Splenic Neutropenia. Neutropenia and granulocytopenia due to primary hypersplenism were first described in 1939. The decrease in white cells due to overactivity of the spleen and the response to splenectomy have been confirmed by numerous observers.

The symptoms are usually those of a recurrent or chronic infectious process. Oropharyngeal and skin infections may be associated with weakness, malaise and inter-

mittent fever. Infections respond poorly to the usual methods of therapy. In some cases, markedly severe anemia and mild jaundice have been noted. Splenomegaly is usually present and the large spleen may cause abdominal discomfort. Joint pains in some patients have suggested Felty's syndrome from which primary neutropenia must be differentiated.

The diagnosis is dependent upon the findings of neutropenia for which there is compensatory myeloid hyperplasia of the bone marrow. Positive reaction to the epinephrine test-splenic contraction with a significant temporary elevation of circulating neutrophils-is of diagnostic value. Anemia and mild thrombocytopenia may also occur in varying degrees. Care must be taken to rule out toxic neutropenias in which the bone marrow is not hyperplastic and shows myeloid maturation arrest.

Although splenectomy is the treatment of choice, the neutropenia usually runs a cyclic course and surgery may await an optimum peak in peripheral white cell count. If severe neutropenia persists, surgery becomes mandatory. Antibiotics are required to combat present infections and prevent operative wound infection. Whole blood transfusions are utilized to replace the consistently noted blood volume deficits and thereby lessen the surgical risk.

Primary Splenic Panhematopenia. The simultaneous occurrence of thrombopenia, neutropenia, and hemolytic anemia due to hyperactivity of the spleen has been delineated as a clinical entity called splenic panhematopenia. Although the degree of the various cytopenias may differ their mechanism of development is considered similar.

Symptoms of panhematopenia vary being somewhat dependent upon the severity of the specific cytopenia. Weakness, malaise, scattered recurrent infections, easy bruising, petechiae and jaundice are the most common symptoms. When platelet counts are markedly lowered, spontaneous mucosal hemorrhage ensues. Splenomegaly is a consistent finding. The differential diagnosis may be difficult, hypoplasia of bone marrow and secondary hypersplenism must be considered. A compensatory hyperplasia of all the cellular elements of the bone marrow must be found to confirm the diagnosis. Even then, lymph node biopsy may lead to the discovery of another systemic disease process, changing the diagnosis to secondary hypersplenism.

Splenectomy is indicated. The indications

and contraindications to preoperative transfusions, antibiotics, adrenocorticotropin (ACTH) or cortisone and the decision of optimum time for surgery are problems trying the judgment of a competent hematologist. Surgical results will depend upon the adequacy of preoperative evaluation.

Secondary Hypersplenism. During the course of certain chronic disease processes which involve the reticuloendothelial system, associated hypersplenism occasionally develops. This produces cytopenias identical with those already described under the various forms of primary hypersplenism. The typical features are those of the various specific blood cytopenias or panhematopenia with a definite compensatory bone marrow hyperplasia. Secondary thrombocytopenic purpura, hemolytic anemia, neutropenia and pancytopenia have all been recognized. Since complications of the hypersplenic cytopenias can be expected, splenectomy is indicated.

The chronic disease processes with which hypersplenism has been associated have been listed under the general remarks on hypersplenism. Secondary splenic anemia of the hemolytic type is the most common form of secondary hypersplenism. In a series of 471 patients with hypersplenism seen over a twelve-year period, 206 had secondary hypersplenism. This included 109 patients having secondary hemolytic anemia, thirty-five with secondary splenic neutropenia, thirty-five with secondary splenic panhematopenia and forty-one with secondary thrombocytopenic purpura.

The diagnostic criteria in secondary hypersplenism are similar to those of the primary form of this syndrome. Signs and symptoms vary widely and may be complicated by those of the primary disease. The spleen is almost invariably enlarged to a much greater degree than in patients having primary hypersplenism. Splenomegaly is due in large part to involvement in the primary chronic disease process.

If surgical mortality and morbidity are to be kept low great attention must be given to preoperative preparation in these subjects. Owing to the nature of the diseases with which hypersplenism is associated, most of the patients are poor surgical risks. They are generally of an older age group often with much deterioration resulting from the original disease. The principles of surgery include attention to details in the preoperative preparation with the establishment of optimum nitrogen balance, blood volume

levels and pulmonary reserve. Close cooperation with a hematologist is mandatory. Although splenectomy cannot be expected to affect the ultimate outcome in the chronic disease with which secondary hypersplenism is associated, the hematologic improvement following splenectomy after adequate preparation strongly justifies surgery even in many debilitated patients.

ACQUIRED HEMOLYTIC ANEMIA

While the etiologic basis of primary and secondary hemolytic anemia is ascribed to a defect in the red cell and a selective sequestration in the spleen, the mechanisms of acquired hemolytic anemia include the factors of increased red cell fragility, destructive agents in the blood serum and erythrophagocytosis. The overproduction of serum hemolysins unantagonized by antihemolysins results in red cell destruction. Autoagglutinins, autohemolysins and isohemolysins have all been found in the blood serum.

Hyperactivity of the reticuloendothelial system is probably the cause of some cases of hemolytic anemia. The spleen may therefore be only one part of this system causing abnormal red cell destruction. Unless the spleen is of great importance in the individual case, at times a diagnostic problem even to the most astute hematologist, splenectomy will be of little value.

The course of this disease is usually more severe than that of congenital hemolytic anemia. Red cell counts are usually below 2,000,000 and hemolytic crises may cause anemia beyond the regenerative capacity of the bone marrow. Red cell fragility in saline solutions may not be greatly altered. The spleen is usually greatly enlarged. Reticulocytosis is marked and normoblasts may be found in the peripheral blood. Autoagglutinins are often present and the blood serum may contain hemolysins. Reaction to the antihuman globulin test (Coombs) is usually positive.

When the cause of excessive hemolysis cannot be accurately determined, splenectomy is usually considered. At least in part, the anemia in many cases is directly due to splenic hyperactivity. Results of splenectomy cannot always be accurately determined preoperatively. Once the diagnosis is established, adrenocorticotrophic hormone (ACTH) or cortisone should be given a trial of therapy. Failure of response to these drugs initially or failure of response to an exacerbation after good initial response, warrants splenectomy. Response of acquired hemo-

lytic anemia to splenectomy occurs in only about 50 per cent of the patients, however, in those failing to respond, ACTH or cortisone may be of benefit after splenectomy.

CYSTS

Cysts of the spleen are classified as non-parasitic or parasitic. All are rare. The pathologic and clinical features of various cysts have been reviewed by Fowler but few other reports occur in the American literature.

Nonparasitic cysts may be divided into primary (true) and secondary (pseudo-cysts) depending upon whether an epithelial or mesothelial lining exists. They may be of various sizes and either multiple or single. The more common type is the single, subcapsular secondary cyst, usually resulting from degeneration in an infarct or tumor or the breakdown of a hematoma.

The hydatid cyst is the only parasitic cyst. It occurs in less than 2 per cent of patients with the echinococcus infection.

Symptoms vary with the size and type of cyst. Most of the smaller cysts are asymptomatic, while the larger ones cause abdominal discomfort and are confused with omental, pancreatic or ovarian cysts. Calcification of the cyst wall may simplify the diagnosis. Eosinophilia and a positive reaction to the skin test may differentiate hydatid from nonparasitic cysts.

Treatment consists of splenectomy if the cyst is centrally located, and marsupialization for drainage, if the cyst is located peripherally and associated with many adhesions. Splenectomy carries a low mortality and is the treatment of choice in most cases. Aspiration, when hydatid cyst is suspected, is dangerous since spillage of cyst contents causes an anaphylactoid reaction.

TUMORS

Tumors of the spleen, both primary and secondary are even more rare than cysts. All types have been described and include angelioma, lymphoma, endothelioma, reticulum cell sarcoma, dermoid epithelial cyst, mesothelial inclusion cysts, fibrosarcoma, fibroma, and leiomyoma. The only important benign tumor is hemangioma and the only important malignant tumor is the sarcoma.

Tumors of the spleen, both primary and secondary rarely produce symptoms. Nausea, constipation, weight loss and left upper abdominal pain may be noted. The spleen is rarely palpated except when there is a malignant inoperable tumor. Splenectomy is the

treatment of choice and leads to cure for benign tumors, carrying a mortality of about 2 per cent. Since sarcoma is the most common malignant tumor, the operative mortality is higher and the five-year cure rate practically zero.

ANEURYSM OF THE SPLENIC ARTERY

The rare finding of aneurysm of the splenic artery at autopsy is even less often noted clinically despite the more recent use of contrast arteriography. This aneurysm is the second most common within the abdomen, occurring more often in women than in men. It may be due to arteriosclerosis, trauma or congenital defects, the majority being due to arteriosclerosis. Spontaneous rupture may occur and produce a fatal outcome.

The symptoms vary but pain, nausea, anorexia, weight loss and occasionally a palpable mass have been noted. These symptoms usually simulate those of chronic pancreatitis, so that diagnosis has been accurately made in less than 10 per cent of the cases. Calcification in the walls of the aneurysm is the only diagnostic radiographic finding, other than that disclosed by the use of arteriography.

Treatment consists of excision of the aneurysm if possible, or proximal and distal ligation when excision is considered unsafe. Splenectomy need not necessarily follow ligation of the splenic artery.

INFECTIONS

During recent years, acute infections involving the spleen have been extremely rare, probably owing to widespread use of antibiotics. Chronic infectious processes, primarily or secondarily involving the spleen, are somewhat more common. Abscess of the spleen may develop with infection in an area of hemorrhage or devitalized splenic pulp. During its early stage the abscess remains within the capsule and may cause no symptoms. With enlargement, perisplenitis may cause pain, chills, fever and splenomegaly and if drainage is not instituted or splenectomy performed, the possibility of splenic rupture exists, carrying a high mortality. If diagnosis can be made early splenectomy is the treatment of choice.

Several chronic infections are characterized by splenomegaly. Splenectomy may produce remission of general symptoms, as in Felty's disease, or may be beneficial to the patient in whom secondary hyper-

splenism is associated. The chronic infections with which the spleen may become involved include Felty's syndrome, malaria, tuberculosis and Boeck's sarcoid.

Felty's syndrome is a clinical entity consisting of splenomegaly, anemia and neutropenia, and occurring most often in association with chronic rheumatoid arthritis in women between the ages of forty and sixty years. The pathologic findings in the spleen are not characteristic and the etiologic basis is controversial. The leukopenia may be the result of multiple chronic infections or a splenic hyperfunction of unknown origin. Leukopenia occurs in variable degrees and the arthritis is of several years duration prior to the onset of the syndrome. The symptoms consist of weakness, frequent episodes of fever, weight loss, swollen and painful joints and multiple skin or oral infections.

Splenectomy has given variable success but in a number of patients has produced relief not only of the neutropenia and anemia, but also marked improvement in the joint symptoms. Adrenocorticotropin (ACTH) and cortisone may also give temporary and, at times, long standing symptomatic relief.

The involvement of the spleen in patients with malaria is well known. In certain instances of chronic malaria, the disease may not be diagnosed from peripheral blood smears alone, whereas numerous parasites are harbored within the spleen. No significant diagnostic criteria of malarial infection are present, other than splenomegaly, with out other known cause in patients who have been known to have malaria. The only problem presented by the enlarged spleen is the occasional discomfort which it may cause and the susceptibility to either spontaneous or easy traumatic rupture, both of which are direct indications for splenectomy.

As with other portions of the reticuloendothelial system, the spleen may become involved in the chronic systemic disease processes, *tuberculosis* and *Boeck's sarcoid*. The importance of this involvement lies in whether the disease is active and whether it produces hematologic complications. Thrombocytopenia is common but may not necessarily be due to hypersplenism. Other forms of hypersplenism secondary to these chronic disease processes, however have been reported and the results of splenectomy in the relief of various cytopenias have been quite favorable. The only other indication for surgery in these patients is the occa-

sional markedly enlarged spleen, causing enough abdominal discomfort to warrant its removal.

MISCELLANEOUS DISEASES

Banti's Disease. Portal hypertension is the common denominator of the group of hepatosplenopathies loosely termed Banti's disease. Whether intrahepatic (cirrhosis) or extrahepatic (thrombosis) portal obstruction is present, the splenic changes are similar. The spleen is invariably enlarged. The capsule is thickened and there are usually numerous vascular adhesions. The splenic parenchyma is firm, deep red in color and markedly congested. Fibrosis with hyperplasia of the reticuloendothelial lining of dilated sinuses is prominent.

Mediterranean Anemia. This is a familial chronic hemolytic anemia, also called erythroblastic or Cooley's anemia, which occurs usually in children descended from parents of Mediterranean countries. The abnormalities producing anemia consist of a defect in hemoglobin formation and an increased resistance of the red cells to hypotonic saline. Characteristic thickening of the frontal bone with depression of the nasal bridge and a mongoloid slant to the eyes help to differentiate this disease. The microcytic anemia may be associated with leukocytosis and is invariably associated with marked hyperplasia of the bone marrow. Splenectomy has been advocated when marked hemolytic activity is present. Most patients will at least maintain a higher red cell count and hemoglobin level and require fewer transfusions following splenectomy.

Storage Diseases. Several diseases due to abnormal lipid metabolism may produce splenomegaly of surgical importance. These include Gaucher's disease, Niemann-Pick disease and Hand-Schüller-Christian disease, in which there is abnormal reticuloendothelial storage respectively of cerebroside, sphingomyelin and cholesterol. Only in Gaucher's disease is splenectomy frequently indicated.

Idiopathic splenomegaly is another term formerly used to denote Gaucher's disease. Patchy pigmentation of the face, conjunctival thickening, hepatomegaly, splenomegaly, flaring of the distal end of the femur, thrombocytopenia and neutropenia may suggest the diagnosis which may be confirmed by the findings of foam cells in the bone marrow. The thrombocytopenia and neutropenia are often due to hyper-

splenism. The diagnosis of Gaucher's disease as the cause of secondary hypersplenism is often not made, however until the time of splenectomy. Thrombocytopenia and neutropenia may be greatly relieved by splenectomy but improvement in anemia is only temporary.

READING REFERENCES

- Carter, B. V.: Combined Thoraco-abdominal Approach with Particular Reference to Its Employment in Splenectomy. *Surg. Gynec. & Obst.* 84: 1019, 1947.
- Curtis, G. M., and Movitz, D.: Surgical Significance of Accessory Spleens. *Ann. Surg.* 123: 378, 1946.
- Dalre, J. V.: Acquired Hemolytic Anemia; with Special Reference to the Antiglobulin (Coombs) Reaction. *Blood* 8: 813, 1953.
- Dameshek, W. and Welch, C. S.: Hypersplenism and Surgery of the Spleen. New York, Grune & Stratton, 1952.
- Doan, C. A., Wiseman, B. A. and Bouroude, B. A.: Hypersplenic Cytopenic Syndromes: A Twenty-Year Experience with Special Reference to Splenectomy. *Proc. Internat. Soc. Hematol.* 428, 1958.
- Doan, C. A., and Wright, C. S.: Primary Congenital and Secondary Acquired Splenic Panniculitis. *Blood* 1: 10, 1946.
- Dodd, M. C., and others: The Immunologic Specificity of Anti-Serum for Trypsin-Treated Red Blood Cells and Its Reactions with Normal and Hemolytic Anemia Cells. *Blood* 8: 640, 1953.
- Dumphy, J. E.: Splenectomy for Trauma. *Am. J. Surg.* 71: 450, 1948.
- Ellison, E. H.: Spontaneous Rupture of the Diseased Spleen. *Arch. Surg.* 59: 299, 1949.
- Evans, R. D., and others: Primary Thrombocytopenic Purpura and Acquired Hemolytic Anemia. *Arch. Int. Med.* 87: 48, 1951.
- Fowler, R. H.: Hydatid Cysts of the Spleen. *Internat. Abstr. Surg.* 96: 105, 1953.
- Fowler, R. H.: Non-parasitic Benign Cystic Tumors of the Spleen. *Internat. Abstr. Surg.* 96: 209, 1953.
- Glenn, F., Cornell, C. N., Smith, C. H. and Schellman, L.: Splenectomy in Children with Idiopathic Thrombocytopenic Purpura, Hereditary Spherocytosis, and Mediterranean Anemia. *Surg. Gynec. & Obst.* 99: 669, 1954.
- Harbison, S. P., Patton, V. G., and Moyer, J. P.: Spontaneous Rupture of the Spleen. *Ann. Surg.* 139: 248, 1954.
- Harrington, W. J. and others: Immunologic Mechanisms in Idiopathic and Neonatal Thrombocytopenic Purpura. *Ann. Int. Med.* 33: 433, 1953.
- Israels, M. C. G.: The Reticuloses. A Clinicopathological Study. *Lancet* 2: 525, 1953.
- Kiernan, P. C. and Hunter, O. B., Jr.: The Use of ACTH in Hypersplenism. *S. Forum* 1: 71, 1951.
- Kissmeyer-Nielsen, F.: Demonstration of a Thrombocytopenic and a Leukopenic Factor in the Blood of Patients with Thrombocytopenia and Leukopenia. *Acta haemat.* 9: 337, 1953.
- LeRoy, G. V.: Diseases of the Spleen. *M. Clin. North America* 37: 181, 1953.
- Lichtman, H. C., and others: Studies on Thalassemia: Part II: The Effect of Splenectomy in Thalassemia.

- Major with Associated Acquired Hemolytic Anemia. *J. Clin. Invest.* 32:1329 1953.
- Loeb, V., Seeman, W. B., and Moore, C. V.: The Use of Thorium Dioxide Solution (Thorotrast) in the Rontgenological Demonstration of Accessory Spleens. *Blood* 7:904 1952.
- Medoff, A. S., and Bayrd, E. D. Gaucher's Disease in 29 Cases, Hematologic Complications and Effect of Splenectomy. *Ann. Int. Med.* 40:481 1953.
- Nelson, R. P.: Cyclic Neutropenia with Giant Follicular Lymphoblastoma and Lymphosarcoma. *Blood* 8:923, 1953.
- Owen, J. C., and Coffey, R. J.: Aneurysm of the Splenic Artery Including Report of Six Additional Cases. *Internat. Abstr. Surg.* 97:313, 1953.
- Ponder, E.: Certain Hemolytic Mechanisms in Hemolytic Anemia. *Blood* 6:559, 1951.
- Reid, S. E., and Lang, S. J.: Abscess of the Spleen. *Am. J. Surg.* 68:912, 1954.
- Riley, J. F.: The Floating Spleen. *Brit. M. J.* 1:1335 1953.
- Robb-Smith, A. H. T.: The Reticuloses. *Lancet* 2:619 1953.
- Rogers, H. M., and Dann, P.: Chronic Neutropenia. Report of Five Cases Treated by Splenectomy. *Am. Surgeon* 19:1071 1953.
- Roussetot, L. M., and Stein, C.: Malignant Neoplasms of the Spleen, Primary and Secondary. *S. Clin. North America* 33:493, 1953.
- Stefanini, M., and Dameshek, W.: Idiopathic Thrombocytopenic Purpura. *Lancet* 2:209, 1953.
- Ungar, G.: Endocrine Function of the Spleen and Its Participation in the Pituitary Adrenal Responses to Stress. *Endocrinology* 37:329 1945.
- Walker, R. M., Middlemiss, J. H., and Nanson, E. M.: Portal Venography by Intrasplenic Injection. *Brit. J. Surg.* 40:392, 1953.
- Whipple, A. O., Parpart, A. J., and Chang, J. J.: A Study of the Circulation of the Blood in the Spleen of the Living Mouse. *Ann. Surg.* 140:268, 1954.
- Wilkinson, J. F.: Investigation of Hemolytic Anemia. *Brit. M. J.*, 2:4876 1438, 1954.
- Whisman, B. L., and Doan, C. A.: Primary Splenic Neutropenia. *Ann. Int. Med.* 16:1097 1942.
- Zollinger, R. M., Martin, M. M., and Williams, R. D.: Surgical Aspects of Hypersplenism. *J.A.M.A.* 149:24, 1953.

THE URINARY SYSTEM

By FRANK HINMAN JR. M.D.

FRANK HINMAN JR., is a native Californian who received his college education at Stanford University and his medical education at Johns Hopkins Medical School. He had basic training in general surgery before his graduate training in urologic surgery. He is an Associate Clinical Professor of Urology at the University of California School of Medicine. He has made significant contributions in his experimental studies upon ureteral regeneration.

A picture of an early surgeon operating on the urinary tract (Fig 1) illustrates two things which have made urology a special branch of surgery. One the urologist uses special instruments and, two even though he is treating a disease of the lower and middle urinary tracts (in this case removing a stone) he is simultaneously relieving the kidneys of infection and back pressure. In the field of urology, ultimate or exact diagnosis is usually made by special techniques for instance, looking into the bladder (cystoscopy) or examining the kidneys with x-rays (pyelography or urography). Diseases of the urinary system are interrelated—what affects one part commonly affects other parts.

A diagram of the urogenital system shows that obstruction at one place along the system causes damage in other parts. As an example, in Figure 2 it is seen that obstruction at the meatus sets up a train of changes: stony infection and stasis with stone formation and, finally renal failure. Renal damage alone is not always reversible and may ultimately cause death.

There is a purpose, therefore in insisting on a reasoned and systematic approach to the diagnosis of urinary diseases.

THE DIAGNOSTIC STEPS IN URINARY TRACT DISEASE MAKING A PRESUMPTIVE UROLOGIC DIAGNOSIS

A physician need not own a cystoscope to examine a patient with urinary complaints and arrive at a satisfactory presumptive diagnosis. Because the parts of the urinary tract are interrelated, deductions are possible from findings at a distance from the seat of the trouble. The tools for making these deductions are common tools and if all are used in every urologic case, few disorders will be overlooked. These tools are

1. History with emphasis on urologic features

2. General physical examination

3. Examination of the external genitalia

4. Examination of the urine

5. Rectal examination and vaginal examination of female

6. Abdominal examination

7. Total renal function tests

8. Roentgenographic study

History The art of obtaining the urologic history is to elicit all the essential clues so that the subsequent diagnostic steps will be confirmatory rather than exploratory. The complaint is the best lead, especially in

urologic diagnosis for urologic symptoms are likely to be well localized both in time and in position

Certain symptoms should be inquired about particularly

General symptoms. High spiking fever with chills, not chilliness is characteristic of pyelonephritis. Gastrointestinal symptoms often accompany acute or chronic obstructions of the kidney since they are supplied by the same nerve tracts. Weight loss increases the seriousness of other complaints.

Pain can give important leads. General areas of reference are the rule. Renal pain (Fig. 3A) has a dull aching quality due to distention of the renal capsule as in pyelonephritis or less often, in acute obstruction. It is referred to the soft triangle between the lower rib and the vertebral muscles and extends around anteriorly in the area corresponding to the tenth, eleventh and twelfth dermatomes. Pain which is even more localized is suggestive of perirenal involvement.

Acute obstruction to the outflow of the kidney produces renal colic, which is sharp

and severe but with radiation similar to that produced by capsular distention. Chronic or mild degrees of upper tract obstruction may give only vague gastrointestinal symptoms these may lead to fruitless investigations of the digestive tract.

Ureteral pain (Fig. 3C) is similar to but lower than, renal pain. This would be expected because of the lower innervation of the ureter. Low ureteral pain for example from a stone at the point where the ureter enters the bladder is referred down the urethra and mimics the pain of prostatitis or posterior urethritis.

Vertical pain (Fig. 3B) arises typically from increased intravesical pressure, as in overdistention of the bladder or from irritative stimuli within the bladder which in turn produce spasm and thence pain. This pain is usually referred to the suprapubic area. Irritative lesions at the neck of the bladder cause frequent urination and pain referred along the urethra. Prostatic pain may be referred to the suprapubic area and stimulate bladder pain.

Prostatic pain (Fig. 3D) is referred prin-



Figure 1 From E. Deanos, *Histoire de l'Urologie*. Figure 66 from *L'Encyclopédie française d'Urologie*, Vol. I, Paris, Octave Doin et Fils, 1914.

THE URINARY SYSTEM

By FRANK HINMAN JR M D

FRANK HINMAN JR. is a native Californian who received his college education at Stanford University and his medical education at Johns Hopkins Medical School. He had basic training in general surgery before his graduate training in urologic surgery. He is an Associate Clinical Professor of Urology at the University of California School of Medicine. He has made significant contributions in his experimental studies upon ureteral regeneration.

A picture of an early surgeon operating on the urinary tract (Fig. 1) illustrates two things which have made urology a special branch of surgery. One the urologist uses special instruments and, two even though he is treating a disease of the lower and middle urinary tracts (in this case removing a stone) he is simultaneously relieving the kidneys of infection and back pressure. In the field of urology ultimate or exact diagnosis is usually made by special techniques for instance looking into the bladder (cystoscopy) or examining the kidneys with x rays (pyelography or urography). Diseases of the urinary system are interrelated—what affects one part commonly affects other parts.

A diagram of the urogenital system shows that obstruction at one place along the system causes damage in other parts. As an example, in Figure 2 it is seen that obstruction at the meatus sets up a train of changes atony infection and stasis with stone formation and, finally, renal failure. Renal damage alone is not always reversible and may ultimately cause death.

There is a purpose therefore, in insisting on a reasoned and systematic approach to the diagnosis of urinary diseases.

THE DIAGNOSTIC STEPS IN URINARY TRACT DISEASE

MAKING A PRESUMPTIVE UROLOGIC DIAGNOSIS

A physician need not own a cystoscope to examine a patient with urinary complaints and arrive at a satisfactory presumptive diagnosis. Because the parts of the urinary tract are interrelated, deductions are possible from findings at a distance from the seat of the trouble. The tools for making these deductions are common tools and if all are used in every urologic case, few disorders will be overlooked. These tools are

- 1 History with emphasis on urologic features
- 2 General physical examination
- 3 Examination of the external genitalia
- 4 Examination of the urine
- 5 Rectal examination and vaginal or amniotomy of female
- 6 Abdominal examination
- 7 Total renal function tests
- 8 Roentgenographic study

History The art of obtaining the urologic history is to elicit all the essential clues so that the subsequent diagnostic steps will be confirmatory rather than exploratory. The complaint is the best lead, especially in

bleeding is within the urethra, prostate or the vesical neck. Similarly terminal hematuria indicates lower tract bleeding, since the blood is squeezed out during the peristaltic emptying of the urethra.

Cloudy urine Causes phosphate crystals in an alkaline urine, especially in urine after standing, white blood cells, and bacteria. The addition of dilute acid will clear the urine of crystals.

Sterility Causes impotency or improper coital technique or timing, infection of accessory sex organs, obstruction to passage of sperm, usually congenital obstruction of vas or epididymis or infectious epididymitis, defective spermatogenesis due to developmental defect in testis, increased scrotal temperature or previous testicular disease.

Impotency Causes lack of sexual desire or libido, inability to achieve erection or lack of potency. Accompanying disorders may be premature ejaculation, inability to ejaculate or early subsidence of erection. Few of these disorders arise from organic causes.

The urologic history, then, often gives the first presumptive evidence of the nature and location of disorder in the urinary system.

General Physical Examination. For the urologic patient evaluation of the cardiovascular system should be included. Other areas distant from the genitourinary tract should also be examined. If the patient's history suggests the presence of genitourinary neoplasm, examination of the supraclavicular nodes is in order. The presence of testicular tumor would necessitate examination of the breasts.

Examination of the External Genitalia. A systematic survey of the visible and palpable genitalia is important to the examiner.

The penis is inspected, and particular note is made of urethral discharge, meatal anomaly causing stenosis, or ulceration. If any discharge is present, it is expressed and smeared on a slide and air dried, heat fixed, stained with methylene blue and examined. Mixed organisms and gonococci are looked for particularly.

The scrotal contents are palpated by using both hands simultaneously. This allows separation of each testis from its accompanying epididymis in order to distinguish each structure absolutely. Testicular tumors will be within the testis whereas epididymal enlargement will involve one or both poles of

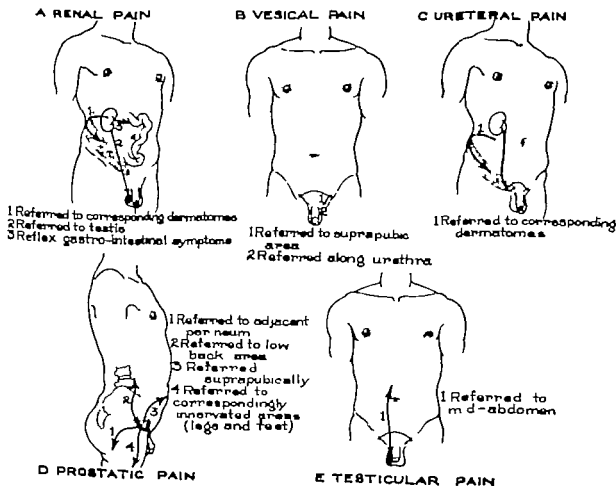


Figure 3. Referral of genitourinary pain.

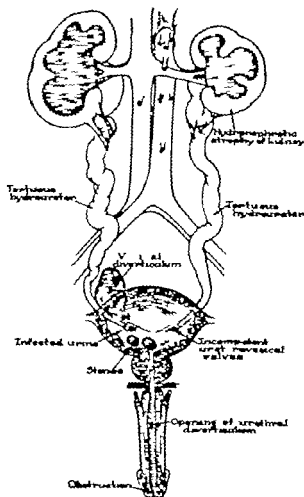


Figure 2. Obstruction by stricture at the urethral meatus can result in disease throughout the urinary tract.

cipally to the perineum but may be referred to the low back, the epididymides and down the legs. The source is congestion and inflammation in the prostatic acini and ducts.

Testicular pain (Fig. 3E) is referred to the midabdomen, to the site of the embryologic origin of the gonads. Pain perceived in the testis may arise from the prostate and seminal vesicles, or from the terminal ureter. Renal pain also may be referred to the testis, by the same route along which testicular pain is referred upward.

As can be seen, the referral of urologic pain is not precise so that it will be more often suggestive than diagnostic.

Other symptoms with special urologic significance are:

Frequency Causes (1) excessive fluid intake; (2) small bladder capacity; (3) hyperactive bladder reflexes, of neurogenic origin, which increase motor activity or irritation in the bladder or posterior urethra, which increases the sensory stimuli.

Urgency Cause irritative lesions in blad-

der or prostate which prematurely stimulate the sensory side of the voiding reflex arc.

Nocturia. Causes excessive fluid intake, incomplete emptying of bladder so that capacity and increased intravesical pressure lead to more frequent desire to void, mobilization of dependent edema. Other factors are involved which are not understood.

Dysuria. Causes irritation, especially of the trigone and prostatic urethra by infection, stone or chemicals.

Strangury is very severe dysuria, especially during increasingly frequent passage of smaller and smaller quantities of urine.

Oliguria. Causes are reduced fluid intake, excessive fluid loss (perspiration, vomiting, diarrhea), renal tubular failure, cardiac failure (rarely) and hypotension.

Anuria. Causes of anuria include prolonged hypotension, renal tubular failure and obstruction to the outflow of urine (both ureters or below the neck of the bladder).

Hesitancy. The cause of this condition is inability to relax the bladder neck because of interference by the hypertrophied prostate. Also intravesical pressure must be raised to a greater height before urine is forced past the obstructing prostate gland.

Dribbling. Since resistance to outflow is increased by prostatic hypertrophy, the detrusor muscle continues to contract at the time the stream stops, rather than immediately relaxing as is normally the case when there is no residual urine. This continuing contraction is not strong enough to maintain sizable urinary flow but is enough to force out slowly a few more cubic centimeters of urine.

Incontinence Causes (1) stress incontinence from incompetent sphincter muscles, due to relaxation of the pelvic floor or to paralysis or injury; (2) urgency incontinence which is an extreme form of urinary frequency and urgency such that the voiding reflex is stimulated so strongly that voluntary control of the sphincter is intermittently lost; (3) overflow incontinence, when the pressure in the overfilled bladder is higher than the resistance of the sphincter so that intermittent leakage occurs whenever intra-abdominal pressure rises; (4) fistula, and (5) reflex incontinence in the neurogenic bladder in which filling sets off reflex emptying.

Hematuria. Causes tumors, stones, infection, tuberculosis injury. Dark blood suggests upper urinary tract bleeding or residual urine. Initial hematuria with the urine clearing during voiding means that the source of

It is necessary to make trial assumptions:

a. assume 100 cc. residual urine; then the 30' excretion would have to contain 90 per cent of the dye since each 50 cc. contains 30 per cent dye. This would be an abnormally high 30' excretion so it is impossible for the residual urine to be this great.

b. assume 50 cc. residual urine; then 30' excretion would have to contain 60 per cent of the dye. This is a "high normal" but possible percentage excretion. The higher level of the second, 60' specimen results from the addition of the dye left over from the first voiding.

c. assume no residual urine; but the 30' specimen has to contain more dye than the 60' specimen.

It can be concluded that this patient has less than 100 cc. and more than 0 cc. of residual urine, and probably has a little less than 50 cc.

Röntgenography A plain film of the abdomen (kidneys, ureter and bladder—KUB) of every patient with symptoms suggestive of disease in the middle or upper tract of the urinary system is obtained.

The film should be read systematically since it may reveal atrophy of one kidney with compensatory hypertrophy of the other renal tumor or renal displacement by an adrenal tumor or metastatic disease, as well as stones in the urinary tract.

Often, greater delineation of the middle and upper urinary tract is necessary. An intravenous urogram (Fig. 7) gives a clearer

outline of the kidney itself and, of course, outlines the drainage system, often clearly and in detail. A postvoiding film allows detection of residual urine, resulting from, for example, prostatic obstruction. Urograms are made before resorting to instrumentation, since they usually make it possible to rule out upper tract abnormalities. If they show abnormalities, retrograde ureteral catheterization and pyelography may be required.

THE PRESUMPTIVE DIAGNOSIS

If the eight steps outlined above are gone through thoughtfully and faithfully for each patient, few diagnostic errors will be made and, in most cases, a presumptive diagnosis which is based on good reasoning will be reached.

For example, a patient is examined with (1) a history of colicky right-flank pain radiating to the testis, (2) abdominal distention, (3) normal external genitalia, (4) microscopic hematuria with bacteriuria, (5) enlarged prostate, discerned by rectal examination, (6) somewhat distended bladder judged by percussion, (7) reduced total renal function evaluated by PSP test yielding percentages which indicate the presence of residual urine and (8) a calcific density along the course of the right ureter. These eight steps allow one to arrive simultaneously at two sound presumptive diagnoses: right ureteral calculus and benign prostatic hypertrophy with obstruction. Instrumental steps, which may be both diagnostic and therapeutic, can now be done rationally.



Figure 7 Detail of intravenous urograms compared with a three-dimensional drawing and a coronal section of the same normal kidney. Note cupped calyces on their infundibular stalks and rounded pelvis with funnelled ureteropelvic junction.

30	60 cc.	50%
60'	75 cc.	15%
Total	135 cc.	65%

Admittedly small errors are inherent in such a method, but they do not exceed 10 per cent and are more than outweighed by the advantages of availability and economy. The results are immediately available to the examiner for correlation with other findings while the patient is still at hand. Finally it is of value in detecting residual urine which is important in the urologic evaluation of patients.

Residual urine from incomplete emptying of the bladder cannot be measured directly by the PSP test but can be inferred if normal renal function is assumed. If this assumption cannot be made the test separates normal renal function and no residual urine from decreased renal function, residual urine or both. This separation is of the utmost importance for it detects those patients who require further urologic study.

As an example a normal patient receives a PSP test while he is dehydrated, and again while he is hydrated usually by drinking two glasses of water at the start of the test.

Dehydrated		
30	30 cc.	50%
60	30 cc.	15%
Total	60 cc.	65%

Hydrated		
30'	180 cc.	50%
60	180 cc.	15%
Total	360 cc.	65%

Note that with normal renal function without residual urine, hydration makes no difference in the excretion of the dye.

Now consider a patient with normal function but with 100 cc. of residual urine in the bladder as a result of incomplete emptying due to prostatic obstruction.

Dehydrated.		
30	30 cc. 11%	The normal 50% PSP was excreted by tubules in 30 cc. of urine, but was diluted by 100 cc. residual bladder urine to 130 cc., of which 30 cc. was voided.
		130 cc. 50% 30 cc. x
		x = 11%
60'	30 cc. 12.5%	15% PSP was excreted by tubules in 30 cc. urine, but was diluted by 100 cc. residual bladder urine which already contained 39% PSP (50% minus 11%).
		130 cc. 39 + 15% 30 cc. x
		x = 12.5%

Total 60 cc. 23.5%

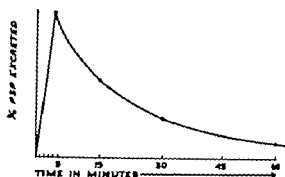


Figure 6. Curve of PSP excretion from the kidney. Lesser amounts are put out as the blood is cleared of the dye.

Note two things (1) the total PSP excretion is low in spite of normal kidney function and (2) the second specimen contains a little more PSP than the first. Since PSP is put out by the tubules in proportion to the concentration presented to them, the kidneys always excrete more in early periods than in later periods (Fig. 6) but if residual urine is present, this relationship will appear to be reversed.

If the same patient is hydrated, has normal renal function but 100 cc. of residual urine

Hydrated		
30	180 cc. 32%	180 cc. renal output containing 50% PSP diluted by 100 cc. residual bladder urine = 280 cc. then voided 180 cc. of the mixture.
		280 cc. : 50% = 180 cc. x
		x = 32%
60'	180 cc. 21%	180 cc. renal output containing 15% PSP diluted by 100 cc. residual urine containing 39% PSP (50% minus 11%).
		280 cc. 33% = 180 cc. x
		x = 21%
Total	360 cc.	53%

A comparison of this last example with the preceding one demonstrates how small urine volumes emphasize the presence of residual urine. During dehydration, the test indicated either poor renal function or residual urine. After hydration, it demonstrated good renal function but did not give information about residual urine. In order to detect residual urine with the PSP test, limit fluids in order to measure renal function, provide a large fluid intake.

That the PSP test will often give an estimate of the actual volume of residual urine can be shown by an example.

30	50 cc.	30%
60'	50 cc.	30%
Total	100 cc.	

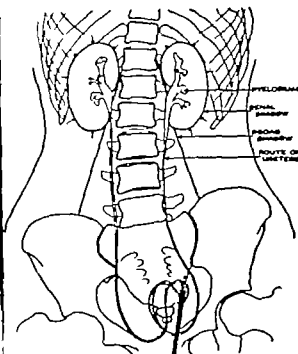


Figure 8. Retrograde pyelogram, drawing, overlying cystoscope and ureteral catheters show principal landmarks.

required and the best that medical treatment will assure is arrest, not cure.

Or is it from obstructive renal disease? Then instrumental diagnosis and surgical treatment will locate and remove the obstruction and so allow consequent improvement in renal function (Fig. 9)

INTRINSIC OBSTRUCTIVE

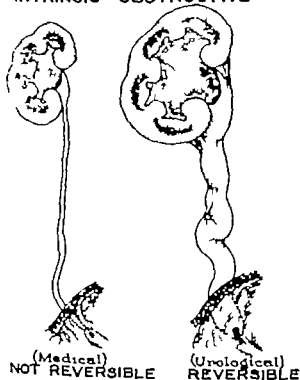


Figure 9. Comparison of medical renal disease, which is not helped by instrumentation or operation, with urologic renal disease, which is.

Table 1 outlines the conditions causing obstruction.

HYDRONEPHROSIS

The ultimate effect of obstruction anywhere along the urinary tract, from the urethral meatus to the ureteropelvic junction is hydronephrosis.

When the meatus is partially occluded

1. The ischio-cavernosus muscle does not empty the urethra completely so dilatation and even sac-like outpouchings appear

2. The detrusor muscle of the bladder first contracts with more vigor to push past the obstruction and hypertrophies. The muscle bundles thicken, the submucosa between weakens and gives way forming diverticula.

When this compensatory process can no longer balance the obstruction, decompensation occurs. The detrusor muscle is no longer able to empty the bladder completely and urine remains after voiding.

Finally if the obstruction is not relieved, the muscle, being overstretched, loses its contractibility and atony results.

3. The ureteral pressure increases, since each peristaltic effort must force urine past the thickened bladder wall so that the ureter too, goes through the stages of hypertrophy decompensation and atony (Fig. 10) The lower ureter decompensates first, leaving the upper ureter unprotected. The decompensated portions gradually extend upward, until the renal pelvis and finally the calyces are dilated, and residual urine remains in

CONFIRMING A PRESUMPTIVE DIAGNOSIS

Catheterization. The ability to pass a catheter through the urethra indicates that there are no urethral strictures. Catheterization also confirms the presence and permits measurement of the amount of residual urine if the PSP test gave equivocal results. It is not used to secure a sterile urine specimen from the male but may be so used in the female. Catheterization also precedes cystography which is filming of the bladder filled with a contrast medium, and is necessary for cystometrography.

The rationale for completing noninstrumental steps before introducing catheters or other instruments can be made obvious by one example. A patient with symptoms suggestive of urethritis or prostatitis is catheterized, in order to determine whether residual urine is present, immediately after his urologic history is taken. The catheter irritates the urethra, so that when the first glass of urine is collected, it contains red blood cells and massage of the prostate produces more blood cells. The chance has been lost for knowing if an underlying urethritis is present.

Catheterization in the male is done by cleansing the meatus, introducing a liquid or jelly anesthetic agent without pressure, waiting five minutes, gently introducing 5 or 10 cc. of lubricating jelly from a tube or syringe and then slowly inserting the catheter. Often the passage of the catheter is obstructed at the external urethral sphincter; gentle persistent pressure applied by the examiner allows it to proceed; each time the patient relaxes the reflexly contracted sphincter. It is good practice to prescribe an antispasmodic and a sulfonamide for twenty-four hours following any instrumentation in order to reduce irritation and to decrease the chance of provoking infection.

Urethral Exploration. If the catheter does not pass through the urethra, fine woven filiforms, to which larger metal followers are attached, may be introduced to determine the site and extent of the obstruction and to overcome it. Metal sounds are used for the same purpose in cases of strictures of larger caliber.

Pandendoscopy the visualization of the urethra with an instrument containing a lens and light, adds to our knowledge of the lesion in the urethra. Filling the urethra from the meatus with opaque jelly allows x-ray visualization which is especially valuable for detecting abnormalities in the posterior urethra.

Exploration of the Bladder. A cystoscope permits excellent views of the entire bladder wall, including the trigone and vesical neck. It is for this reason that cytologic study of bladder urine for tumor cells has not been very widely used. It is too easy to examine the bladder wall directly.

Upper Urinary Tract Studies. In most cases intravenous urograms give adequate and physiologic information about the upper urinary tract. However, if detail is lacking, if renal function is depressed or if infection must be traced to its source, retrograde ureteral catheterization is necessary.

Small catheters are passed via the cystoscope through the ureteral orifices and into the renal pelvis in order to collect specimens of urine from the separate pelvis. These specimens are utilized for microscopic examination, culture preparation and for differential function tests. They also allow retrograde injection of opaque media to produce x-ray pictures of great contrast of the renal pelvis, calices and ureters (Fig. 8).

Other studies are used for dealing with special problems. Percutaneous pneumograms outline the renal and adrenal areas with oxygen and produce sharp contrast on the x-ray film. Aortography demonstrates the renal arteries and vascular bed of the kidneys and is of special value in diagnosing vascular obstructions in hypertensive patients. Antegrade pyelography accomplished by inserting a needle directly into the dilated renal pelvis and injecting dye, can be used when retrograde filling is not possible. Cine-radiography using an image amplifier demonstrates dysfunction of peristalsis and of micturition.

Reaching the correct diagnosis in urology is not so much a matter of sagacity as it is of careful and systematic observation and use of precise techniques which lead inductively to the correct conclusion.

OBSTRUCTION OF THE URINARY TRACT

Obstruction is the watchword of urologists. Not only can it destroy the kidneys by back pressure alone, but it can indirectly damage the renal parenchyma by fostering infection and encouraging the formation of stones.

When poor renal function is detected by an impaired PSP excretion or later by an increase in the serum nonprotein nitrogen level, the physician asks himself

Is it due to intrinsic renal disease (Fig. 9)? In this case active intervention is not

all parts of the urinary tract. Renal secretory pressure now is the only force remaining to push past the obstruction.

4. The renal tubules dilate, but, more important, the arteries between the cortex and medulla are stretched and constricted by the pelvocaliceal distention of the pelvis and calices. This results in partial ischemia, which speeds the destruction of the tubules and glomeruli.

Secondary effects of obstruction are often as damaging as the obstruction itself (Fig. 11). The presence of residual urine allows a single contaminating bacterium to multiply before it is flushed from the urinary system, so that infection follows obstruction in the majority of cases. It is axiomatic that pyuria is due to obstruction until proved otherwise and bacilluria accompanied by relatively few white blood cells is virtually always good evidence for stagnation. Calculous formation occurs more readily after obstruction too. The nidus which would normally be passed is retained and is augmented by further precipitation in the presence of alkaline infection.

RENAL ADJUSTMENTS TO OBSTRUCTION

Backflow (Fig. 12) from the renal pelvis and calices into the tubules, veins and lymphatics probably begins as a safety mechanism during minor degrees of obstruction or diuresis. With more complete blockage, backflow carries back into the circulation only a small portion of the secreted urine. However these routes make possible a slow circulation of urine and prevent the simple atrophy that would occur should the urine have no egress.

Compensatory hypertrophy of the other

kidney begins as soon as one kidney is obstructed apparently in response to the doubled load of waste products circulating to it. After one kidney has been obstructed for two weeks the normal kidney has the functional power that both kidneys originally had.

DIAGNOSIS

The diagnosis of obstruction is made on the presumptive findings plus the instrumental findings. It can rarely be made from the history and symptomatology alone. In the final analysis, the pyelogram will give the most information concerning the degree of obstruction, degree of damage and the site of obstruction if it is in the upper tract. An x-ray film made ten or fifteen minutes after injection of the dye into the renal pelvis through a ureteral catheter will often give valuable information as to the site and degree of obstruction by showing the retention of dye after this interval. The normal kidney will empty itself in this period.

Two factors are of great importance in the study of a patient with hydronephrosis: the estimation of individual renal function and determination of the site of the obstruction. An estimation of the relative function of the two kidneys assesses the degree of damage to the hydronephrotic kidney to determine whether it is worth preserving and ascertains that the contralateral kidney is capable of supporting life in case of removal of the damaged kidney. If the damaged kidney is not capable of supporting life alone, it should be removed. Consideration of the effects of renal counterbalance will enter into the decision as to the proper treatment of hydronephrosis since repair of a very

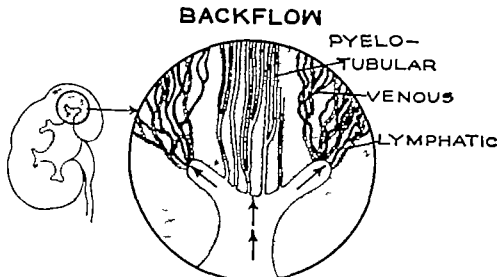


Figure 12. Obstruction distally causes urine to flow back up the tubules or into the veins or lymphatics at the fornices.

Table 1 Conditions Causing Obstruction

ANATOMIC LOCATION	CONGENITAL TYPES	ACQUIRED TYPES
<i>Lower tract</i> Urethral	Congenital phimosis Atracia Stricture (meatus) Diverticulum Abnormal openings and anomalies	Acquired phimosis Stricture Stone Tumor Diverticulum Fistula
Urethrovaginal	Prostatic urethral valves Hypertrophied verumontanum Contracture of the neck Hypertrophy of the neck	Prostatism Hyperplasia Cancer Contracture or median bar Cysts Essential sphincter spasm
Vesical	Anomalies Congenital diverticulum	Tumor Stone Diverticulum Hypertrophy of interureteric ridge Neurogenic bladder
<i>Upper Tract</i> Ureteral	Neurogenic bladder Valves, folds Structures Ureterocele Ectopic ureteral orifice	Ureterovesical conditions Stone, blood clots, etc. Tumor Strictures, ureteritis and perireteritis Seminal vesiculitis Pregnancy Valves (acquired) Pressure (extraureteral)
Pelvic and renal	Ureteropelvic junction obstruction Anomalous entry High insertion	Ureteropelvic conditions Pelvic and caliceal Stone Tumor Probs Stricture

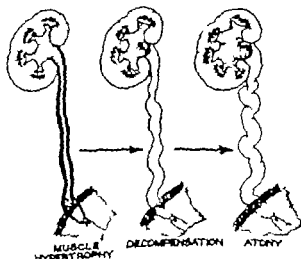


Figure 10 The consequences of distal obstruction are: first, compensatory hypertrophy of the smooth muscle to overcome the obstruction, second, muscular decomposition and inability to expel the contents completely (residual urine) and, finally irreversible atony.

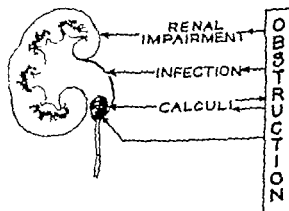


Figure 11 Obstruction not only damages the kidney directly by back pressure, but also fosters infection and stones, which in turn injure the kidney.

can be reached by palpation. Is the infection "simple" or caused by mixed organisms? Mixed bacteria indicate a complicated infection. Are the external genitalia normal and free of vulvitis or vaginitis? Is the total renal function normal? Decreased PSP excretion means residual urine, bilateral renal damage, or both, and requires detailed instrumental study. Are abnormalities detected by intravenous urography especially upper tract anomalies and residual urine?

If any of these diagnostic steps indicates the presence of a disease process or a structural abnormality instrumental steps are in order: catheterization both for culture preparation and to detect residual urine; cystometrography to determine the presence of any abnormality of bladder function; cystography to detect any incompetency of the ureterovesical valves or abnormalities of the vesical neck. Instillation of 5 cc. of light Lipiodol into the bladder will demonstrate residual urine when the child cannot void on command. Since Lipiodol floats it will still be seen in an x ray film obtained two days later

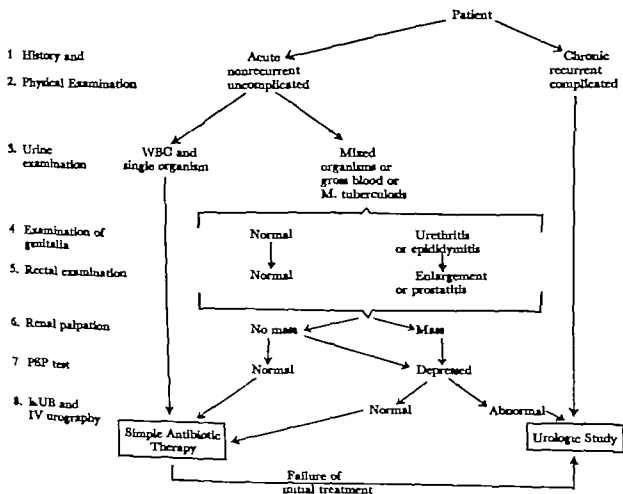
if the child has not completely emptied his bladder during that time.

If upper urinary tract abnormalities are seen in the intravenous urograms, small catheters can be passed to the kidneys and specimens obtained for differential culture preparations and PSP tests and pyelograms which show greater detail than that present in intravenous urograms, can be made by injection of a concentrated radiopaque solution into the renal pelvis.

These instrumental steps lay bare the anatomic and physiologic details of the entire urinary tract and serve as the basis for specific therapy.

Treatment may be correction of the vesical neck obstruction by repeated, increased dilatation of the urethra or by a plastic operation on the vesical neck, correction of the incompetency of the ureterovesical valves, which become incompetent from long-standing obstruction at the bladder neck, or correction of the obstruction at the junction of the renal pelvis with the ureter a common site for congenital abnormality

Table 2.



poor kidney is useless if the normal kidney has taken over the whole excretory load. The damaged kidney will not be stimulated to regain its function.

The second factor of importance is the location of the site of the obstruction. De termination of exactly what pathologic process has resulted in the obstruction is of the greatest importance. Examples would be benign prostatic hypertrophy in the urethra, neurogenic bladder tumor or stone in the ureter and at the ureteropelvic junction, aberrant vessel or congenital stricture. If the hydronephrotic kidney is to be preserved, the attack must be leveled at the site of the obstructive lesion.

TREATMENT

The treatment of hydronephrosis resulting from supravescical obstruction is more complicated than that arising from prostatic enlargement since the delicate technical procedures on the ureter ureteropelvic junction and renal pelvis are fraught with hazards of judgment and of technique. The principal factors affecting surgical treatment of obstruction of the upper urinary tract with hydronephrosis are as follows:

1. If the patient is young, all portions of the renal tissue should be conserved since injuries or disease later in life may damage the remaining kidney. Older people on the other hand, do not need this insurance and would be more greatly harmed by a later nephrectomy should the conservative operation fail.

2. If the contralateral kidney is somewhat damaged or is involved in a process which could be progressive then a greater effort must be made to save the ipsilateral kidney. On the other hand, if the other kidney is quite normal and the hydronephrotic kidney is greatly damaged, one can expect little recovery in the hydronephrotic kidney after the repair since the factors of renal counterbalance will come into play. Then nephrectomy is often the wiser choice.

3. If there is severe infection in the hydronephrotic kidney which has involved the parenchymal tissue there is less chance of this kidney returning to relatively normal function. This would be a factor in advocating nephrectomy rather than repair.

However in actual practice the treatment decided upon for unilateral or bilateral hydronephrosis will in the last analysis be reached in assessing the many factors involved and weighing them against experi-

ence with the kind of surgical repair required.

Obstruction is the cause of irreversible renal insufficiency and eventual death. Obstruction is the result of some other usually correctable, disease of the urinary tract. It fosters secondary disorders, such as infection and the formation of stones. Finally presence of obstruction can be detected by urologic tests and instrumentation and can be corrected surgically by the application of urologic principles.

INFECTION

When confronted with infection anywhere in the urinary tract, it should be remembered that its parts are connected by a column of fluid, by a continuous transitional cell lining and by lymphatic channels. Bacteria in one area can therefore spread to adjacent areas through the urine, along the surfaces of the urethra and prostate or through lymphatic drainage from the bladder to the kidneys.

Stasis of urine from any obstruction to its outflow greatly enhances the spread of bacteria in the urine and perpetuates the resulting infection. Foreign bodies also keep infection active.

ACUTE PYELONEPHRITIS

Sudden infection of one or both kidneys may occur in either sex and in any age group but it is rare in men without some specific underlying disease such as prostatitis, prostatic obstruction with residual urine or calculi.

In children especially little girls, acute pyelonephritis is rather frequently seen. Without prodromata, or following a mild infection of the upper respiratory tract, a high fever and frequency or burning of urination will appear. Sometimes no symptoms are apparent and the diagnosis is made by examination of the urine. The short female urethra probably allows bacteria to ascend easily from the vulva to the bladder. However more than accessibility is required for contaminating bacteria to multiply in sufficient numbers to constitute infection. The necessary additional factor is some abnormality of structure or function of the bladder and bladder neck or less often, of the upper urinary tract.

Steps must be taken to detect these abnormalities in any child who has a second bout of pyelonephritis (Table 2). Is the infection in one kidney or both? A decision

ally this can be accomplished without the necessity of interrupting the pregnancy. The therapeutic intent is to maintain the patient with a minimum of symptoms making no heroic attempt to clear the infection entirely. Thus, the patient may go through pregnancy to begin intensive treatment more safely and effectively in the puerperium. If the infection is associated with stone or actual ureteral obstruction an attempt is made to assist the patient through her pregnancy before a surgical procedure is proposed.

TUBERCULOSIS OF THE URINARY TRACT

From a pulmonary lesion, the tubercle bacilli are disseminated by the blood stream and lodge in the renal cortex. Most of them are eliminated by the tissue defense mechanism, but occasionally a clump will establish itself. A tubercle forms which grows and throws off red and white blood cells and bacilli. Although the bacterial spread is initially bilateral, often it actually causes disease in only one kidney.

The first sign the patient notices may be gross total hematuria. As the disease progresses, the ureters and bladder become infected, giving symptoms of cystitis and often extreme burning and urgency. More bleeding may occur. Eventually scarring of the vesical wall produces a bladder of very small capacity.

Carried from the kidney by the urine, the tubercle bacilli lodge in the posterior urethra and spread to the seminal vesicles, prostate and, by the ejaculatory ducts to the epididymides. Such involvement usually provokes few symptoms.

The diagnosis of genitourinary tuberculosis is made on the basis of a past history of exposure to and of pulmonary involvement by tuberculosis. Upon examination of the external genitalia, a thick, hard epididymis with beading of the vas deferens may be noted. A methylene blue smear made from a urine specimen contains pus cells without organisms. Stains for acid-fast bacteria will be positive in over half the cases. Pyuria without bacteriuria means tuberculosis until proved otherwise. Large overnight specimens are obtained for concentration in the laboratory for smear culture and guinea pig inoculation. Rectal examination may allow a nodular prostate or thick firm seminal vesicles to be discerned. Total renal function is usually normal unless the disease is bilateral and advanced. A plain film of the abdomen often will allow visualization of renal parenchymal calcification, typical of

the slow necrotic reaction of the tissues to the bacilli.

Instrumental study aims at localizing the disease. Retrograde ureteral catheterization is performed to determine whether the infection is in one or both kidneys and whether renal damage is greater on one side.

Treatment is primarily medical. Triple-drug therapy is prescribed for the patient: streptomycin, 1 gm. twice weekly; para-aminohippuric acid, 5 gm. three times a day; and isoniazid, 100 mg. three times a day. If one kidney is greatly destroyed and the other normal, nephrectomy may be done after one to three months of medical treatment. The trend, however, is toward dependence on long-term drug therapy alone controlled by making new cultures semi-annually. The treatment is tentatively stopped after two negative cultures are obtained, but new cultures are made to be sure reactivation has not occurred. Bed rest is also important for the patient.

Infection of the urinary tract almost always means impaired drainage. If the infection is not cleared readily the reason must be found by detailed urologic study. The real purpose is to find the obstruction or foreign body which is keeping the infection active and then to remove the cause.

ANOMALIES OF THE KIDNEY URETER AND BLADDER

ANOMALIES OF THE KIDNEY AND URETER

The ureter is formed embryologically as a bud from the wolffian duct and grows up retroperitoneally to touch the metanephrogenic tissue, where it branches to form the calices and collecting tubules of the kidney, joining the distal convoluted tubules. It is easy to see that developmental arrest occurring while these complicated connections are being formed would result in anomalies. For example, agenesis results if no bud forms (Fig. 13A). If two buds form on one side, or one bud branches, a double kidney results, composed of two ureteral and pelvocaliceal systems (Fig. 13B).

Fusion of the embryonic tissue, if at the lower pole produces a horseshoe kidney (Fig. 13C). Drainage in the case of this anomaly is often poor and obstruction and infection result.

Improper connection of the collecting tubules to the distal convoluted tubules results in congenital polycystic kidneys in which the greater portion of the parenchyma is replaced by cysts. Polycystic disease is

Other urinary abnormalities must be looked for since the urinary tract is the most common site of congenital anomalies.

Only when the anatomic and resulting functional disturbances which permitted the infection to become established have been corrected should an attempt be made to eradicate the infection. Otherwise recurrence and progression are inevitable and good antibiotics will be wasted because of the emergence of a resistant flora.

In adult women acute pyelonephritis similarly arises from urethral sources but is less often associated with bladder and renal abnormalities. One attack may be treated with a sulfonamide only. However, if the infection is not cleared or if it recurs, investigation is needed. The schema shows when complete investigation, including retrograde urologic study is required (Table 2).

Most often episodes of acute infection of the kidney are interspersed with recurrent bouts of cystitis. Cystoscopy will reveal reduction of the caliber of the urethra. Appropriate treatment consists of urethral dilations, then antibiotic therapy with vaginal estrogen suppositories if indicated.

In adult men, acute pyelonephritis arises most commonly from infection of the prostate associated with some prostatic obstruction. The residual urine gives the bacteria a chance to multiply and ascend to the kidneys. A three-glass test will show shreds arising from infected prostatic ducts in the first glass and subsequent massage will produce pus. Intravenous urograms are needed to rule out the upper tract as the source of the infection.

Antibacterial therapy will usually clear the initial attack promptly but treatment directed at the prostate may be necessary for permanent control.

CHRONIC PYELONEPHRITIS

This is an entity to be contrasted to chronic renal infection secondary to obstruction or stone. It occurs most commonly in middle aged women and ultimately destroys the kidneys. Hypertension may occur from renal ischemia. If the disease is unilateral hypertension may be reversed by nephrectomy. The pathologic changes may occur either unilaterally or bilaterally. Characteristically the process is distributed in patches throughout the kidney. Many different pathologic changes may be going on at the same time. For instance, focal abscesses, patchy interstitial inflammation with or without scarring and fibrosis, tubular atrophy as

well as tubular dilation and some destruction of glomeruli may all be found in the same kidney. In late stages there is a great amount of diffuse scarring which constricts the infundibula in particular and an x-ray picture characteristically shows a small kidney with long, narrow spider like infundibular channels joining the calices to the renal pelvis. In addition, changes occur secondary to the parenchymal inflammation in the epithelium of the pelvis and ureter. Such changes are pyelitis cystica, in which small cysts dot the surface, pyelitis granulosa and emphysematosa. There may be metaplasia or leukoplakia and often there is dilation and scarring of the structures. Of particular importance is the fact that the scarring and other changes occurring with pyelonephritis provide impregnable positions for the lodging of bacteria so that the process is self-perpetuating.

The diagnosis is made after examination of the urine of a patient who may or may not have had acute attacks of pyelonephritis. Total renal function will be diminished. Intravenous urograms will show reduction in renal mass, with spidery calices but no obstructive dilatation. A lower tract source must be eliminated and intensive prolonged antibiotic therapy administered. Recurrence is the rule, since only suppression is achieved. The same organism in the same concentration is found in cultures when the drug is stopped.

RENAL INFECTION DURING PREGNANCY

Renal infection during pregnancy occurs in approximately one out of every twelve pregnant women. Etiologically since the colon bacillus or one of the colon bacillus group is found in over 80 per cent of the cases, it is probable that the intestinal tract is the common source of the infection. The secondary factors which allow the infection to lodge and progress are two: (1) Stasis produced by the obstruction of the lower ureter—most often the right ureter—by the fetus. However infection often occurs early in the course of pregnancy before the uterus has grown large enough to be the cause of such obstruction. (2) Probably of more importance is a hormonal factor which causes lowered ureteral tone and consequent dilation. Dilation of the ureter occurs in from 80 to 90 per cent of all pregnant women, although, of course, infection does not invariably accompany it. The diagnosis and treatment in these patients are similar to those in ordinary acute pyelonephritis. Us-

ally this can be accomplished without the necessity of interrupting the pregnancy. The therapeutic intent is to maintain the patient with a minimum of symptoms, making no heroic attempt to clear the infection entirely. Thus, the patient may go through pregnancy to begin intensive treatment more safely and effectively in the puerperium. If the infection is associated with stone or actual ureteral obstruction an attempt is made to assist the patient through her pregnancy before a surgical procedure is proposed.

TUBERCULOSIS OF THE URINARY TRACT

From a pulmonary lesion, the tubercle bacilli are disseminated by the blood stream and lodge in the renal cortex. Most of them are eliminated by the tissue defense mechanism, but occasionally a clump will establish itself. A tubercle forms which grows and throws off red and white blood cells and bacilli. Although the bacterial spread is initially bilateral, often it actually causes disease in only one kidney.

The first sign the patient notices may be gross total hematuria. As the disease progresses, the ureters and bladder become infected, giving symptoms of cystitis and often extreme burning and urgency. More bleeding may occur. Eventually scarring of the vesical wall produces a bladder of very small capacity.

Carned from the kidney by the urine the tubercle bacilli lodge in the posterior urethra and spread to the seminal vesicles, prostate and, by the ejaculatory ducts, to the epididymides. Such involvement usually provokes few symptoms.

The diagnosis of genitourinary tuberculosis is made on the basis of a past history of exposure to and of pulmonary involvement by tuberculosis. Upon examination of the external genitalia, a thick, hard epididymis with beading of the vas deferens may be noted. A methylene blue smear made from a urine specimen contains pus cells without organisms. Stains for acid-fast bacteria will be positive in over half the cases. Pyuria without bacilluria means tuberculosis until proved otherwise. Large overnight specimens are obtained for concentration in the laboratory for smear culture and guinea pig inoculation. Rectal examination may allow a nodular prostate or thick firm seminal vesicles to be discerned. Total renal function is usually normal unless the disease is bilateral and advanced. A plain film of the abdomen often will allow visualization of renal parenchymal calcification, typical of

the slow necrotic reaction of the tissues to the bacilli.

Instrumental study aims at localizing the disease. Retrograde ureteral catheterization is performed to determine whether the infection is in one or both kidneys and whether renal damage is greater on one side.

Treatment is primarily medical. Triple-drug therapy is prescribed for the patient: streptomycin, 1 gm. twice weekly; paraaminohippuric acid, 5 gm. three times a day; and isoniazid, 100 mg. three times a day. If one kidney is greatly destroyed and the other normal nephrectomy may be done after one to three months of medical treatment. The trend, however, is toward dependence on long-term drug therapy alone, controlled by making new cultures semiannually. The treatment is tentatively stopped after two negative cultures are obtained, but new cultures are made to be sure reactivation has not occurred. Bed rest is also important for the patient.

Infection of the urinary tract almost always means impaired drainage. If the infection is not cleared readily the reason must be found by detailed urologic study. The real purpose is to find the obstruction or foreign body which is keeping the infection active and then to remove the cause.

ANOMALIES OF THE KIDNEY URETER AND BLADDER

ANOMALIES OF THE KIDNEY AND URETER

The ureter is formed embryologically as a bud from the wolffian duct and grows up retroperitoneally to touch the metanephrogenic tissue where it branches to form the calices and collecting tubules of the kidney, joining the distal convoluted tubules. It is easy to see that developmental arrest occurring while these complicated connections are being formed would result in anomalies. For example, agenesis results if no bud forms (Fig. 13A). If two buds form on one side or one bud branches, a double kidney results, composed of two ureteral and pelvocaliceal systems (Fig. 13B).

Fusion of the embryonic tissue, if at the lower pole, produces a horseshoe kidney (Fig. 13C). Drainage in the case of this anomaly is often poor and obstruction and infection result.

Improper connection of the collecting tubules to the distal convoluted tubules results in congenital polycystic kidneys in which the greater portion of the parenchyma is replaced by cysts. Polycystic disease is

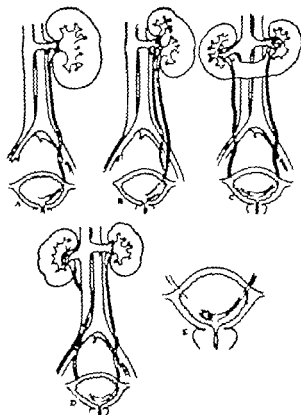


Figure 13 A, Renal agenesis. B, Renal and ureteral duplication. C, Horseshoe kidney. D, Retrocaval ureter. E, Ureterocele.

hereditary. It is seen either in infants in whom bilateral masses are felt at, or soon after birth and in whom the disease is usually fatal well before adolescence, or in adults over the age of thirty. In adults pain, hematuria or infection may lead to the diagnosis or the first external evidence may be the symptoms of renal insufficiency often with hypertension. Pyelography allows the typical elongated and curved calices to be seen. Treatment is usually medical.

With developmental anomaly of the vena cava, the right ureter may remain behind and medial to it, resulting in a retrocaval ureter (Fig. 13D). It is then more liable to obstruction. Treatment is division and reanastomosis of either the ureter or the vena cava.

If the ureteral bud forms improperly at its junction with the bladder stenosis remains, which forces dilatation and prolapse of the ureteral wall into the bladder forming a ureterocele (Fig. 13E). The result is upper tract obstruction. A filling defect in the bladder can be seen on the intravenous urogram; cystoscopically the cystlike structure can be identified. Cure is effected by incising the stenotic orifice and wall of the ureterocele.

ANOMALIES OF THE BLADDER

Exstrophy of the bladder is the most common major anomaly. Development of the anterior wall of the bladder and urethra, as well as of the overlying abdominal wall, is incomplete so that these organs lie open on the abdomen. If only the urethra fails to close, epispadias results.

Treatment ideally should provide a functional urinary reservoir. Restoration of the bladder to the abdomen has not been very successful because its wall is abnormal and because it possesses no effective urethral sphincter. Diversion of the urine to the rectum retains a reservoir but encourages infection, while diversion to the skin via an ileal conduit reduces the chance of renal infection but does not provide urinary continence.

Should obliteration of the allantois fail, a fistulous connection persists between the dome of the bladder and the umbilicus. During voiding, a fine stream of urine issues from the umbilicus. If both ends of the allantois become closed, a urachal cyst results forming a midline subfascial mass which may periodically burst into the bladder or drain out through the umbilicus.

Anomalous development is a common source of obstruction in the urinary tract, whether the abnormality is in the kidney, ureter, bladder or urethra. Early detection is important so that the defect can be corrected before the appearance of irreversible changes.

INJURIES TO THE KIDNEY, URETER AND BLADDER

RENAL INJURIES

Direct blows to the kidney can cause (Fig. 14) contusion without distortion of the renal outline or of the collecting system (A), external rupture through the capsule with severe retroperitoneal bleeding and obliteration of the renal outline and psoas shadow (B), internal rupture, tearing into a calyx, with bleeding and pyelographic distortion (C), and combined rupture through the pelvis and capsule (D).

Symptoms range from slight flank tenderness with hematuria, detectable only microscopically to severe pain in the flank, a mass in the retroperitoneal area and gross bleeding. If the bleeding into the retroperitoneal space is profuse, shock supervenes.

Diagnosis of renal injury is aided by intravenous pyelography. Severe damage is present if the kidney fails to excrete contrast material usually.

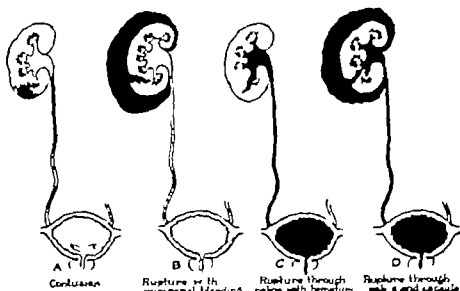


Figure 14 Renal injuries.

in whole or in part. Retrograde pyelography may be needed to define the damaged area so that primary surgical repair can be performed.

Treatment is usually nonsurgical unless the bleeding continues or more rarely infection occurs. If the mass in the flank grows progressively larger if the shock is not readily corrected by blood transfusions or if the kidney is shown by pyelography to be greatly deranged, operation is necessary either to repair the lacerations and control the bleeding or more often, to remove the kidney.

URETERAL INJURIES

The ureter because of its size and its movable position in the retroperitoneal fat, is rarely injured by external violence but is occasionally damaged during gynecologic operations in the pelvic area or less often, during instrumental attempts to remove stones.

VESICAL INJURIES

The bladder must be distended to be injured by a direct blow. It may be perforated by neighboring bony structures.

The most constant symptom or sign is hematuria. In severe injuries the patient has marked pain over the area and abdominal rigidity if the rupture extends intraperitoneally. Often he is unable to pass urine at all. Shock is frequently present.

The diagnosis is best made by cystogram, so that the site and extent of the extravasation can be actually seen on the x-ray film. The recovery of a measured amount of water instilled through a catheter is less reliable assurance that rupture has not occurred. In

travenous urography is a safe method to show the presence of extravasation.

The treatment is drainage of the bladder suprapubically with closure of the laceration if it is intraperitoneal or if it is large.

Injuries of the urinary tract are dangerous because of the frequently severe hemorrhage and especially because of the extravasation of urine with its malign consequences. Restoration of continuity of the urinary passages and adequate drainage of areas of leakage are the prime purposes of treatment.

CALCULOUS DISEASE

THE CAUSES OF STONE FORMATION

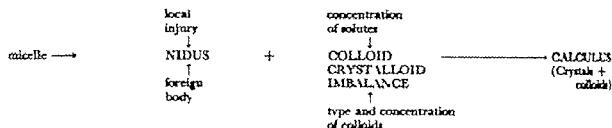
Two factors must be present for a stone to develop (1) a nidus to serve as a center for the precipitation of calcareous materials and (2) an imbalance between the amount and type of crystalloid substance held in the urine and the concentration and type of colloid substances which hold the crystalloids in solution (Table 3).

The Nidus. Because of its surface activity a nidus provides a nucleus for the precipitation of the components of stone. By spectrophotometry the chemist can not only analyze the bulk of the stone, but he can also determine the composition of the original core. Then the physician knows the original metabolic factor which initiated the formation of the stone.

There are four sources for a nidus

1 *Subepithelial injury* By careful study of hundreds of sections of kidneys obtained at postmortem examination, Randall found calcified plaques resulting from local tissue

Table 3 Factors in Renal Calculi Formation



injury in the renal papillae of almost one fourth of them. In 8 per cent he found actual stones adherent to the papillae. Similar changes have been observed beneath the epithelium of the renal collecting tubules. Somewhat analogous have been the findings of Carr that calcifications occur in the lymphatics draining the calices, which may grow and break through into direct contact with the caliceal urine.

2. *Epithelial changes to which colloids adhere* Stones can be made to form in rats maintained on a diet deficient in vitamin A. Such a deficiency would be rare in the human and these experiments merely illustrate that epithelial changes themselves are enough to initiate stone formation. Infection and surgical injury, are much more common as clinical causes for such alteration of the epithelium of the renal pelvis.

3. *Formation of micelles* A micelle is an aggregate of crystalloids which reaches colloidal size. It then can act as a colloid to bind further crystalloids. A micelle can be thought of as an embryonic stone.

4. *Foreign body* This is the obvious nidus, a residual stone nonabsorbable suture, retention catheter and the like.

Colloid-Crystalloid Imbalance. More than a nidus is necessary for stone formation. The salts must be precipitated in an organic matrix for a calculus to grow. Ordinarily the urine is supersaturated with crystalloids which are held in solution by colloidal substances. This balance may be upset by the urine providing too many crystalloids, by its having too few protective hydrophilic colloids or by its having a preponderance of nonprotective hydrophobic colloids.

Concentration of Solutes. Obviously water is a major factor since, generally the more dilute the solutes, the less likely are aggregation and concretion to take place. Overhydration is good basic treatment for the patient who recurrently forms stones.

Increased calcium excretion in the urine can be detected by adding Sulkowitch reagent, which precipitates the calcium as the

insoluble oxalate. This test should be made on the concentrated urine specimen of all patients with stones, since 5 per cent of them have hyperparathyroidism. Hyperactivity of the parathyroid glands causes an increase in serum calcium and a decrease in serum phosphorus together with a rise in the urinary excretion of both substances. Hyperparathyroidism should be suspected in any patient with opaque stones especially if he also has excessive calcium in the urine. Proof of the diagnosis rests, however, on finding an elevated serum calcium level by a reliable laboratory. The treatment is removal of the parathyroid adenoma.

Since the bones form so large a portion of the body any factors which cause skeletal breakdown will produce hypercalciuria. An outstanding factor is prolonged immobilization. Resorption of bone begins upon removal of weight bearing. The result is an outpouring of calcium and phosphorus for eight or nine weeks until equilibrium of bone stress is restored. Not only is there increased calcium available for stone formation, but renal drainage is impaired and small nidi are retained. The administration of calcium to such a patient in an attempt to reverse the osteomalacia would only increase the level of calcium in the urine.

Renal tubular acidosis, while not common, is an interesting example of hypercalciuria. It occurs because the tubules cannot form ammonia and so calcium and potassium are used to neutralize the acid. The excessive calcium excretion causes nephrocalcinosis or renal calculi, as well as producing osteomalacia. These derangements can be corrected by the oral administration of enough sodium bicarbonate to provide sodium ions to substitute for the ammonium ion.

Other substances besides calcium can be excreted in excessive amounts. In individuals suffering from a familial disorder of excessive amino acid excretion, cystine competes unsuccessfully with lysine in the defective tubular resorption mechanism and is excreted in excessive amounts forming stones.

Since cystine is soluble in alkaline urine recurrence can be reduced by alkalization.

Uric acid excreted in excess by patients with gout, may pass as crystals to produce colic, may form the nidus for calcium stones or may gather into aggregates as a true stone. Larger amounts of uric acid are also excreted in patients with leukemia, especially after cortisone therapy.

Colloid effects. The urine must be thought of as an extension of the extracellular ground substance. The colloids in the urine are mucopolysaccharides. An example is hyaluronic acid, normally disposed in the extracellular cement. These colloids are either protective or stone forming. The protectiveness of the colloid depends on its quantity and its molecular weight. By absorption on the hydrophobic stone-forming colloid, the hydrophilic colloid exerts its protective action. The protective colloid apparently, before agglomeration, acts to prevent crystal orientation and growth.

The hydrophobic, stone-forming colloids, on the other hand, compose the organic matrix of the stone. They are present in "stone formers" in three to eleven times the normal quantity. Because of different electrophoretic characteristics from the hydrophilic colloids, they absorb on a nucleus and initiate a stone. Several micelles plus these colloids yield a nucleus.

Stasis. Obstruction to the urinary outflow has two effects. One is retention of the nidus (Fig. 15) or embryonic stone, and the other is the promotion of infection, which in turn enhances the deposition of calcific substances.

Infection. Urea-splitting organisms decrease the solubility of calcium phosphate by altering the pH of the normally acid urine. For example, if normal urine at pH 5.6 is just saturated with calcium phosphate, it will be saturated two times at pH 6 and eight times at pH 7.

In addition, infection alters the composition of the colloids in the urine and the altered colloids from the damaged lining help make up the matrix of the stone.

Etiologic Study. First, hyperparathyroidism must be ruled out by means of a Sulzowitch test and by serum calcium and phosphorus determinations in a reliable laboratory.

Second, the urine must be tested for cystine with the nitroprusside test, uric acid crystals in the urine must be looked for.

Third, the calculus must be recovered and analyzed.

Prophylaxis. The prevention of recurrence, rather than the prevention of the initial stone, is the problem. Certain general measures should be prescribed for all patients: hydration, mobilization and elimination of infection.

Forcing fluids up to amounts of 4000 cc. per day is probably the single most valuable preventive measure. Micelle formation is inhibited and precipitation is arrested. In addition, any existing nidus or small stones tend to be washed out by the increased urinary flow. For example, Suby has evidence that recurrence in a solitary kidney which has twice the normal output of urine, is less frequent than when both kidneys are present. Priens' suggestion is to start the day with twenty pennies in one pocket and to transfer one for each glass of water taken, until at the end of the day all are in the other pocket.

Mobilization can be a critical factor. Bed rest promotes resorption of calcium from the skeleton so that excess urinary excretion results. It also enhances stasis, so that the urinary tract is more susceptible to infection and any embryonic stone stays behind to grow larger.

Elimination of the infection which is usually present at the time the stone is removed is most important but is often not easily done. Urea-splitting organisms, which make the urine alkaline and so increase the deposition of calcium phosphate and carbonate, are introduced by retention catheters. Low dosage chemotherapy, irrigation with Suby's solution G, a buffered citric acid solution in which calcium is soluble, and frequent change of catheters all help.

In addition, plain roentgenograms or intravenous urograms should be made regularly and the urine should be examined periodically for pus and organisms.

More specific measures may be needed in certain cases, especially if recurrence is rapid. Recurrence of calcium phosphate stones is slowed by a low calcium intake.

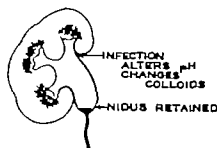


Figure 15 The sequelae of stasis.

but a low phosphorus-Basaljel or sodium phytate regimen may be required. Phytates benzene ring with phosphate radicals forms a complex with calcium and so reduces its availability. Acidification by an acid-ash diet is disappointing for although calcium is more soluble in an acid urine, it is likewise absorbed more readily from the acidified gastrointestinal tract. Salicylate therapy, thought to promote protective glucuronide excretion, has not proved effective. Calcium oxalate stones are inhibited by measures similar to those outlined above: reduction in oxalate intake does not seem to reduce the formation of stones.

Uric acid stones are inhibited in an alkaline urine maintained by the ingestion of sodium or potassium citrate. A similar program is applicable for cystine stones although it is somewhat less effective.

Clinical Findings. The symptoms of a urinary tract stone are limited to the pain which the stone causes to the secondary changes brought about by actual irritation by the stone, to the symptoms associated with the accompanying infection and to the train of symptoms of renal failure due to occlusion by the stone.

Pain is caused by a stone when it moves and irritates the surfaces or when it actually blocks the outflow of urine causing colic if in the ureter or distention of the renal capsule and pelvis if at the ureteropelvic junction. Since many stones grow without causing much local irritation and do not move from the site where they were originally formed, a high percentage of calculi give no symptoms of their presence although occasionally a large calculus in the renal pelvis or in the bladder may cause direct pain when the patient moves suddenly or is jarred.

Hematuria is common, resulting from contact of the stone with the lining of the pelvis or ureter. Microscopic blood will be found in almost all patients with urinary tract stone.

Infection causes the usual symptoms of acute pyelonephritis. Very often, after subsidence of these acute attacks, low grade pyelonephritis is fostered by the stones. If the stone almost completely obstructs the kidney, this low grade infection can be extremely debilitating. Since the symptoms do not point directly to the kidney the diagnosis is often delayed.

Dysuria is caused by reflex reaction of the bladder to ureteral hyperactivity from

the ureteral calculus, by actual irritation of the stone in the bladder itself or by the secondary infection. Occasionally the stone may act as a ball valve, intermittently occluding the urethral outlet. This may be so marked that the patient must assume acrobatic positions to empty his bladder.

Renal failure is occasionally associated with a urinary tract calculus. The classic example is a calculus obstructing the ureter of a solitary kidney. Overdosage of sulfonamides without adequate alkalization of the urine has resulted in chemical calculi obstructing the tubules or obstructing the renal pelvis and ureters. It is also possible for acute obstruction of one kidney to act reflexly through the sympathetic nerves and cause renal shunt and anuria on the contralateral side. While this has not been demonstrated clinically there are good experimental reasons for its occurrence.

Diagnosis. The history may or may not be characteristic. Physical examination may reveal enlargement or tenderness of one kidney. Rarely by combined rectal and abdominal palpation, a calculus may be felt in the bladder. Examination of the urine will reveal hematuria in a high percentage of cases. It is best to see the patient during an attack in order to reveal this clinical finding. Of course, if the urinary tract is infected as well, there will be pus and bacteria in the urine. Decrease in total renal function, while not diagnostic, is of great value in assessing the damage done by the stone. A plain film is important in the diagnosis of urinary tract calculi since by knowing the usual position of the ureter and by visualizing the outline of the kidney one may make a fairly reasonable guess as to whether the calcification lies within or outside the urinary tract. In addition, familiarity with the x ray shadow cast by stones will help differentiate them from phleboliths, calcified mesenteric nodes and foreign bodies within the gastrointestinal tract. Since about 8 per cent of renal calculi cast no shadow the roentgenogram cannot be used to rule them out completely.

Intravenous pyelography is of great value in confirming the location of densities seen on the plain film and in assessing the degree of obstruction. Whereas urethral calculi are best identified and localized by palpation and the use of a metal sound which grates on the stone, bladder stones are suspected by examination of the plain film and are diagnosed absolutely by cystoscopic inspection.

tion. Calculi in the ureter are localized partly by intravenous urography and partly by retrograde catheterization. An occasional valuable help in nonopaque calculi is the use of a catheter prepared with a soft waxy tip so that it will receive a scratch upon passing the stone. Renal calculi will be found to lie within the shadow of the calices by intravenous and retrograde pyelography and, in addition, information gained regarding relative function by the retrograde study will help decide whether removal of the stone or removal of the kidney is the proper course.

Routine examination of the urine done before operation for appendicitis may reveal red blood cells, which suggests that a search for ureteral calculi should be made. Occasionally however an appendix lying near the course of the ureter will so irritate the ureter that hematuria, perhaps even with pyuria and bacilluria, may occur. If a plain film is obtained and intravenous urography is performed, a differentiation of the ureteral calculus and the abnormally located appendix can be made. The symptoms of a ureteral calculus may also be mimicked by a blood clot passing down the ureter from tumors or tuberculosis of the kidney. A tumor of the renal pelvis or ureter may intermittently obstruct the outlet so that renal colic occurs. A plain film and retrograde study will help to rule out these diseases. Intercostal neuritis, especially of branches of the twelfth thoracic and first lumbar nerves may occasionally mimic a ureteral calculus until the typical skin lesion occurs.

TREATMENT

Urinary tract calculi may be managed expectantly by manipulative procedures or by open operation. Attempts to dissolve calculi have met with little success so far.

Urethral calculi may occasionally be manipulated from the urethra by fairly simple means, especially if they are not too large and are situated distally in the urethra.

Vesical calculi are usually retained ureteral stones. A stone able to pass through the ureter should be able to pass out through the urethra unless some obstruction is present at the vesical neck. Treatment of a bladder stone must then be accompanied by treatment of the obstructing lesion. If prostatic hypertrophy is the factor involved, then the stone may be removed at the time of prostatectomy.

The first crushing instrument was de-

veloped over a hundred years ago. The lithotrite is passed through the urethra to grasp the stone blindly and elevate it from the floor of the bladder and crush it. This process can be repeated until the fragments are small enough to be evacuated through a large bore sheath. Numerous irrigating and visual modifications of the lithotrite have been developed but they all have the fundamental disadvantage that addition of lenses or irrigating tube weakens their structure so that they are subject to bending and breaking. Such unfortunate occurrences can leave a foreign body in the bladder that is worse than the original stone. Stones larger than 2 inches in diameter or stones formed upon a foreign body too large to be withdrawn through the urethra, and some large stones of extreme density, should not be subjected to litholapaxy but are best removed suprapubically by open operation.

Ureteral calculi can be treated in several ways. One may (1) wait for the stone to pass of its own accord urging it along by suitable measures, (2) introduce instruments through the urethra and ureter to manipulate and withdraw the calculus or (3) perform open operation. The factors which influence judgment are (1) Size of the stone. A stone greater than 1 cm. in diameter especially if situated in the upper ureter probably should be attacked surgically. One should avoid operation for small stones, especially small renal stones, because they may move and be difficult to locate. They can be expected to pass spontaneously. (2) The position of the stone in the ureter. A stone arrested in the upper ureter has farther to go and less chance of passage. Therefore, operation may be warranted for a small stone high in the ureter a stone which would be expected to pass if it lay in the lower ureter. In the upper ureter manipulation is more difficult and hazardous whereas operation is easier. (3) The duration of impaction. If a stone shows signs of progression down the urinary tract, its passage can be awaited. If the stone has become impacted in one area and has not moved over a long period, it will need manipulation or operation for removal. The long period of nonmovement is itself an indication that the stone is too large for the ureter at that point. In addition, after a time in one location, the stone causes irritation and ureteral thickening which obstruct its passage. (4) The presence of infection. If infection is present above the stone, then

waiting for spontaneous passage or manipulative treatment should be persisted in for only a brief period. Early open operation is desirable for these patients, since pyelonephritis or multiple cortical abscesses can not only severely damage the kidney but may prove fatal. (5) The function in the opposite kidney. If the contralateral kidney has less than normal function, one is not justified in prolonging expectant and manipulative procedures. Delay may be enough to push the patient into renal failure. (6) The general condition of the patient. Judgment must be used in choosing the least traumatic and most certain method of removal of the stone in patients ill from sepsis or debilitation.

Expectant treatment is begun by forcing fluids to increase the rate and volume of ureteral peristalsis. This in turn increases the possibility that the stone will be passed. Ambulation of the patient also accelerates movement of the stone. Morphine is administered in adequate dosage to control the pain of ureteral colic. Urinary antibacterial agents may be given to decrease the likelihood of infection. The patient must be observed closely for pain or tenderness in the flank and should be studied by intravenous urography for evidence of ureteral blockage which would produce hydronephrosis and renal damage.

Manipulative treatment is reserved for stones less than 1 cm. in diameter in the lower ureter which have not been impacted too long. The loop catheter which contains a fine steel wire so that traction on the wire produces a loop at its tip is passed to the renal pelvis and the loop is formed. The loop is slowly drawn down until the stone is engaged and is pulled down intermittently for from twelve to twenty four hours in order to withdraw the stone gradually. Special wire basket extractors may be used instead of the looped catheter.

If the stone lies just outside the bladder it may be encouraged to drop in if the orifice is incised with a cautery or scissors.

Operative treatment is indicated for upper ureteral stones, which are approached through the flank, and for lower stones which are removed through the lower abdomen. If nephrectomy is necessary because the kidney is severely damaged by hydronephrosis or pyelonephrosis the ureter must be removed down to the site of the stone, since a stone left in a blindly ending ureter will maintain infection, producing intermittent pyuria which can infect the opposite

kidney. Ureterolithotomy for a stone in the terminal portion of the ureter may be difficult because this area lies so deep in the pelvis. After the stone is removed, the incision in the ureter is left open and adequate drainage provided to the area. The ureter will quickly heal if obstruction is not still present.

Renal calculi, if small, in patients who give a history of having passed gravel previously should be treated conservatively since they may be expected to pass spontaneously. Conservative treatment is appropriate if renal damage is not evident, if symptoms are few the stones are small and there is no evidence of renal damage. Surgical treatment also should probably be avoided in those patients who rapidly reform stones after operation and in patients who have cortical calcifications in conjunction with hyperparathyroidism. However renal stones of sufficient size to cause symptoms promote infection or cause renal damage through irritation or obstruction should be removed by open operation. The fact that nephrectomy has been found necessary in more than 30 per cent of patients with renal calculi shows that diagnostic procedures were not applied early enough or astutely enough. The decision as to whether the stone should be removed by pyelotomy or nephrotomy, incising through the renal cortex, or whether the entire kidney should be removed by nephrectomy will rest on a number of findings: (1) the condition of the opposite kidney, (2) the degree of damage in the affected kidney, and (3) the size of the stone. Stag horn calculi requiring extensive nephrotomy are occasionally best treated by nephrectomy whereas a large pelvic stone can often be removed by the less damaging operation of pyelotomy. If stones are present bilaterally one must be more conservative, for the incidence of recurrent calculi is high. For instance, large bilateral stag horn calculi in an older patient should perhaps be left alone since the formation of such large calculi in itself indicates sufficient renal function to provide this copious excretion of salts and since operation is so damaging to the kidney. In a younger patient, however, bilateral nephrotomy with removal of the stag-horn calculi and careful reanastomosing of the calices and renal parenchyma is better since these patients after operation have a greater life expectancy than they would if the stones were left in place. If stones lie in the ureter as well as

in the kidney the lowermost stone should be attacked first since ureteral stones are usually more damaging to kidney function than are renal stones.

RECURRENCE OF CALCULI

Renal stones recur in about 30 per cent of the patients upon whom nephrolithotomy has been performed. This recurrence rate is partly an indication of inadequate removal done without roentgenographic studies at the operating table and partly a measure of the continued action of the intrinsic factors which produced the stone in the first place. Meticulous care in removal, avoidance of encrustation on retention catheters and insistence on prophylactic procedures will be of aid in preventing recurrence.

Prophylaxis Against Occurrence and Recurrence of Urinary Tract Calculi. Measures to prevent recurrence are obvious from a study of the factors in the formation of renal calculi which are listed in Table 3. Obstruction must be treated by dilation or by operative procedures and infection must be eradicated, especially that due to urea splitting organisms. Hyperparathyroidism must be treated by removal of the parathyroid adenoma. Patients with cystinuria or with uric acid stones can prevent recurrence of the stones by maintaining an alkaline urine. Patients who are immobilized must be moved about as much as possible, even by using a tilting bed if necessary. The factors involved in crystalloid-colloid balance are best maintained by a high water intake which so dilutes the crystalloids that precipitation is less likely. If sulfonamide drugs are being administered in high dosages, fluid intake should be adequate and the urine made alkaline. Foods which contain calcium should be avoided in patients with calcium stones. The value of the acid ash diet is in doubt and the high proportion of meat necessary in carrying out this diet increases the excretion of urea, which in turn may furnish an excellent medium for the growth of the urea splitting organisms especially if they are so firmly established that the urine cannot be rendered acid. A low phosphorus-aluminum hydroxide or a phytate regimen may be needed if simple measures fail.

DISSOLUTION OF STONES

Instillation into the bladder of Suby's solution G essentially a buffered citric acid solution, through a retention catheter has dissolved some vesical calculi, but usually

litholapaxy or open operation is better because much less hospitalization is required and the results are more certain. In the treatment of recurrent urinary tract calculi solution G can be used immediately after operation when a nephrostomy tube is in a kidney which still contains calcareous remnants. It is also valuable in postoperative patients in whom an alkalinizing, urea-splitting organism causes rapid encrustation of the nephrostomy tube and of the renal pelvis. The attempt at dissolution of stones by solution G supplied through ureteral catheters has almost uniformly met with failure because, even though solution G dissolves the crystalloids on the surface of the stone, it cannot dissolve the layer of organic material forming the matrix of the stone. Attempts at adding urease and other proteolytic enzymes have not been successful because of the inability of weaker solutions of these substances to dissolve the organic matrix and because of the tendency of the enzymes to attack the renal parenchyma as well as attack the stone.

Urinary calculi form by precipitation upon a nidus of salts unstably present in the urine. Prevention and treatment are thus directed at assisting the urinary tract to rid itself of these foreign bodies by removing obstructions, and at reducing the concentration of dissolved crystalline substances discharged through the kidneys by diet, by high fluid intake, or by reducing the availability of these substances by eliminating infection, altering the pH of the urine or adding compounds with a particular affinity for the salts.

Surgical or instrumental removal of an established stone precedes the prophylactic measures since they will not be effective in the presence of obstruction or infection.

NEOPLASMS OF THE URINARY TRACT

TUMORS OF THE URETHRA

The majority of tumors of the urethra arise after periods of inflammation. In women nonmalignant masses are by far the more common. In men, any urethral mass must be assumed to be malignant until tissue examination proves it to be otherwise.

Diagnosis is made by direct examination of the urethra through a panendoscope in the patient who complains of urethral symptoms such as bleeding, frequency, urgency, dysuria, urethral discharge and decrease in caliber of the stream.

waiting for spontaneous passage or manipulative treatment should be persisted in for only a brief period. Early open operation is desirable for these patients, since pyelonephritis or multiple cortical abscesses can not only severely damage the kidney but may prove fatal. (5) The function in the opposite kidney. If the contralateral kidney has less than normal function, one is not justified in prolonging expectant and manipulative procedures. Delay may be enough to push the patient into renal failure. (6) The general condition of the patient. Judgment must be used in choosing the least traumatic and most certain method of removal of the stone in patients ill from sepsis or debilitation.

Expectant treatment is begun by forcing fluids to increase the rate and volume of ureteral peristalsis. This in turn increases the possibility that the stone will be passed. Ambulation of the patient also accelerates movement of the stone. Morphine is administered in adequate dosage to control the pain of ureteral colic. Urinary antibacterial agents may be given to decrease the likelihood of infection. The patient must be observed closely for pain or tenderness in the flank and should be studied by intravenous urograph for evidence of ureteral blockage which would produce hydronephrosis and renal damage.

Manipulative treatment is reserved for stones less than 1 cm. in diameter in the lower ureter which have not been impacted too long. The loop catheter which contains a fine steel wire so that traction on the wire produces a loop at its tip, is passed to the renal pelvis and the loop is formed. The loop is slowly drawn down until the stone is engaged and is pulled down intermittently for from twelve to twenty-four hours in order to withdraw the stone gradually. Special wire basket extractors may be used instead of the looped catheter.

If the stone lies just outside the bladder it may be encouraged to drop in if the orifice is moistened with a cantary or scissors.

Operative treatment is indicated for upper ureteral stones, which are approached through the flank, and for lower stones, which are removed through the lower abdomen. If nephrectomy is necessary because the kidney is severely damaged by hydronephrosis or pyonephrosis, the ureter must be removed down to the site of the stone, since a stone left in a blindly ending ureter will maintain infection, producing intermittent pyuria which can infect the opposite

kidney. Uterolithotomy for a stone in the terminal portion of the ureter may be difficult because this area lies so deep in the pelvis. After the stone is removed, the incision in the ureter is left open and adequate drainage provided to the area. The ureter will quickly heal if obstruction is not still present.

Renal calculi, if small, in patients who give a history of having passed gravel previously should be treated conservatively since they may be expected to pass spontaneously. Conservative treatment is appropriate if renal damage is not evident and symptoms are few; the stones are small and there is no evidence of renal damage. Surgical treatment also should probably be avoided in those patients who rapidly reform stones after operation and in patients who have cortical calcifications in conjunction with hyperparathyroidism. However, renal stones of sufficient size to cause symptoms, promote infection or cause renal damage through irritation or obstruction should be removed by open operation. The fact that nephrectomy has been found necessary in more than 50 per cent of patients with renal calculi shows that diagnostic procedures were not applied early enough or astutely enough. The decision as to whether the stone should be removed by pyelotomy or nephrotomy, meaning through the renal cortex, or whether the entire kidney should be removed by nephrectomy will rest on a number of findings: (1) the condition of the opposite kidney; (2) the degree of damage in the affected kidney; and (3) the size of the stone. Stag-horn calculi requiring extensive nephrotomy are occasionally best treated by nephrectomy, whereas a large pelvic stone can often be removed by the less damaging operation of pyelotomy. If stones are present bilaterally, one must be more conservative, for the incidence of recurrent calculi is high. For instance, large bilateral stag-horn calculi in an older patient should perhaps be left alone since the formation of such large calculi in itself indicates sufficient renal function to provide this copious excretion of salts and since operation is so damaging to the kidney. In a younger patient, however, bilateral nephrotomy with removal of the stag-horn calculi and careful resuturing of the calices and renal parenchyma is better, since these patients after operation have a greater life expectancy than they would if the stones were left in place. If stones lie in the ureter as well as

the present time, it is reasonable to insist that the patient take large quantities of water to dilute the carcinogens.

Pathology. The most common tumor of the bladder is papillary carcinoma. It is simplest to think of vesical carcinomas as if they arose in successive stages. The first manifestation is a simple papilloma, with a narrow stalk and delicate fronds. On microscopic examination, one sees no evidence of malignant change in the fronds, stalk or in the base where the tumor is attached to the bladder wall. These lesions may or may not progress to a malignant form. Increasing malignancy is signaled by thickening of the stalk and clubbing of the papillary fronds. Ulceration follows, with increasing infiltration by malignant cells at the base, first penetrating through the mucosa and submucosa, later invading the muscularis and finally going through all layers of the bladder wall. It is not possible in the case of a small tumor to say definitely that it is non-malignant even after microscopic examination. It is therefore necessary to consider all papillary tumors of the bladder as potentially malignant and treat them accordingly.

The fact that bladder tumors may be multiple and recurrent makes careful follow-up cystoscopic examinations after treatment imperative.

Squamous cell carcinoma, although less common, is highly malignant. It is infiltrating from the onset and is usually unrecognized until the surface ulcerates. It is extremely hard grossly and microscopically it

consists of squamous cells with or without cornification.

Symptoms. Hematuria is the presenting symptom in more than 77 per cent of these tumors. Often recurrent cystitis or dysuria will lead to cystoscopy and to discovery of the tumor. Ureteral obstruction is not rare, because the areas about the ureteral orifices are most commonly involved by tumor. A routine search for the source of the obstruction of the upper urinary tract and infection may reveal the presence of tumor.

Diagnosis. The diagnosis is made after cystoscopic examination. The bladder is inspected area by area in a routine fashion so that multiple tumors will not be overlooked. A biopsy of the tumor is made at the same time. The ideal excision for biopsy will include a portion of the base and some of the muscle, so that the degree of infiltration can be estimated. Bimanual palpation with the patient anesthetized must be performed in all large tumors to estimate the depth of infiltration and so determine operability.

Treatment. Small, pedunculated tumors are resected transurethrally with the resectoscope which removes a portion of the submucosa and muscle underlying the tumor. The base is then thoroughly fulgurated with the high frequency current. Larger tumors which have been found to have penetrated into the muscle, but not to have extended through it, are treated by deep resection or preferably by partial cystectomy or by total cystectomy with diversion of the urine. If the tumor has already infiltrated the walls

CELL GRADE



DEPTH GROUP		1 BENIGN	2	3	4 ANAPLASTIC
	A	 TUMOR FULGURATION	TUR		
	B ₁	 TUR; PARTIAL CYSTECTOMY		↑ LOCAL RADIATION	
	B ₂		PARTIAL OR TOTAL CYSTECTOMY; DEEP RADIATION		
	C			RADICAL CYSTECTOMY; DEEP RADIATION; INJECTION	

Figure 16 Relation of cell grade to depth of infiltration.

Pathologic Changes. Squamous cell carcinoma of the urethra is most common occurring in more than 55 per cent of the cases.

Since the urethra is lined with transitional cell epithelium, metaplasia probably occurs initially. Urethral tumors are highly malignant and are likely to metastasize directly to the deep nodes, especially the iliac and pelvic nodes without involving the superficial inguinal nodes. The process in the urethra extends along the urethral lumen and infiltrates the periurethral tissues, causing the symptoms of obstruction and local infection.

Clinical Course. The onset is insidious with tumor and urethral stenosis appearing first, then extension with ulceration, formation of fistulas and hemorrhage and, last, infection and metastasis leading to cachexia and death.

Diagnosis. The disease is easily confused clinically with the common urethral stricture and periurethral abscess. Perhaps greater induration and more involvement of surrounding tissues will suggest the diagnosis of carcinoma. The fact that neoplasm, in contrast to stricture, is more common in the pendulous portion and more common in men in later years is an aid. Endoscopy will often help. Unfortunately biopsy can be misleading, since often only the more superficial, inflammatory tissue is obtained.

Treatment. Urethral carcinoma needs radical treatment. Amputation of the penis high above the lesion should usually be followed by excision of the regional lymphatic drainage area. This means complete excision of the iliac and pelvic nodes. If such radical excision is not possible because of the advanced state of the disease, palliative radium or radiation therapy offers the patient little and may cause an increase in the stricture of the urethra which then responds very poorly to dilation.

In women, unless the lesion is very small, radiation is followed by recurrence.

TUMORS OF THE BLADDER

Classification. From a descriptive and etiologic point of view the tumors may be divided into two groups by cell type.

I. Epithelial

A. Carcinoma

1. Papillary (90 per cent)
2. Squamous (5 per cent)
3. Adenocarcinoma (rare)

II. Mesothelial and other (5 per cent)

A. Sarcoma

In addition to cell type, the tumors may be graded by degree of differentiation (Fig. 16). Grade 1: Histologically benign papilloma with cellular uniformity and little nuclear abnormality or mitotic activity. Grade 2: Papillary tumors which show nuclear abnormalities and more than a rare mitotic figure. Grade 3: Papillary tumors exhibiting atypism and excessive mitotic activity. Grade 4: Frankly anaplastic tumors.

An additional and clinically useful classification is based on depth of infiltration as gauged by resection biopsy and by bimanual palpation under anesthesia (Fig. 18). The more benign tumors confined to the mucosa and submucosa of the bladder are classified group A. Group B₁ includes those infiltrating through less than half of the muscle and B₂ through more than half. In group C, the muscle layer has been traversed and perivesical involvement is present. The A and B₁ tumors, which are not palpable bimanually, are potentially curable by complete surgical removal.

Etiology. Workers in the aniline dye industry have a high incidence of bladder tumors, due to ingestion of carcinogenic substances which, on excretion, come in contact with the lining of the urinary tract. Because of the similarity between, and often concomitant occurrence of tumors of the bladder, ureters and renal pelvis and because of the frequent recurrence of similar tumors at areas distant from the site of the initial tumor, we might assume that a carcinogen is present in the urine.

The administration of β -naphthylamine orally to dogs produces bladder tumors. If the ureters are transplanted to the bowel, no tumors form in the bladder but may appear in the ureter. If half the bladder is isolated from the urinary flow, no tumors form, but they will grow if they are implanted there. It has also been shown that it is the unconjugated metabolite of β -naphthylamine, 2-amino-1-naphthol, which produces the experimental tumors. Patients with spontaneous bladder tumors have excessive amounts in the urine of the metabolic products of tryptophan which resemble 2-amino-1-naphthol. In normal individuals these substances occur in small amount and are excreted, conjugated and detoxified with a glucuronide. Urine from patients with bladder tumors contains large amounts of glucuronidase which, splitting the detoxified substance, releases the active carcinogen.

It may be then that administration of a conjugating substance will be practical. At

done, in which filling defects are sought. Filling defects may come from nonopaque calculi and blood clots as well as from tumor. Neoplasms of the renal parenchyma may be difficult to differentiate from renal cysts. Calcification which is typical of these tumors, can be an aid in diagnosis. When blood streams from one ureteral orifice on cystoscopy this is a great help in localizing the lesion. All patients with urinary bleeding should have immediate cystoscopy if the source is not obvious.

Treatment. Surgical approach to the kidney must not be made until complete investigation has established the diagnosis, since exposure of the kidney rarely allows diagnosis and incision of the kidney for biopsy may allow spread of the tumor. In children, a transperitoneal approach is best since the renal pedicle can be ligated and divided before mobilization of the kidney is carried out to reduce the chance for tumor embolism. In adults it is generally easier to mobilize the kidney from the usual lumbar approach, but care must be taken to manipulate the kidney as little as possible before ligating the pedicle and to remove as much of the perirenal fat as possible. If the tumor is growing into the renal vein and vena cava, it is worth while to attempt to remove these prolongations even though the prognosis is grave.

Prognosis. The late mortality after nephrectomy for renal carcinoma is extremely high. Death usually occurs within a year but may occur after five years. The over-all five year rate of cure is about 10 per cent. Irradiation cannot substitute for nephrectomy. Radiation therapy however has a place in late palliation of renal carcinoma and is useful in Wilms tumors. In these cases preoperative irradiation will reduce the size of the tumor and facilitate its removal and postoperative irradiation will give a higher percentage of cures than operation alone.

Tumors of the urinary tract are suspected when hematuria occurs but they may instead produce obstruction and secondary infection. Least often are they discovered as palpable or visible masses.

Diagnosis of renal, ureteral and vesical tumors rests on instrumental and radiologic localization in order that they may be excised surgically. The prognosis in general is poor because bleeding and obstruction are late symptoms.

NEUROGENIC BLADDER

Two components of the bladder smooth muscle and nerves, are concerned in the functional anatomy of neurogenic bladder.

The smooth muscle of the detrusor has the intrinsic quality of all smooth muscle in that it maintains tone even after all motor nerves to the bladder are blocked. An example of this persistence of tone is seen after spinal anesthesia. The characteristics of the bladder whether innervated or denervated, are such that during filling a constant pressure is maintained, each increment of urine being accommodated by appropriate relaxation. Alterations in this property of accommodation occur after overstretching of the bladder or in instances in which there are too many motor impulses to the bladder usually as a result of a short circuit of the reflex arc by injury or with excessive sensory stimuli from the bladder. In Figure 17 are illustrated the sources of nervous impulses to and from the bladder. The supply is double parasympathetic and somatic. The sympathetic nerves are an unimportant portion of the nerve supply. The parasympathetic supply constitutes the main reflex arc and is carried by way of the hypogastric plexus and the pelvic nerve. Stimulation causes contraction of the detrusor. Section first causes complete inability to empty the bladder but later weak uncoordinated contractions of the detrusor occur with fasciculation and hypertrophy.

The somatic supply is carried through the pudendal nerve. Stimulation causes closure of the external sphincter a striated muscle. Section results in relaxation of the external sphincter. Incontinence occurs only if both the internal sphincter and external sphincter are absent.

Physiology. Normal urination is accomplished in five phases: (1) cerebral release of inhibition, (2) detrusor contraction (parasympathetic), (3) relaxation of the internal sphincter (detrusor action), (4) relaxation of the external sphincter (somatic via pudendal) and (5) contraction of the external sphincter bulb and perineal musculature at the termination of urination.

The reflex arcs involved in urination (Fig. 17) are (1) proprioceptive sensation from the filling of the bladder passes over the parasympathetic fibers of the pelvic nerve, by way of the hypogastric plexus to the spinal cord and ascends by way of (2) the fasciculus gracilis to (3) consciousness in the sensory cortex, resulting in inhibition if

of the bladder it is best to resect it as adequately as possible transurethrally and then treat it by radiation therapy. Papillomatosis, in which large areas of the wall of the bladder are dotted with small, fairly benign tumors, is now treated with cobalt head therapy. A bead of radioactive cobalt is placed in the geometric center of the bladder in a water filled Foley bag, so that radiation is evenly distributed to the entire bladder mucosa. Contact irradiation by the way of suprapubic cystostomy, high voltage external irradiation and open resection with deep fulguration are seldom used at our clinic because of poor results and high morbidity. However, since the results of radical surgery in these patients have been disappointing, we have to keep an open mind regarding treatment.

TUMORS OF THE URETER AND RENAL PELVIS

Since tumors of the ureter and renal pelvis arise from the same transitional epithelium as do tumors of the bladder the classification is essentially the same. More tumors of the ureter have been reported in recent years, indicating that they are less rare than formerly supposed.

Pathologic and Clinical Signs. The pathologic changes in transitional epithelium of the ureter and renal pelvis are similar to those which have been discussed under tumors of the bladder. The ureteral tumor as it grows usually produces obstructive symptoms with superimposed infection but it may produce only hematuria, as does the pelvic papillary carcinoma. Diagnosis is made by pyeloureterography, which will often show the characteristic filling defect. Because symptoms appear late too often these cases are overlooked until the tumor is incurable.

Treatment. Ureteronephrectomy is necessary. The percentage of cure is relatively small because extension outside the ureter and pelvis occurs early metastasizing to the bones and liver. Simple excision of a portion of the ureter for a small tumor will be applicable only with a solitary kidney.

TUMORS OF THE KIDNEY

Classification of Renal Parenchymal Tumors.

- I Epithelial tumors
 - A Adenoma
 - B Carcinoma
- II Tumors of connective tissue (sarcoma)

III Embryonal tumors (Wilms tumor)

IV Hare tumors from adrenal rests (true hypernephromas)

Etiology. The origin of parenchymal tumors is unknown. The so-called embryonal (Wilms) tumor is of developmental origin.

Pathology. Adenomas are frequently found at autopsy. These are small benign tumors composed of papillary fronds or tubules.

Carcinoma of the kidney is the most common renal neoplasm. Since Crawitz first suggested that they arose from adrenal rests in the renal cortex, there has been much discussion of the origin of these tumors. His theory is no longer held to be valid, yet these tumors are still often called hypernephromas. Renal carcinoma is a more suitable term. Adenocarcinoma is the common form; it is made up of tubular overgrowth, either papillary or solid. The tumors are yellow and hemorrhage and infarction are commonly seen. The typical cell is large and clear resembling an adrenal cortical cell with a shrunken nucleus. The tumor invades the blood vessels early and metastasizes to the bones and lungs where it forms the typical cannon ball metastases, which are occasionally the first finding. Removal of the lung for a presumed primary tumor of that organ occasionally reveals the renal source; subsequent nephrectomy is carried out with occasional cure.

Embryonal carcinoma (Wilms tumor) is the common renal tumor in children. On microscopic section these tumors show a variety of mature and immature cells especially smooth muscle glandular tissue and sarcomatous tissue usually of a spindle cell variety as well as nervous tissue.

Symptoms. Unfortunately, when any of the classic triad of symptoms of renal carcinoma (hematuria, pain and tumor) is present, the lesion is often well advanced and difficult to cure. Hematuria is the initial symptom in about 40 per cent of the cases, but pain occurs first in 30 per cent and tumor in about 10 per cent. Tumor is the most frequent presenting symptom in children. Approximately a third of patients have all three symptoms at the time of initial examination. Neoplasm may be overlooked as a cause of hematuria because it may be transient, among 148 patients with hematuria from renal neoplasm, only one had hematuria lasting as long as fourteen days.

Diagnosis. If no vesical or urethral source of bleeding is found on cystoscopy and if the bleeding is total, a retrograde study is

function from the usual neurologic examination, but it is needed for correlation and to allow classification of the neurogenic bladder. It also offers a background for application of methods of therapy particularly those employing blocking or sectioning of nerves.

2. Cystometry is done by measuring intravesical pressure during filling of the bladder. Although articles have been written proving statistically that the specific type of neurologic disturbance of the bladder cannot be determined by cystometry alone, the value of the procedure can be readily realized if it is considered as an additional means of determining the type and degree of disturbance rather than as a single independent and absolute diagnostic procedure. A normal cystometrogram is shown in Fig. 18. It should be noted that with each 50 cc. increment of fluid there is accommodation by the bladder so that the pressure remains at a little above 10 cm. of water pressure until bladder capacity is reached between 400 and 450 cc. At this time the patient is instructed to cease inhibition of his voiding reflex. He voids around the catheter.

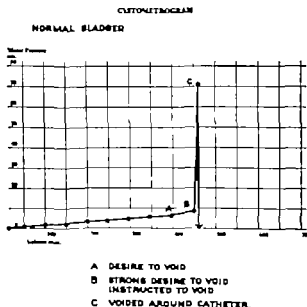
3. Cystoscopy or better panendoscopy with observation of the vesical neck and external sphincter is a valuable adjunct in the diagnosis of neurogenic bladder. For instance, long-standing failure to relax the internal sphincter results in a collar of tissue at the vesical neck which is obstructive in itself. Spasm and lack of relaxation of the external sphincter are also observed and, in addition, any actual obstructive hypertrophy of the prostate in older patients may be observed at this time. The condition of the bladder wall, such as the degree and type of detrusor hypertrophy may also be seen. Concomitant abnormalities are also noted, the principal one being vesical diverticula, which, of course, make restoration of normal bladder function more difficult.

4. Intravenous urography permits changes of the upper urinary tract secondary to disorders of the bladder to be seen and cystography will add information to and confirm the findings of cystoscopy. A cystogram made in conjunction with a urethrogram will allow detection of dilatation of the bladder neck such as is found with lesions of the cauda equina. In addition, ureteral reflux, which results in renal damage is detected. Such a finding warns against the use of tidal drainage, since this will cause further renal damage.

5. Determination of sphincteric (urethral) resistance is important in the diagnosis of imbalance of the bladder since voiding depends on a vesical pressure higher than the opposing urethral resistance. Normal values of urethral resistance are between 20 and 30 mm. of mercury but often only the strength of the sphincteric contraction is measured since the patient cannot relax the reflexly contracted external sphincter. If the vesical imbalance is found to be due to increased urethral resistance, its location should be determined by urethrography and panendoscopy.

6. The function test is perhaps the most important of all, since normal bladder function depends on a correct balance between the forces of filling and expulsion against the resistance of the sphincters and urethra. The crucial test, therefore is measurement of the vesical capacity and of the residual urine since an adequately functioning bladder normal or neurogenic, must have a capacity greater than 200 cc. and must allow retention of less than 30 cc. or no residual urine. These conditions are necessary if serious chronic infection is to be avoided. Any thing short of this cannot be called an adequate bladder.

Treatment of Spinal Shock. After an injury to the spinal cord, almost regardless of site or degree, a period follows of absence of function or of reflex activity below the site of injury. During this period of spinal shock the bladder functions as a denervated organ. Since it is unable to empty itself



ACCOMMODATIONS TO INCREMENTS WITHOUT SENSATION UNTIL NEARLY CAPACITY VOLUNTARY VOIDING AND COMPLETE EMPTYING.

Figure 18

THE NEUROANATOMY OF VESICAL FUNCTION

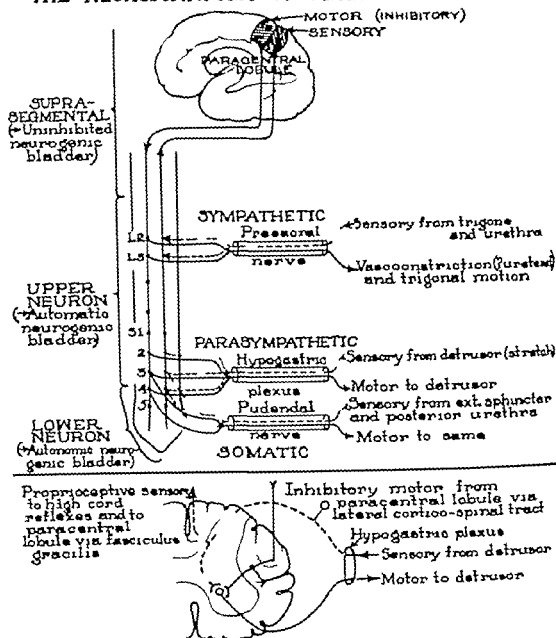


Figure 17

necessary thence to (4) the cerebral motor cortex and (5) down the lateral corticospinal tracts to (6) parasympathetic fibers by way of hypogastric plexus and pelvic nerve to (7) the detrusor

It is important to recognize that this reflex arc can be deficient in any part, consequently disease or trauma can produce an almost infinite variety of functional results. This fact, of course, makes it hard to formulate an exact classification, since the groups are actually continuous with each other.

In classifying the individual patient, the site and degree of the nerve lesion should

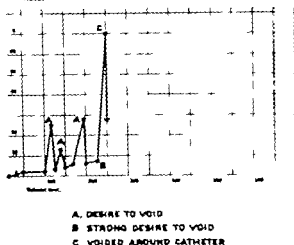
be determined first. This in turn will suggest the effect on the bladder. It is necessary to utilize all the historical information, neurologic data and urologic findings that can be obtained about the patient. Often the type of neurologic lesion is determined by its effect on bladder function. The examination of the individual patient should include the following procedures:

1. A neurologic examination should be performed to establish the level and severity of the lesion as it affects the sensory and motor components of the somatic structures. Little can be learned of and rectal

Table 1 Characteristics of Neurogenic Bladders

I. UNINHIBITED	II. AUTOMATIC (reflex)	III. AUTONOMOUS	IV. SENSORY (atonic)	V. MOTOR
SITE OF LESION (See Figure 17)				
Cerebral or high cord (underdeveloped central control)	A and B Upper neuron	A and B Lower neuron	Dorsal columns	Anterior horn cells
CHARACTERISTICS				
Voiding reflexes to filling are not suppressed	A, Spastic (imbalanced) Bladder takes part in general hypertonicity B, "Normal" (balanced) Bladder independent of control but reflex arc functions well	A, Flaccid (imbalanced) No external nervous control, but atonic B, "Normal" (balanced) No external nervous control, but bladder tone adequate for urethral resistance	Loss of sensation allowed excess filling ending in flaccidity and stony	Sensation normal, but motor paralysis
DISEASE PROCESS				
A, Congenital Delayed development of inhibitory pathway B, Acquired Hemiplegia, brain tumors, multiple sclerosis	A and B Transection of cord	A and B Transection of conus or cauda equina	Tubercle, pernicious anemia, multiple sclerosis, diabetes, syringomyelia	Poliomyelitis
SYMPTOMS				
Eurastia, urgency and frequency occasional incontinence	A Reflex, in voluntary voiding without sensation B Same occasional trigger zones cause voiding	A Overflow incontinence B Continence by periodic forceful evacuation	Painless overflow incontinence	Painful overflow incontinence
TONE				
Normal or increased	A Increased B Normal or increased	A Decreased B Decreased	Decreased (late)	Increased (early)
CAPACITY				
Decreased	A Decreased B Normal or increased	A Increased B Increased	Increased	Increased (late)
RESIDUAL URINE				
0	A 20-50 cc. B 0-50 cc.	A ~ 300 cc. B ~ 30 cc.	500 cc. +	300 cc. +
TREATMENT				
Atropinization (parasympathetic block)	A 1 Remove irritants (infection, calculi) 2, Sacral neurectomy 3 alcohol subarachnoid block B. None.	A and B 1 Presacral neurectomy 2, pudendal block 3, TUR 4 Crede (5 Urecholine)	1 Preserve bladder tone 2, evacuate with straining (3 presacral neurectomy if sphincter tight, 4 pudendal block, 5 Urecholine)	Catheter drainage expectantly
RESULTS				
Good in children	A Fair if treatment prolonged. B Good.	A Poor B Fair	Poor	Good

CYSTOMETROGRAM

UNINHIBITED NEUROGENIC
BLADDER

MANY SMALL CONTRACTIONS NOT CONTROLLED BY
PATIENT ENDING IN STRONGER CONTRACTION AND
VOIDING

Figure 19

because intrinsic detrusor tone is less than urethral resistance the bladder distends with urine. If the distention persists the result is a flaccid, atonic bladder. Consequently the aim of immediate therapy after a spinal cord injury should be to prevent overdistention, which can be accomplished occasionally by intermittent catheterization, more commonly by retention catheterization, or even by suprapubic cystostomy. The last should be reserved for patients treated under conditions where sterile handling of urethral catheters is impossible. The catheter is connected either to a tidal irrigating apparatus set at a suitable level by cystometrographic determinations so that the bladder will fill without overdistention and be periodically emptied, or to a Y tube arrangement with a water filled Kelly bottle which allows manual filling and evacuation of the bladder at intervals. In addition, it is wise to begin prophylactic chemotherapy to reduce the chance of invasive infection since it is impossible to keep the bladder sterile no matter what form of indwelling catheter is used or what antibiotic is given. As soon as the bladder regains tone and function becomes established the catheter can be removed. Removal of the catheter is part of the therapeutic procedure which will be discussed below.

A neurologic approach to vesical dysfunction recognizes three main areas for interference with nervous pathways from the bladder: the suprasacral, which mainly concerns inhibition of lower reflexes, the

upper motor neuron, which concerns the long extravesical reflexes, release of which results in something like normal voiding, and the lower motor neuron, which means short extravesical and mainly intravesical reflexes which leave the detrusor to its own devices. These sites are usually involved by lesions cutting both sensory and motor pathways. If only sensory pathways are interrupted, the reflex arc is likewise broken and, similarly if the motor pathways are cut, dysfunction results. It is obvious that all degrees and modalities can occur but if these major groups are kept in mind, any particular case can be loosely classified (Table 4).

The uninhibited neurogenic bladder results from decreased cerebral inhibition of bladder reflexes. In the normal infant, urination occurs whenever vesical or other regional stimuli reach a sufficient height to act through an upper motor neuron reflex arc, but as the child grows older bladder training allows the cerebral cortex to exercise an inhibitory effect over this simple reflex arc so that reflex emptying can be delayed. Inhibition is obtained first during the waking hours and subsequently becomes so patterned that it is present even during sleep and enuresis ceases.

In some children, however the cerebral inhibitory control does not become developed, perhaps as a result of deficient pathways or from psychologic defect, and the child continues to have uninhibited contractions (Fig. 19). There is then urinary frequency during the daytime and enuresis at night. The characteristics of this type of bladder are normal or increased tone and decreased capacity without residual urine.

Two groups are recognized: a congenital group and an acquired one. In the congenital group administration of atropine in dosage sufficient to atropinize a child, if effective, is diagnostic of the uninhibited neurogenic bladder and, in addition, is therapeutic. The acquired group however which is composed of individuals with cerebral or high cord damage is less susceptible to this form of treatment.

The automatic neurogenic bladder typically results from a complete transection of the cord at a level above the conus. It may also result from a disturbance of the suprasacral arc which stimulates transection of the cord. The result is a reflex arc running from the bladder to the sacral cord, synapsing and running back down to the bladder. The bladder then, as an organ con-

balancing urethral resistance against intra-venal pressure. For emptying to occur the ineffective detrusor contractions, supplemented by the pressure of the abdomen and the hand, must overcome urethral resistance. If urethral resistance is too high, retention of urine will result. On the other hand, if urethral resistance is too low in continence occurs.

Several measures have been proposed to decrease urethral resistance, since the strength of detrusor contraction cannot be affected directly by therapy even by such agents as the parasympathomimetic drug Urecholine.

Pudendal nerve block and section, either unilateral or bilateral will cause partial paralysis of the external sphincter. Trans-urethral resection of the prostatic urethra especially when definite obstructive elements are seen with the panendoscope, will quantitatively reduce urethral resistance. Often three or four resections must be done to secure a good result since initial over-enthusiasm can result in incontinence.

The sensory neurogenic bladder occurs after interruption of the sensory side of the reflex arc. Tabes dorsalis, occasionally pernicious anemia and multiple sclerosis, and syringomyelia are followed by this dysfunction. The patient does not know when his bladder is full because the afferent stimuli are cut off, so that, after a time, gross overdistention occurs, which results in atony. This atonic bladder devoid of motor power is the end result of the sensory defect. The bladder has a large capacity and urination occurs by overflow during straining. Incontinence often brings these patients to the doctor. They are able to empty their bladders only by forceful abdominal pressure and manual compression. A cystometrogram would show a low filling pressure with a very large capacity and no contractions of any sort. Cystoscopically fine trabeculations are seen. The neck of the bladder is open and relaxed, as are the other portions of the detrusor. The external sphincter is not involved by the atony so that its resistance is the cause of the retention of urine and overflow incontinence.

The object of treatment is primarily preventive. If the patients are seen early before bladder tone is destroyed, they can be instructed to void at predetermined intervals, perhaps every two to three hours. Their bladders then will not become overdistended. After overdistention and atony have

occurred, the object of therapy can only be to reduce the amount of residual urine and hope for return of bladder tone. The patient should be instructed to strain at each voiding, and supplement the straining by manual pressure on the bladder. Urecholine may be of some assistance in increasing detrusor activity. In very severe cases a period of drainage by an indwelling catheter will occasionally reduce capacity to a more normal level and restore some tone. The fundamental disease process should be treated. Transurethral resection of the bladder neck is of little value to these patients since the bladder neck is already widely open. Section of the pudendal nerve, either unilaterally or bilaterally in certain cases will promote reduced urethral resistance because of partial paralysis of the normally active external sphincter and so will decrease the amount of residual urine.

The motor neurogenic bladder is the result of loss of the motor side of the reflex arc. It is most commonly seen after poliomyelitis in adults. Sensation is normal so that distention is painful, but overdistention occurs because the bladder is unable to contract as a result of loss of efferent stimuli to the detrusor. If catheter drainage is not instituted overdistention and atony will result.

In the immediate care of the paralyzed bladder the first aim should be to prevent atrophy resulting from overdistention by allowing catheter drainage, second, to avoid and repress infection and, third, to reduce

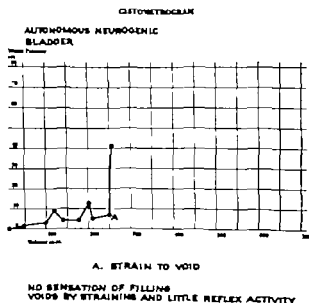


Figure 21

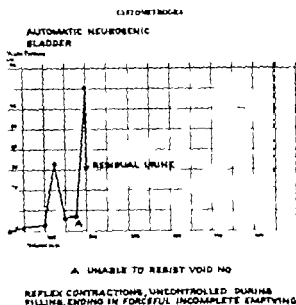


Figure 20

trolled by a simple reflex. The lesions which cause this syndrome are usually injuries to the thoracic or lumbar cord which result in paraplegia.

The diagnosis of automatic neurogenic bladder rests upon locating the site and degree of injury and upon determining the type of bladder which results. The bladder empties by reflex activity arising either from intrinsic stimuli from the bladder wall itself or from extrinsic stimuli which provoke mass movement. These patients have no real sensation of vesical filling, but the increasing size of the bladder as the urine accumulates produces sensations within the abdomen which they may interpret as a full bladder. Urination occurs without warning as soon as the reflex arc is closed, by summation of afferent stimuli.

Two major types of automatic bladders are recognized. The more usual is the spastic type; the other is the so-called normal reflex neurogenic bladder. The former is seen in patients with spasticity of the extremities. It is characterized by small capacity with more or less residual urine. Since urination is of necessity frequent because of the spasticity and small capacity urination is precipitant and inconvenient (Fig. 20). The goal of therapy is a so-called normal reflex neurogenic bladder. If this is obtained, bladder capacity may be as high as 300 cc. Visceral sensation of bladder filling may be enough to give the patient opportunity to reach a convenient place for voiding. Residual urine is low.

The therapeutic aim is first to remove all

irritative foci since the bladder is a purely reflex organ with the sensory stimuli arising within the bladder wall itself both from the mucosa and from the muscle as a stretch reflex. Bladder infection and calculi increase the sensory component of the reflex arc and thereby cause increased stimuli for contraction.

A patient is observed over a period of months and then restudied to learn what sort of bladder function will result from these simple measures. If spasticity persists, blocking of the sensory nerve impulses is necessary and may be accomplished either by alcohol block or by sectioning the sacral roots. Intradural alcohol injection of the lower spinal cord blocks all long reflexes and produces an autonomous neurogenic bladder. The result is greater capacity and relative freedom from the inconvenient reflex voiding of the automatic bladder. A more direct approach is to block the sensory roots of the third, fourth and fifth sacral nerves to cut off the sensory components arising from the irritable bladder and so interrupt the reflex arc. The bladder is then free to relax and to attain a normal capacity. In addition, the irritative stimuli arising from the posterior urethra are cut off releasing the reflex spasm of the sphincter. Thus sphincteric tone is more nearly normal. These procedures may assist patients with automatic bladders to regain almost normal control.

The autonomous neurogenic bladder results from section of the cauda equina and conus usually by trauma, but occasionally by inflammatory lesions and often from such congenital anomalies as meningocele and spina bifida. In contrast to a lesion across the spinal cord above the conus, a lesion through the cauda equina leaves the bladder autonomous (Fig. 21) possessing little or no outside reflex arc. It acts merely by the intrinsic reflex arc through the detrusor ganglia.

Sensation is diminished and coordinated reflex stimulus to the detrusor is absent. The bladder fills against the intrinsic detrusor tone and urination is irregular and incomplete. Depending on the degree of resistance at the vesical neck, the patient will be able to void more or less of the bladder contents by increasing abdominal pressure or by the use of manual pressure over the bladder. The capacity of the bladder may be as high as 350 cc., but the residual urine is, of course, also quite high.

Treatment of these patients is directed at

balancing urethral resistance against intra-vesical pressure. For emptying to occur the ineffective detrusor contractions supplemented by the pressure of the abdomen and the hand, must overcome urethral resistance. If urethral resistance is too high retention of urine will result. On the other hand, if urethral resistance is too low incontinence occurs.

Several measures have been proposed to decrease urethral resistance since the strength of detrusor contraction cannot be affected directly by therapy even by such agents as the parasympathomimetic drug Urecholine.

Pudendal nerve block and section either unilateral or bilateral, will cause partial paralysis of the external sphincter. Trans-urethral resection of the prostatic urethra especially when definite obstructive elements are seen with the panendoscope will quantitatively reduce urethral resistance. Often three or four resections must be done to secure a good result since initial over-enthusiasm can result in incontinence.

The *sensory neurogenic bladder* occurs after interruption of the sensory side of the reflex arc. Tabes dorsalis, occasionally pernicious anemia and multiple sclerosis, and syringomyelia are followed by this dysfunction. The patient does not know when his bladder is full because the afferent stimuli are cut off, so that, after a time, gross overdistention occurs, which results in atony. This atonic bladder devoid of motor power is the end result of the sensory defect. The bladder has a large capacity and urination occurs by overflow during straining. Incontinence often brings these patients to the doctor. They are able to empty their bladders only by forceful abdominal pressure and manual compression. A cystometrogram would show a low filling pressure with a very large capacity and no contractions of any sort. Cystoscopically fine trabeculations are seen. The neck of the bladder is open and relaxed, as are the other portions of the detrusor. The external sphincter is not involved by the atony so that its resistance is the cause of the retention of urine and overflow incontinence.

The object of treatment is primarily preventive. If the patients are seen early before bladder tone is destroyed, they can be instructed to void at predetermined intervals perhaps every two to three hours. Their bladders then will not become overdistended. After overdistention and atony have

occurred, the object of therapy can only be to reduce the amount of residual urine and hope for return of bladder tone. The patient should be instructed to strain at each voiding and supplement the straining by manual pressure on the bladder. Urecholine may be of some assistance in increasing detrusor activity. In very severe cases a period of drainage by an indwelling catheter will occasionally reduce capacity to a more normal level and restore some tone. The fundamental disease process should be treated. Transurethral resection of the bladder neck is of little value to these patients since the bladder neck is already widely open. Section of the pudendal nerve, either unilaterally or bilaterally in certain cases will promote reduced urethral resistance because of partial paralysis of the normally active external sphincter and so will decrease the amount of residual urine.

The *motor neurogenic bladder* is the result of loss of the motor side of the reflex arc. It is most commonly seen after poliomyelitis in adults. Sensation is normal so that distention is painful, but overdistention occurs because the bladder is unable to contract as a result of loss of efferent stimuli to the detrusor. If catheter drainage is not instituted, overdistention and atony will result.

In the immediate care of the paralyzed bladder the first aim should be to prevent atrophy resulting from overdistention by allowing catheter drainage; second, to avoid and repress infection, and, third, to reduce

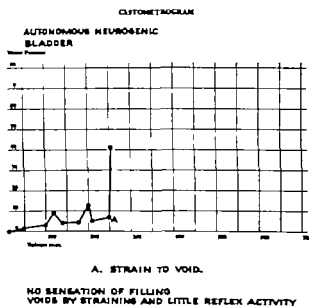


Figure 21

the possibility of stone formation by recommending ambulation, irrigation and a high fluid intake.

In later care, the establishment of the optimum reflex activity must be sought, it often occurs only after six months or longer.

Testing allows differentiation of the autonomic bladder which results from the higher spinal cord lesion, from the autonomous bladder secondary to injury of the cauda equina. But the final goal is balance and the final question is what can the bladder do?

For example a patient with a high lesion voids small amounts frequently. His residual urine is low but his capacity is also low and interpretation of the cystometrogram indicates a quick voiding reflex after small increments during filling. This is an autonomic neurogenic bladder. To achieve balance, the bladder must be calmed by reducing afferent sensations which set off the premature reflex activity. This can be done either by decreasing infection in the bladder and removing stones, or by blocking the afferent nerves by sacral nerve block or subarachnoid alcohol block. If residual urine persists, a transurethral resection of the bladder neck will reduce urethral resistance and help achieve balance.

For another example, a patient suffers a low lesion in the cauda equina resulting in an autonomous neurogenic bladder. He voids by straining, but empties his bladder only incompletely. Interpretation of the cystometrogram indicates that the capacity is good, but adequate voiding pressure is reached only by abdominal compression and straining. To achieve balance in this case urethral resistance must be decreased. Pudendal block, interrupting the somatic nerves to the external sphincter may help. More often, transurethral resection of the bladder neck performed in stages so that total incontinence does not result will achieve balance between the reduced vesical pressure and the surgically reduced urethral resistance.

READING REFERENCES

- Ackerman, L. V., and del Regato, J. H. *Cancer: Diagnosis, Treatment, Prognosis*. St. Louis, C. V. Mosby Company 1954, pp. 725-815.
- Barton, O. V.: The Function of the Vertebral Veins and Their Role in the Spread of Metastases. *Ann. Surg.* 112:138-149, 1940.
- Benken, R. C., and Hinman, F. Jr.: Urinary Tract Injuries in Obstetrics and Gynecology. *Am. J. Obst. & Gynec.* 70:467-485, 1955.
- Born, E., and Cornart, A. E. Effect of Pudendal Nerve Operations on the Neurogenic Bladder. *J. Urol.* 2: 666-670 1951.
- Boyce, W. H., Garvey, F. K., and Norbeck, C. M. Ion-Binding Properties of Electrophoretically Homogeneous Mucoproteins of Urine in Normal Subjects and in Patients with Renal Calculus Disease. *J. Urol.* 72:1019-1031 1954.
- Bull, A. J. *Etiologic Factors in Renal Lithiasis*. Springfield, Illinois, Charles C. Thomas, 1950.
- Campbell, M. F., *Clinical Pediatric Urology*. Philadelphia, W. B. Saunders Company 1951.
- Campbell, M. F., ed. *Urology*. Philadelphia, W. B. Saunders Company 1954.
- Carr, R. J. A New Theory on the Formation of Renal Calculi. *Brit. J. Urol.* 26:105-117 1954.
- Carroll, G. The Changing Flora in Urinary Infections in This Antibiotic Age. *J. Urol.* 73:609-612, 1955.
- Chute, R., and Soby, H. L. Prevalence and Importance of Urine-Splitting Bacterial Infections of the Urinary Tract in the Formation of Calculi. *J. Urol.* 44:590-595 1910.
- Cornart, A. E. Transurethral Vesical Neck Resection, an Adjunct to the Management of the Neurogenic Bladder. *J. Urol.* 72:849-859, 1954.
- Evans, A. T. Renal Arteriography. *Am. J. Roentgenol.* 72:547-585, 1954.
- Fuchs, F.: The Flow of Water Through the Kidney. New York, Manhattan Printing Co., 1944.
- Goldblatt, H. Experimental Hypertension Induced by Renal Ischemia. *Harvey Lect.*, 237-275, 1937 1938.
- Goldman, L., Gordon, G. S. and Chambers, E. L., Jr. Changing Diagnostic Criteria for Hyperparathyroidism. *Ann. Surg.* 146:407-416, 1957.
- Gross, R. E. *Surgery of Infancy and Childhood*. Philadelphia, W. B. Saunders Company 1953, pp. 594-605.
- Higgins, C. C. Ectrophy of the Bladder; Review of 70 Cases. *J. Urol.* 63:85-857 1950.
- Hinman, F. The Pathogenesis of Hydronephrosis. *Surg. Gynec. & Obst.* 58:359-376, 1934.
- Hinman, F. *The Principles and Practice of Urology*. Philadelphia, W. B. Saunders Company 1937.
- Hinman, F. Jr. Congenital Ureteral and Pelvic Dilatation as Evidence of Obstruction. *Monographs in Surgery*. New York, Thomas Nelson & Sons, 1953.
- Hodges, C. V., Gilbert, D. R., and Scott, W. W. Renal Trauma. A Study of 71 Cases. *J. Urol.* 68: 627-637 1951.
- Howard, J. E. Clinical and Laboratory Research Concerning Mechanisms of Formation and Control of Calculous Disease by the Kidney. *J. Urol.* 72:990-1008, 1954.
- Jawetz, E.: *Urinary Tract Infections*. Des Moines, 1934.
- Jewett, H. J. and Strong, G. H. Infiltrating Carcinoma of the Bladder: Relation of Depth of Penetration of the Bladder Wall to Incidence of Local Extension and Metastases. *J. Urol.* 55:366-372, 1946.
- Kegel, A. H. Physiologic Therapy for Urinary Stress Incontinence. *J.A.M.A.* 149:915-917 1951.
- Langworthy, O. R., Kolb, L. C., and Lewis, L. G. *Physiology of Micturition, Experimental and Clinical Studies With Suggestions as to Diagnosis and Treatment*. Baltimore, Williams & Wilkins Company 1940.

- Lapides, J.: The Physiology of the Intact Human Ureter. *J. Urol.* 59:501-533, 1948.
- Lapides, J.: Observations on Normal and Abnormal Bladder Physiology. *J. Urol.* 70:74-83, 1953.
- Lattimer, J. A., Lerman, F., Lerman, P., and Spivack, L. L.: Streptomycin, IAS and Isoniazid in Renal Tuberculosis. *J. Urol.* 69:745-752, 1953.
- Loadbetter, W. F.: The Treatment of Urinary Tract Calculi. *California Med.* 88:269-282, 1958.
- Locké, B.: Lower Nephron Nephrosis. The Renal Lesions of the Crush Syndrome, of Burns, Transfusions, and Other Conditions Affecting the Lower Segments of the Nephrons. *Mil. Surgeon* 99:371-396, 1946.
- Metrowsky, A. M., Scherbert, C. D., and Rose, D. A.: Indications for the Neurosurgical Establishment of Bladder Automaticity in Paraplegia. *J. Urol.* 67:192-196, 1952.
- Miller, E., Wylie, E. J., and Hinman, F., Jr.: Renal Complications from Aortography. *Surgery* 35:885-896, 1954.
- Morales, P. A., and others: Measurement and Significance of Urinary Appearance Time in the Dog. *Am. J. Physiol.* 163:454-460, 1950.
- Narath, P. A.: The Hydromechanics of the Calyx Results. *J. Urol.* 43:145-176, 1940.
- Neublt, R. M., and Lapides, J.: Bladder Tons in Spinal Shock. *J. Urol.* 59:726-733, 1948.
- Prather, C. C.: Injuries of the Bladder. *J.A.M.A.* 154:206-207, 1954.
- Randall, A.: Papillary Pathology as a Precursor of Primary Renal Calculus. *J. Urol.* 44:580-589, 1940.
- Sargent, J. C., and Marypaul, C. R.: Renal Injuries. *J. Urol.* 63:1-8, 1950.
- Short, E., and Carter, A. C.: Aluminum Gels in the Management of Renal Phosphate Calculi. *J.A.M.A.* 144:1549-1553, 1950.
- Silver, H. A.: Wilms tumor (Embryoma of Kidney). *J. Pediat.* 31:643-650, 1917.
- Smith, H. W.: Lectures on the Kidney. University Extension Division, University of Kansas, Lawrence, Kansas, 1943.
- Traut, H. F., McLane, C. M., and Kuder, A.: Physiological Changes in the Ureter Associated with Pregnancy; The Relationship Between Atony and Dilatation of the Tract. *Surg. Gynec. & Obst.* 64:51-58, 1937.
- Wallace, D. M.: Bladder Tumors. In Hanley, H. G.: Recent Advances in Urology. London, J. & A. Churchill Ltd., 1957.
- Weyrauch, H. M., and Goebel, J. L.: Pyelonephritis. In Piernol, G. M.: Cyclopedia of Medicine, Surgery Specialties. Philadelphia, F. A. Davis Company, 1950, vol. 7 pp. 985-1000.
- Williams, D.: Urology in Childhood. In Handbuch der Urologie. Berlin, Springer Verlag, 1958, vol. 15.
- Winton, F. R.: Physical Factors Involved in the Activities of the Mammalian Kidney. *Physiol. Rev.* 17:408-435, 1937.

THE MALE REPRODUCTIVE SYSTEM

By HENRY M WEYRAUCH M.D.

HENRY M WEYRAUCH was born in a small village which nestles in the foothills of the Shawangunk Mountains, one of the small ranges of the Catskills. He became the organist of the First Baptist Church in this village at a salary of fifty cents a week. This first paying job started him off on a career as an organist and choirmaster which helped pay his tuition through Union College and Johns Hopkins Medical School. He received his training in general surgery at the Presbyterian Hospital in New York, and in Urology at the University of Pennsylvania, Duke University and the University of California. At present Doctor Weyrauch is Clinical Professor of Surgery (Urology) and Chief of the Division of Urology at Stanford University School of Medicine.

The male reproductive organs consist of the external genitalia made up of the penis and scrotal contents and the internal genitalia comprising the prostate, seminal vesicles and Cowper's glands (Fig. 1). The functions of the male reproductive system are

1. To produce spermatozoa
2. To perform copulation
3. To produce and secrete hormones

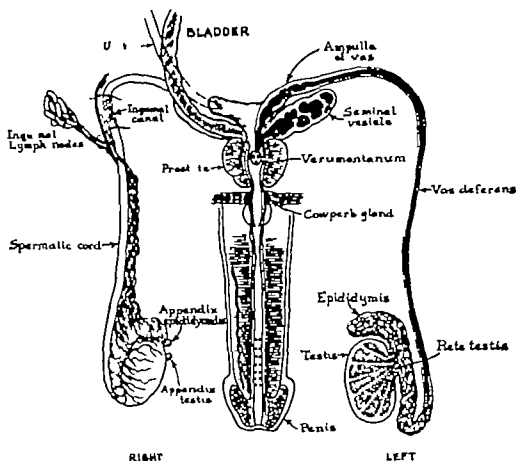
The penis, which is primarily composed of erectile bodies, provides a conduit for the urethra and serves the important function of copulation. The testes produce both spermatozoa and hormones. Other genital structures form the efferent tract by which spermatozoa are collected and conveyed to the outside—the epididymides, vasa deferentia and ejaculatory ducts. Thereafter the spermatozoa are conducted through the

urethra, which serves as a channel for both seminal fluid and urine. The prostate gland, the seminal vesicles and Cowper's glands play an accessory role in providing glandular secretions which serve as a vehicle and lubrication for the passage of spermatozoa.

There will be discussion, first, of pathologic conditions affecting the external genitalia, second, of the internal genitalia and, third, of diffuse abnormalities which affect more than one organ of the reproductive system. These last conditions may be confined to the reproductive system or may participate in systemic abnormality.

EXTERNAL GENITALIA

Although the organs of the external genitalia are in close association they constitute three anatomic units: (1) the penis and



urethra (2) the scrotum and scrotal contents and (3) the inguinal lymphatic system.

PENIS AND URETHRA

The penis is primarily a sexual organ, secondarily it encompasses a portion of the urethra. The three erectile tissue components are the two corpora cavernosa situated dorsally and the corpus spongiosum penis

on the ventral surface (Fig. 2) The corpus spongiosum contains the urethra and is capped distally by the glans penis. The corpora are enclosed in fascial sheaths and all in turn are surrounded and bound together by a dense fibrous envelope known as Buck's fascia. This serves as a barrier against extravasation of urine and the spread of infectious processes. The corpora cavernosa are anchored to the ischiopubic ram. Passing

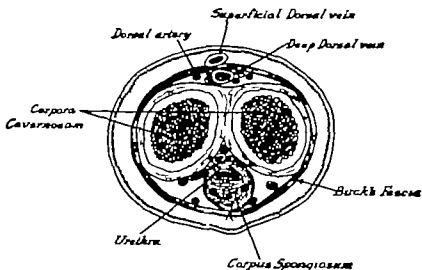


Figure 2. Cross section of penile.

between and below them, the corpus spongiosum surrounds the urethra in its bulbous extent before the urethra enters the external layer of the urogenital diaphragm. The penis is fixed to the pubis at its root by the suspensory ligament. The lax elastic composition of the penile skin permits enormous inflammatory edema and extensive burrowing of subcutaneous blood.

The penis derives a rich blood supply from the internal pudendal arteries. Venous drainage is provided by the dorsal veins of the penis which pass through the prostatic plexuses. This unusually rich vascularity is advantageous in controlling infections and in favoring wound healing. The lymphatic drainage from the penis is to the superficial and subinguinal lymph nodes (that from the glans penis is to the subinguinal and external iliac nodes). The lymphatics from the urethra empty into the hypogastric lymph nodes.

Although the mechanism of erection is poorly understood, it is produced by rapid engorgement of the cavernous bodies by blood through psychic and nervous stimuli. When the blood supply to the penis is impaired or when the nervous supply is damaged the ability to attain erections may be lost.

ANOMALIES

The common anomalies of the penis are phimosis and those in which the urethra is primarily at fault. Anomalies such as absence of the penis, double penis, micropenis, congenital torsion and transposition of the penis and scrotum are rarely encountered in clinical practice. They are likely to be associated with severe abnormalities in other parts of the body which are incompatible with life. Penile anomalies are caused by maldevelopment of the genital tubercle and of those portions of the urogenital sinus involved in the formation of the external genitalia.

Phimosis designates the condition in which the orifice of the prepuce is too small to permit retraction behind the glans penis. It may be either congenital or acquired. Acquired phimosis usually arises from infection and subsequent edema and scarring. Phimosis predisposes to infection of the glans penis and prepuce. In extreme degree it may cause obstruction of the urethra with profound changes in the bladder and the upper urinary tract.

During adult life phimosis impairs the transmission of normal sensation from the nerve endings of the corona of the penis

during coitus. Infection may lead to dyspareunia and difficulty in performing coitus.

Treatment of phimosis consists of circumcision. This operation should be performed during infancy to prevent the described complications. When there is marked infection, preliminary dorsal slit is indicated. At the time of performing circumcision, it is important to release any adhesions between the prepuce and the glans penis and to be certain that the external urethral orifice is of adequate size. If the orifice is too small it should be enlarged by external urethrotomy at the time of circumcision.

Hypospadias is a common deformity of the penis and urethra in which the anterior urethra terminates at some point on the undersurface of the penis, proximal to the normal position at the tip of the glans. Embryologically, hypospadias results from imperfect closure of the urethral groove the margins of which normally fuse in the midline to form the floor of the urethra. It is a hermaphroditic manifestation in which the urogenital derivatives in the male tend to develop toward the female side.

Depending upon the degree of hypospadias the types are designated as balanitic or glandular penile, penoscrotal and perineal (Fig. 3). The fundamental defect is in the urethra and the corpus spongiosum which, excepting in the balanitic type, produces a shortening of the ventral aspect of the penis. This causes a downward curvature of the

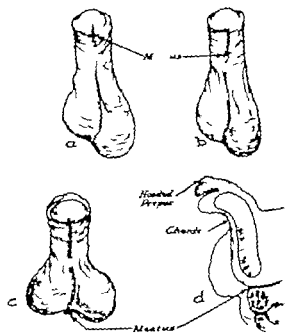


Figure 3. Types of hypospadias: a, glandular; b, midpenile; c, perineal; d, side view. u—perineal.

penis which is most marked during erection known as chordee (Fig. 3d). Deficiency of the prepuce on the ventral aspect causes the dorsal portion to be prominent giving a hooded appearance (Fig. 3d).

Although balanitic hypospadias is largely asymptomatic the other types lead to difficulty in urination due to the abnormal location of the external urethral orifice and to difficulty in coitus caused by chordee. Since the defect is confined to the anterior urethra there is neither deficiency of the urinary sphincters nor incontinence.

Diagnosis is established by inspection of the external genitalia. It is occasionally difficult to distinguish simple hypospadias from hermaphroditism without biopsy of the gonads. Bilateral cryptorchism may be present and complicate the clinical picture.

The treatment is surgical—to enable the patient to urinate and to copulate in the normal manner. As a general rule, disability is so slight in the balanitic type that treatment is not required. Surgical treatment of the other types consists of correcting the chordee by excision of fibrous tissue and constructing a new urethra by plastic operation. Circumcision should be avoided in hypospadias since the skin of the prepuce often proves useful in surgical correction of the anomaly.

Epispadias is a congenital absence of the upper wall of the urethra at some point proximal to the glans which has a "spade-like" appearance. It is an inevitable counterpart of exstrophy of the urinary bladder in the male, when it occurs alone it may be considered as a mild exstrophy. Although epispadias is much less common than hypospadias it is more disabling. The usual site of the urethral opening is at the abdomino-

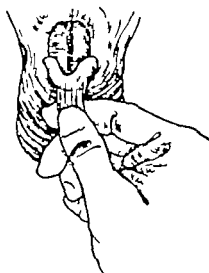


Figure 4 Epispadias.

penilo junction (Fig. 4). There is commonly an associated defect of the urinary sphincters which causes urinary incontinence.

Treatment is surgical by plastic repair of the penis and urethra—to correct urinary incontinence and the inability to copulate.

TRAUMA

With the exception of paraphimosis injuries of the penis are extremely rare. Self-mutilation, such as contusion from pressing or pinching during masturbation, or strangulation from forcing the organ into the neck of a bottle or other firm constricting ring, accounts for many lacerations or gunshot wounds may lead to profuse hemorrhage. A severely injured penis will usually heal promptly if immediately sutured. When the penile blood supply has been completely severed the organ must be amputated.

Paraphimosis is a condition in which the prepuce when once retracted behind the

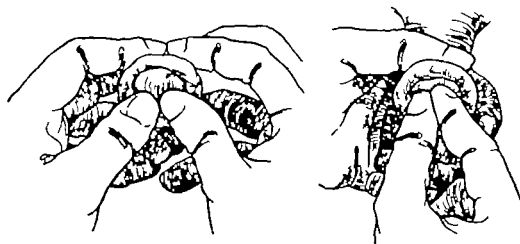


Figure 5 Methods of manual reduction of paraphimosis.

glans penis cannot be replaced in its normal position. It is frequent in the uncircumcised, particularly during infancy. Infection of the prepuce predisposes to development of this abnormality. Constricting rings of skin may lead to vascular occlusion and superficial thrombosis or gangrene. The glans penis is not affected because of its independent blood supply through the corpus spongiosum.

Paraphimosis can usually be reduced manually (Fig. 5) hyaluronidase being first injected into the subcutaneous tissues. In chronic cases and particularly when infection is marked, dorsal slit of the prepuce may be required. Circumcision should be carried out when the inflammatory reaction has subsided. Of chief importance is prophylaxis performing circumcision when the prepuce is tight or redundant and cautioning mothers in the care of infants to avoid leaving the prepuce in the unretracted position.

Dislocation of the Penis results from severe trauma in the erectile state. There is usually associated rupture of the urethra. The penis is inverted and disappears into the scrotum or the adjacent tissues of the inguinal or suprapubic region. There may be considerable swelling from subcutaneous hemorrhage and urinary extravasation. An immediate attempt should be made to restore the penis to its normal position and urinary diversion should be performed.

INFECTIONS

Aside from the venereal infections, which rarely constitute a surgical problem, the penis is relatively immune to infection. The abundant blood supply is helpful in combating pyogenic infections such as cellulitis, erysipelas and furuncles. Nonvenereal infections of the urethra are usually secondary to obstruction or urethral instrumentation.

Posthitis, Balanitis, Balanoposthitis. Balanitis is inflammation of the glans penis; posthitis of the prepuce; the combined inflammatory lesion is known as balanoposthitis. These inflammations are usually caused by retention of secretions and bacteria beneath a redundant prepuce.

Local signs and symptoms vary according to the severity and etiology of the infection. The patient usually complains of local itching, burning and pain and in the more virulent infections a purulent discharge is present. The presence of a discharge may cause confusion in making a differential diagnosis between urethritis and balanoposthitis.

When the infection is not a complication of gonococcal urethritis or other specific disease the treatment is routine cleansing and drying of the involved surfaces. In acute infection the type of bacteria is determined by smear and culture and appropriate antibacterial agents are administered. In infections that resist treatment a dorsal slit is performed to facilitate drainage. When the acute process has receded circumcision is usually indicated to prevent recurrence.

Herpes Progenitalis is a common lesion of the penis caused by a virus. It is manifest by groups of vesicles on the surface of the glans or prepuce along the distribution of the dorsal penile nerve. The lesions are similar to herpes elsewhere. They appear as small, superficial red spots which become vesiculated and rupture leaving superficial ulceration. Mild itching and burning are the chief symptoms. Local cleansing and the application of a dusting powder such as zinc stearate are helpful. When the prepuce is redundant, circumcision is indicated for recurrent herpes.

Condylomata Acuminata, usually known as venereal warts, are soft, painless, cauliflower-like growths which appear on the prepuce, glans penis and within the adjacent urethra. The lesion is aggravated by secretions and dampness. A redundant prepuce is usually the chief predisposing factor. Despite the common name venereal warts are not contracted sexually. Like herpes, they are caused by a virus. The lesions are usually numerous and vary in size from a few millimeters to a centimeter in diameter. Occasionally they attain tremendous size.

In treatment, cleanliness and dryness are the important factors in prophylaxis and in preventing recurrence. For this reason, circumcision is helpful. Once developed, the lesions can usually be eradicated by local application of 25 per cent podophyllin in castor oil. Care must be exercised to avoid application of the drug to uninvolved portions of the skin and mucous membrane. In resistant cases, electrofulguration proves effective.

Cavernositis is an infection of the erectile bodies of the penis, usually secondary to acute gonorrhea or acute nonspecific urethritis. It may complicate trauma, especially with urinary extravasation, and occurs in some systemic diseases such as leukemia.

The condition is evident as a tender indurated area in one of the cavernous bodies.

The urethra may be compressed causing difficult urination. Occasionally cavernositis leads to priapism. Like periurethral abscess, a cavernous abscess may rupture into the urethra. Treatment consists of specific antimicrobial therapy and incision and drainage in resistant cases.

NEOPLASMS

Carcinoma of the penis, the usual malignant lesion, comprises only 1 per cent of all cancers of the urogenital tract. Such common skin neoplasms as nevi, hemangioma and papilloma offer little clinical difficulty since local excision provides both the diagnosis and definitive treatment.

Carcinoma of the Penis usually develops in men over fifty years of age. Circumcision in infancy grants almost complete immunity. Circumcision later in life is valuable but decreasingly so as the patient becomes older. These considerations suggest that the exciting cause operates over a period of many years, apparently contributed to by preputial secretion (smegma) and/or chronic bacterial irritation.

Most of these carcinomas are composed of squamous cells. They may be divided into the verrucous and ulcerated types. The papillomatous may originate from condylomata acuminata and tend to grow quite large although they are less invasive and are of a lower grade of malignancy than flat, ulcerative types. Metastasis is chiefly by way of the lymphatics so that the inguinal and femoral lymph nodes must be carefully examined in every suspected case. However it must be determined by biopsy whether enlargement of these nodes is caused by secondary infection or carcinomatous invasion. Distant metastasis from carcinoma of the penis occurs late, if at all. Since a portion of the lymphatic drainage of the penis extends directly to the pelvic and iliac lymph nodes, these nodes may become involved without invasion of the superficial inguinal lymph nodes.

Symptoms consist of the presence of a painless, slow-growing lesion, most frequent on the glans penis, which may be obscured by a redundant prepuce. Persistent ulceration and the presence of a discharge are common complaints—"a sore which does not heal."

Differential diagnosis must be made with ulcerative lesions of the penis such as chancre and chancroid. Carcinoma is not excluded by a positive serologic test for syphilis. Diagnosis is established by biopsy

With the use of local anesthesia, a small wedge of tissue is removed from non-necrotic tissue at the edge of the ulcer.

In the treatment of carcinoma of the penis surgical therapy remains the method of choice. There is still disagreement as to the most appropriate type of operation, as to whether amputation of the penis 2 cm. proximal to all visible and palpable evidence of the neoplasm suffices or whether this should be combined with extirpation of the inguinal, femoral and pelvic lymph nodes. Radiation therapy, previously employed in treatment, has proved ineffectual and has been largely abandoned.

Carcinoma of the Male Urethra is a rare form of neoplasm, usually of the epidermoid type. It leads to slowing of the urinary stream, hematuria and a urethral discharge. Differentiation from urethral stricture is established by biopsy. Treatment consists of radical surgical extirpation.

SPECIAL DISEASES

In addition to conditions of known etiology there are some penile lesions of idiopathic or protean manifestations.

Plastic Induration of the Penis (Peyronie's Disease) is a common lesion of the fibrous coverings of the corpora cavernosa, usually involving the dorsal aspect and intercavernous septum of these bodies. It appears as a painless plaque of fibrotic tissue similar to keloid formation and to Dupuytren's contracture of the volar tendons of the hand. The incidental finding of a firm area in the penis may call attention to the lesion. As it progresses the fibrotic area limits erection. The penis remains bent in the direction of the involved area. This leads to difficult coitus and sometimes precludes penetration, erection may be accompanied by pain. In most instances the deformity progresses slowly and is subject to long periods of remission regardless of whether or not treatment is instituted. Spontaneous regression occasionally occurs.

Diagnosis is established by palpation of the shaft of the penis. This reveals a well defined plaque of firm tissue, usually located in the midline of the dorsum and near the base of the organ. Occasionally it is placed distally or laterally or deep between the corpora cavernosa. Occasionally x-rays reveal areas of calcification in the area of involvement.

Numerous forms of treatment have been advocated and their multiplicity bears evidence as to the ineffectiveness of them all.

Of reputed value are x ray or radium, vitamin E, ultrasonic wave therapy and injection of the fibrotic plaque with hydrocortisone. Surgical extirpation of the plaque is usually unsuccessful since excision tends to increase rather than to eradicate the deformity. Although in most cases the etiology is unknown in some instances Peyronie's disease arises as a sequel of trauma.

Priapism is a rare affliction manifest by prolonged painful erection of the penis, unaccompanied by sexual desire. In most instances the etiology is unknown. Sometimes there are specific causes such as invasion of the base of the penis by carcinoma of the prostate, penis or urethra, trauma to the corpora or some neurologic disorder such as injury to the spinal cord. Spontaneous detumescence may occur.

Treatment is notably ineffective. In the early stages of the condition the use of Dicumarol or heparin may be valuable in preventing thrombosis. Aspiration of the cavernous sinuses with a large bore needle or open drainage through incision has been employed with questionable results. Under spinal anesthesia manual expression of thickened blood may provide temporary relief. In a large proportion of prolonged cases priapism is followed by the loss of power of erection.

SCROTUM

The scrotal sac encloses the testes, epididymides and structures of the spermatic cords. Under the corrugated skin lie the dartos fibers within this layer are three fascial layers derived from the abdominal wall during testicular descent. The scrotum is divided into two compartments by a septum of connective tissue. It not only supports the testicles but, by relaxation or contraction of the dartos, helps to regulate their environmental temperature. The arteries arise from the femoral, internal pudendal and inferior epigastric arteries. The veins are paired with the arteries. The lymphatics drain into the superficial inguinal and sub-inguinal lymph nodes.

The diagnosis of most scrotal masses can be made by careful palpation and identification of each structure combined with transillumination, and occasionally by radiography. When doubt remains exploration of the scrotum may be simple and safely performed.

ANOMALIES

Anomalies of the scrotum are usually associated with anomalies in other parts of

the reproductive system. The most common are a bilobate scrotum resulting from arrest of fetal development and failure of union of the lateral halves along the line of the median raphe. This results in the formation of a separate pouch for each testicle, frequently associated with perineal hypospadias. When there is congenital absence of the testicle or cryptorchism, the affected side of the scrotum fails to develop.

TRAUMA

Although scrotal injuries are few, trauma is occasionally sustained by kicks, blows or gunshot wounds. In any penetrating injury the chief concern is whether there has been damage to the scrotal contents and whether any foreign material has been introduced. Such wounds should be promptly debrided and tetanus antitoxin administered. As a general rule a compression bandage will check mild hemorrhage but arterial bleeders require ligation and unless this is promptly executed a large scrotal hematoma will develop. The scrotal skin has a large capacity for regeneration and its loss does not usually require skin grafting, although in reconstruction of the scrotum, the testicles may be initially left in a bizarre position. The presence of urinary extravasation, as from a coincident rupture of the urethra, demands definitive treatment. Serious infection may ensue unless the urine is diverted, proper drainage of the involved tissues instituted and appropriate antimicrobial agents administered.

INFECTIONS

Most infections of the scrotum are secondary to infection of the testicle, urethra or rectum or to urinary extravasation. Scrotal cellulitis and furunculosis are similar to these infections elsewhere on the skin surface.

SPECIAL DISEASES OF SCROTUM

Scrotal Edema. Because of the laxity of the scrotal skin and subcutaneous tissues, edema may progress to startling dimensions. The penis may be similarly involved and so dislocated by invagination as to cause interference with urination. If the urine is discharged over the scrotum the skin becomes macerated, predisposing to infection and ulceration. Scrotal edema is frequently associated with cardiac or renal disease, blood dyscrasia or abdominal neoplasms which interfere with venous and lymphatic return. On the other hand, it may be secondary to infection of the testicle.

structure. Treatment of scrotal edema consists of a high scrotal support while definitive treatment is being applied to the primary cause. In the absence of complicating infection, surgical drainage is unnecessary and contraindicated because of the danger of producing cellulitis and bacteremia.

Elephantiasis is a chronic disease caused by interruption of the lymphatic drainage of the scrotum leading to hypertrophy of the skin and edema of the subcutaneous tissue with tremendous enlargement of the affected areas. There are two forms the nonfilarial and the filarial, which is limited to tropical and semitropical regions. Non-parasitic elephantiasis may result from excision or postoperative sclerosis of the inguinal lymph nodes into which the scrotal lymphatics drain. Parasitic elephantiasis is common in many Asian countries, especially India. The condition results from infection with the nematode *Wuchereria bancrofti* an organism which utilizes two hosts man and certain mosquitoes, to complete its life cycle. Filariae are blood borne to the lymphatics where they interrupt drainage of the regional lymph nodes.

In elephantiasis the scrotum may enlarge to such an extent as to reach the level of the knees or even touch the ground as the patient walks. The tissues have a brawny hardness attributable to fibrous infiltration and hyperplasia of the cutaneous tissues which become coarse in texture. Since the cutaneous blood supply is preserved gangrene seldom develops.

In the differential diagnosis it is important to recognize that pyogenic inflammation of the scrotum and urinary extravasation may simulate elephantiasis.

Aside from treating the primary disease if it is recognizable, treatment of elephantiasis consists of plastic excision of the scrotum, preserving the testicles, which are unaffected by the lesion.

Scrotal Gangrene is caused by interference with the cutaneous blood supply of the scrotum. It may result from any one of several causes (1) infected wounds (2) chemical or mechanical trauma without infection, (3) any condition such as cardiovascular disease leading to scrotal edema (4) systemic diseases such as diabetes which predispose to infection (5) urinary extravasation with secondary infection.

Diagnosis is made by inspection. Gangrenous areas of skin show a purple dark green or black discoloration. Treatment consists of appropriate antimicrobial therapy

when there is complicating infection together with excision of gangrenous tissues. Incision and drainage prove inadequate.

NEOPLASMS

Neoplasms of the scrotum are extremely rare. Although a few benign tumors such as polyps, fibromas, adenomas, lipomas and hemangiomas have been reported, a malignant skin neoplasm chimney-sweep cancer holds chief clinical interest. It is occupational, being associated with prolonged exposure to bacterial or chemical products which contain carcinogens. Some sensitivity of the skin in persons of fair complexion may be a possible contributing factor. Pathologically this carcinoma has many of the characteristics of carcinoma of the penis. Treatment consists of wide excision, with removal of one or both testicles in advanced stages of neoplasm. As in carcinoma of the penis radiation therapy is ineffective.

TESTES

The testes are two ovoid, tunic-covered organs, suspended in the scrotum by the spermatic cord. At the upper pole of each is a small pedunculated body, the appendix testis. The testes are enclosed in a dense fibrous capsule, the tunica albuginea, which forms into fibrous septa that penetrate the glandular structure, converging at the hilum. Between the septa are the spermatic tubules which produce and conduct spermatozoa to the vasa efferentia and thence to the epididymis (Fig. 1). Within the framework of the septa are the interstitial cells of Leydig. These cells elaborate an androgenic hormone which influences sexual development and activity. Posterolaterally the testes are attached to the epididymides. Elsewhere they are firmly covered by the tunica albuginea which forms the visceral layer of their investment by the tunica vaginalis testis.

The blood supply to the testis is closely associated with that to the kidney because of a common embryonal origin of the two organs. The arteries to the testis the internal spermatic, arise from the aorta below the renal artery. The venous return is through the pampiniform plexus of the spermatic cord to the spermatic vein. On the right the spermatic vein enters the vena cava below the right renal vein, on the left it empties into the renal vein. The longer blood column on the left with its increased hydrostatic pressure may be a participating cause of varicocele.

ANOMALOUS LOCATION

Cryptorchism	Interrupted During Descent	Abdominal Inguinal Prepubic
Ectopic	Vicarious excursion	Superficial inguinal Sympphysial Penile Femoral Crural Perineal

ANOMALIES OF DEVELOPMENT

In Number	Deficient	Complete Absence (Bilateral) Anorchism Lack of One (Unilateral) Anorchism
	Excess	Polyorchism
In Size	Deficient	Hypoplasia
	Excess	Hypertrophy
Abnormal Position	Lower Pole Upward	Inversion
	Posterior Aspect Anterior	Retraction
Fusion Anomaly	Synorchism	

Figure 6 Testicular anomalies.

The lymphatic drainage from the testis is to the common iliac and lumbar lymph nodes, which in turn communicate with the mediastinal and supraclavicular chains, an anatomic arrangement intimately concerned with the spread of carcinoma of the testis.

The testicular nerves are from the aortic and renal plexuses by way of the spermatic cord. Because of the intimate connection of the spermatic, aortic and solar plexuses abdominal pain may be produced by trauma to or acute infection of the testicle.

ANOMALIES

Of the congenital anomalies of the testicle, anomalous location is the most frequent, cryptorchism much more so than ectopy. Absence of one or both testes or their duplication is a rare anomaly. Hypoplasia is unusual in the normally descended organ. The possible variations are listed in Figure 6.

Cryptorchism, or undescended testicle, is the most common of all anomalies of the male reproductive system. The term connotes the existence of a hidden testicle. The location may be any place along the course of normal descent—intra-abdominal, inguinal or prepubic (Fig 7). In many animals the testes do not descend normally until after birth. In some they descend only during the mating season in others they remain in the abdominal cavity at all times. In man the testes are usually located in the scrotum at birth, but in some instances descent is delayed and may occur at any time up to puberty. Occasionally the testes migrate, being in the scrotum at some times and absent from it at others, being retracted during cold or nervous stimulation by an active cremasteric reflex.

The cause of cryptorchism is obscure. In a few cases (less than 15 per cent) it is the result of endocrine dysfunction, such as

hypopituitarism. At one time mechanical factors were considered to be the cause; in recent years more emphasis is being given to an incapacity of the testis to respond to normal stimuli. The disclosures of testicular biopsy indicate that there is inherent testicular deficiency which may be the cause of arrested descent. Biopsy has shown that in undescended testes there is failure of tubular development which normally takes place after the age of ten years. This failure is observed even after orchidopexy.

The chief diagnostic signs of cryptorchism are absence of the testicle from the scrotum and underdevelopment of the empty scrotal sac. If these findings are complemented by the palpation of a testicle in the inguinal or prepubic region, the diagnosis is established. However if the testicle is in the abdomen and cannot be palpated, cryptorchism must be differentiated from agenesis. As the testicle is palpated in the inguinal canal or abdomen, the patient may feel the typical sensation of the testicle being compressed. The examiner should have warm hands, make certain that the patient is fully relaxed and conduct the examination as gently as possible. When there is doubt, the examination should be conducted on more than one occasion. The differential diagnosis must exclude migrating testicle, the condition with which it is most likely to be confused. When the patient is relaxed, even though a migrating testis has been retracted into the inguinal canal, it can usually be "milked"

into the scrotum—a maneuver which cannot be accomplished with undescended testis.

In cryptorchism androgens are secreted, but sperms are not produced. As a result, the adult with bilateral cryptorchism yields an azoospermatic ejaculate although he has normal libido and potentia. Most adults with unilateral cryptorchism are normally fertile.

Pain in an undescended testicle usually signals the onset of some complication such as inguinal hernia, torsion of the spermatic cord, inflammation or neoplasm. The inguinal testis is relatively fixed and is more easily injured than the mobile scrotal testis.

In all types of undescended testis the incidence of malignancy is about thirty three times more than for the scrotal testis; the intra-abdominal testis being four times more vulnerable than the inguinal one. Orchidopexy does not lessen the incidence of neoplasm.

Since the *processus vaginalis* precedes the testis in its descent, it may reach the scrotum independently and remain patent, even when the testis remains in the abdominal cavity. The condition must be recognized at operation so that the sac can be obliterated and the danger of development of a hernia allayed.

For the treatment of cryptorchism, chorionic gonadotropin may be used in an attempt to produce descent. Small doses of testosterone propionate may also be given, either alone or in combination with chorionic gonadotropin. If there is hypothyroidism, thyroid extract should be given in doses

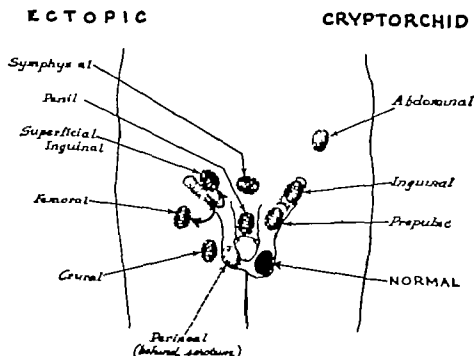


Figure 7 Anomalous positions of testicle.

commensurate with the degree. In assessing the value of hormonal therapy it must be recognized that normal descent may take place up to the time of puberty.

If descent fails to occur spontaneously or following hormonal therapy three alternatives are possible: continued observation, orchiopexy or orchiectomy. The stated objectives of treatment are to produce fertility, eliminate a potential focus of malignant degeneration, correct an associated inguinal hernia and for cosmetic effect. Unfortunately operation rarely if ever restores fertility and apparently it does not reduce the incidence of carcinoma. One questionable reputed advantage is that after orchiopexy the testis is in a better location for observation to detect the earliest changes of malignancy.

In attempting to promote spermatogenesis, some observers advise that orchiopexy be performed before the patient reaches the age of five years; others think that ten years is sufficiently early. There is general agreement that the operation should be performed before the patient reaches puberty.

Orchiectomy is indicated at the time of operation when it is found that it is not possible to bring an undescended testis into the scrotum, providing the opposite testis is present and normally located in the scrotum.

Ectopic Testis. In rare instances the testicle lies outside its line of normal descent. *Vicarious excursion* usually takes place after the testicle has emerged from the inguinal canal. The most common site is in the superficial inguinal region, less common are the subcutaneous tissues of the perineal and femoral regions. The penile position is extremely rare (Fig. 7).

Diagnosis is made by palpating the ectopic testis in its anomalous position. Treatment is surgical, placing the organ in the scrotum.

In extremely rare cases the testis is located in the true pelvis, having strayed from the normal path of descent before entering the inguinal canal. This condition usually is discovered only at necropsy.

TRAUMA

Because of their mobility and the protection afforded by the pelvic arch, the testes are seldom injured. Occasionally straddle or perforating injuries lead to contusion, laceration, puncture or dislocation. Extravasation of blood may cause infarction and atrophy of the testis. In some Oriental countries eunuchs are prepared for the harem by prolonged compression of the testicles with

resultant atrophy. Trauma to the testicle, even mild, may cause excruciating pain referred to the abdomen, associated with nausea and vomiting, faintness and shock. On rare occasions a severe blow to the scrotum dislocates the testicle into the abdomen or the subcutaneous tissues of the penis or perineum. Diagnosis is made on the basis of the history and local signs of injury. Operation is indicated for evacuation of blood and to replace the dislocated organ. Penetrating wounds require suture of the tunica albuginea and scrotal lacerations.

Torsion of the appendix testis is primarily a prepubertal condition. The cause is unknown. It leads to exquisite testicular pain and tenderness on palpation. In the early stages the diagnosis can sometimes be established by palpation of the tiny indurated body. Later with edema, diagnosis may not be made until operation. The condition is frequently mistaken for torsion of the spermatic cord or acute epididymitis. Treatment is operative—excision of the affected appendix.

INFECTIONS

In contrast to the epididymis, which is usually invaded by pyogenic bacteria, the testis is usually invaded by viruses. This exemplifies Cabot's doctrine of the prepared soil. Much like many vegetables which require a certain soil for growth, bacteria and viruses require a particular cellular environment. Bacterial orchitis almost always represents an extension from infection of the epididymides. Only in rare instances, as during bacteremia, do metastatic foci develop in the testis without first infecting the epididymis.

Of the infections, mumps orchitis is most frequent. It rarely develops before puberty but complicates epidemic parotitis in one-fifth of male patients beyond the age of puberty. It begins four to six days after the onset of parotitis, in most instances is unilateral and causes fever.

Physical examination reveals an enlarged, tender testis. Differentiation is made from acute epididymitis by a lack of induration in the epididymis and a sharp sulcus dividing the testis from an inflamed epididymis. The testicle is not elevated, as it is in torsion of the spermatic cord. Clinical evidence of parotitis or of systemic viral infection aids the diagnosis. Less common than mumps orchitis is orchitis complicating smallpox, varicella, measles, influenza and dengue fever.

Prophylaxis consists of keeping patients with viral infections in bed during the acute illness. Treatment is symptomatic elevation of the scrotum and either warm or cold applications to relieve pain. Surgical incision of the tunica albuginea has been advised to prevent atrophy particularly in bilateral lesions. Estrogens and cortisone have been used with some favorable results.

Although the testis retains its hormonal function the testis usually atrophies and spermatogenesis is usually terminated by viral orchitis.

Tuberculous orchitis is usually secondary to tuberculous epididymitis. Syphilitic orchitis has been rendered a medical curiosity by the modern treatment of syphilis in the countries where this is possible.

NEOPLASMS

Fortunately tumors of the testis are uncommon, for they are one of the most dangerous forms of malignancy in the male. The onset is insidious, growth is rapid and early metastasis is frequent. Whereas nearly all tumors of the epididymis are benign, practically all tumors of the testis are malignant. Testicular neoplasms comprise from 1 to 2 per cent of all malignant tumors and account for about 3 per cent of the malignant conditions of the male urogenital tract. Although their age range is from infancy to old age, most are found in patients during the ages of greatest sexual activity twenty to forty years.

Testicular tumors arise from totipotent sex cells capable of reproducing any of the tissue derived from the three primary germ layers. Although indicted by many as a cause, injury has nothing to do with initiating a testicular neoplasm. It may call attention to an existing tumor. The cryptorchid testis is more likely to be the site of a neoplasm than is a normally descended gonad. If the patient has bilateral cryptorchid testes and carcinoma in one, there is such a high incidence of carcinoma in the opposite testis (about 25 per cent) that bilateral orchiectomy is justified.

Testicular tumors may be placed in any one of four groups: (1) seminomas, (2) carcinomas, (3) teratomas and (4) interstitial cell tumors.

Seminoma is more common and less malignant than the other forms; is more radiosensitive, has a lower incidence of metastasis and a lower mortality rate. Tumors with a carcinoma cellular pattern may be either adenocarcinoma or chorioepithelioma. They

tend to invade nearby structures and to metastasize early. Teratomas are bizarre neoplasms which contain differentiated adult structures such as bone, cartilage, brain, skin and organs resembling teeth and hair. Interstitial cell tumors comprise less than 1 per cent of testicular tumors. They arise from Leydig's cells to which they are cytologically similar. In general, they cause masculinization and most are benign.

Early diagnosis of testicular neoplasms is difficult because of a lack of symptoms. Despite the relatively exposed position of the testicle, patients may be unaware of the tumor for a long time. The initial symptom is usually a painless swelling in the scrotum. With further growth, a dull, dragging sensation develops. In the later stages, an abdominal mass representing metastatic lesions in the lymph nodes, may be noted. Gynecomastia has occurred in patients unaware of any testicular swelling, as a result of hormonal secretion by the tumor.

Not infrequently the first manifestation of a testicular neoplasm arises from a metastatic lesion, such as hemoptysis from pulmonary metastasis. Dissemination is commonly by way of lymph channels, although the blood also may be responsible especially in the more malignant types (Fig. 8). Metastatic lesions tend to advance rapidly.

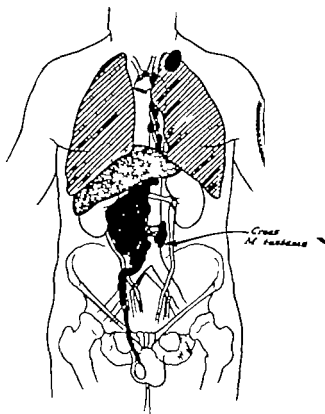


Figure 8 Metastatic areas, carcinoma of testis.

Sometimes the cellular pattern of the metastatic lesion differs from that of the primary neoplasm. Since lymphatic drainage from the testicle is periaortic, rather than to the inguinal lymph nodes, extension is to the peri iliac and thence to the periaortic lymphatics. The metastasis tends to skip glands and to cross to the opposite side. Involvement of the glands surrounding the renal pedicle causes displacement of the kidney and upper portion of the ureter. Spread may be as high as the mediastinum and supraclavicular lymph chains (Virchow's node). The thoracic duct and its surrounding lymphatics are frequently involved. Blood borne metastasis is commonly to the lungs and the liver.

The size of the primary tumor bears no close correlation with the time of onset or multiplicity of metastatic lesions. Many neoplasms 1 cm. or less in diameter may spread to other organs. In some patients the primary growth is discovered only after metastatic lesions appear. Others come to necropsy before the true nature of the metastasis becomes known.

In examination of the external genitalia it is important to determine whether any scrotal mass lies in the testis or in the epididymis since enlargement of the epididymis is usually benign. The typical characteristic of a testicular neoplasm is a stony hard mass of increased weight confluent with the structure of the testis. Although tumors are rarely painful, the fact that tenderness is present does not rule out a neoplasm. Occasionally a tumor coexists with epididymitis. Increased vascularity of the scrotum and invasion of the spermatic cord are late signs. With extensive lymphatic involvement there may be edema of the lower extremities.

Auxiliary diagnostic studies are x rays of the chest and intravenous urograms. The former discloses pulmonary metastasis, the latter displacement of the kidney or ureter by enlarged periaortic lymph nodes (Fig. 8). The urine should be examined for chorionic gonadotropin since this hormone is excreted in many testicular tumors. It is most marked in the carcinoma group, less common in seminomas. It usually does not occur in the teratoid group. Since hormonal tests may be negative in all types of tumor their value in this diagnosis is limited.

Testicular neoplasms have been mistaken for hydrocele, spermatocele, hematocele, epididymitis, torsion of the spermatic cord and orchitis. If hydrocele is present, aspira-

SEMINOMA

600 to 800 r

(1000 r adequate dosage)

CARCINOMA

2000 to 3000 r

CHORIOEPITHELIOMA

5000 to 6000 r

Lethal dose for mature TERATOMA cannot be reached with safety

Figure 9 Radiosensitivity of testicular neoplasms.

tion of the fluid permits better examination of the testis. Whenever the nature of an intrascrotal enlargement is doubtful, operation should be performed.

The treatment of malignant tumors of the testis is orchiectomy supplemented by radiation of the primary lymph zone, providing the tumor is radiosensitive. Radiosensitivity is not always easy to determine because the metastatic lesions may show a different pattern than the primary tumor. Seminomas are the most radiosensitive; chorioepithelioma and carcinoma are more resistant, the lethal dose for mature teratoma cannot be reached with safety (Fig. 9). In treating the latter types, excision of the regional lymphatics, including those about the renal pedicle, is advised by some surgeons. Orchiectomy for neoplasm consists of ligation of the spermatic cord at the uppermost extent of the inguinal canal and removal of the intact cord and scrotal contents. In gland dissection, lymph nodes on both sides of the aorta should be removed because of the tendency to cross-metastasis.

Prognosis depends largely upon the type of neoplasm. Teratomas without malignant elements can be cured by orchiectomy alone. Seminomas respond well to orchiectomy and deep x ray therapy. Embryonal carcinomas and teratocarcinomas have a higher mortality rate and chorioepitheliomas are the most malignant. As a general rule, decrease in the chorionic gonadotropic hormone indicates removal or regression of chorionic tumor tissue; increase means spread of the disease. However after operation, negative hormonal tests are occasionally noted in the presence of widespread metastasis.

EPIDIDYMIDES

The epididymides are a pair of crescentic structures composed of tremendously coiled ducts. They lie posterolateral to the testes (Fig. 1) to which the rete testis provides

communication. The appendix epididymis is a small, pedunculated body attached to the upper pole. The elongated upper end of each epididymis, termed the globus major, is flattened against the testis; the body comprises the midportion; the lower end, the globus minor, gives rise to the vas deferens. An infection in the globus minor may seal off the spermatic ducts, causing sterility of the involved testis.

In addition to providing conduits, the epididymides contribute to the maturation of spermatozoa. Spermatozoa first become motile after passage through the head of the epididymis. However, the fertilizing capacity of spermatozoa recovered from the head is low; those recovered from the body or tail are more fertile.

The arterial supply to the epididymis comes from the internal spermatic artery and the artery of the vas deferens. The venous drainage is through the pampiniform plexus into the spermatic vein. The lymphatics drain into the external iliac and hypogastric lymph nodes.

ANOMALIES

The most usual anomaly is in position, the epididymis lying anterior rather than posterior to the testis. Lack of fusion between the epididymis and testis has been reported. Congenital absence of the epididymis is extremely rare.

TRAUMA

Injuries of the epididymis accompany trauma to the testis. Differential diagnosis is made at operation. Clinical recognition and treatment are similar to those of injury of the testis.

INFECTION

Epididymitis is the most common disease of the scrotal contents and is relatively frequent in males of all ages. It may be non-specific, gonococcal or tuberculous.

Nonspecific Epididymitis is commonly a sequel to prostatitis, the usual pathway of invasion being the vas deferens. Acute nonspecific epididymitis, spread by the blood from a distant focus, is rare. Any of the bacteria which invade the urinary tract may cause epididymitis.

Acute epididymitis is a fulminating process manifest by diffuse cellulitis with swelling and edema. The inflammation usually arises in the tail of the epididymis and thereafter involves the entire organ. The chief symptoms are pain and swelling. There

is frequently a prodromal ache in the inguinal region or discomfort along the spermatic cord. Systemic symptoms such as chills, fever, headache, nausea and vomiting occur with more virulent infection.

Diagnosis is established by gently palpating the enlarged, exquisitely tender epididymis. An important physical sign is detection of the sulcus between the enlarged epididymis and the testis. Involvement of the vas deferens by coincident vasitis contributes evidence of epididymitis. The spermatic cord may be thickened by edema. Hydrocele may develop secondary to infection. Specific diagnosis is made by identifying the bacteria present in any urethral discharge or in the urine. Digital rectal examination should be performed to learn whether there is coincident prostatitis or seminal vesiculitis. Palpation must be gentle to avoid producing bacteremia and dissemination of the genital infection.

In making a differential diagnosis it is important to rule out torsion of the spermatic cord or of the testicular appendages. Elevation of the scrotum usually relieves the pain of epididymitis but not of torsion. In torsion of the spermatic cord, the testicle lies high in the scrotum. When examination is unsatisfactory because of exquisite tenderness, injection of the spermatic cord with 1 per cent Xylocaine will facilitate the procedure. The physical findings in trauma to the testicle may simulate those of acute epididymitis, but the history of injury and the absence of a urethral discharge or pyuria aid in differentiation. When doubt exists, diagnosis is established by operation.

Treatment consists of prompt administration of the appropriate antimicrobial agent and elevation and immobilization of the scrotum. The patient with acute epididymitis should be kept in bed until any fever subsides in order to help limit spread of the infection. Indications for epididymotomy are a high unremitting fever, excruciating pain and bilateral epididymitis. It is believed that by performance of epididymotomy, resolution is more likely to take place without scar tissue formation and resulting sterility.

Epididymitis secondary to operation on the prostate has been largely eliminated by performing bilateral vasectomy before or at the time of prostatectomy.

Chronic epididymitis is usually a sequel of acute epididymitis but at times arises insidiously as a subacute process giving rise to little pain or discomfort. In chronic infection, the organ is thickened and slightly

enlarged. It may or may not be tender. Differential diagnosis must be made from tumor of the testis by carefully palpating the testicle to be certain that any induration is confined to the epididymis. Since the two may occur together whenever there is a coincident mass in the testis operation is indicated to learn the true state of affairs. Nonspecific epididymitis is treated by eliminating the source of the infection usually a prostatic infection. In such cases, vasectomy usually prevents acute exacerbations. If chronic epididymitis causes persistent pain, epididymectomy is indicated. Administration of antimicrobial agents is usually fruitless. Epididymovasostomy with anastomosis of the vas to the upper part of the epididymis, may restore fertility if fibrosis is limited to the globus minor.

Gonococcal Epididymitis used to be a common complication of gonococcal urethritis. With the reduced incidence of gonorrhea, brought about by modern antimicrobial therapy it is now rare. The infection nearly always results from improper treatment of anterior gonococcal urethritis, allowing extension to the posterior urethra and thence down the vas deferens to the epididymis. The lesions and symptoms of both acute and chronic gonococcal epididymitis are similar to those of nonspecific epididymitis with one significant difference. With resolution of the acute inflammation the tissues affected by the gonococcal process are more likely to be replaced by scar tissue which occludes the epididymal tubules. If this is bilateral, sterility results. Scar tissue easily palpable causes permanent induration in the globus minor of the epididymis.

Tuberculous Epididymitis. The epididymis is one of the common sites of urogenital tuberculosis. The incidence of epididymitis is highest in patients from twenty to forty five years old and is usually secondary to tuberculous prostatitis or seminal vesiculitis. Extension is usually by way of the vas deferens, less commonly it spreads by means of the blood from a distant focus, for example in the lungs or kidneys. Tuberculous epididymitis usually presents as an indurated enlargement of the organ. Upon palpation, nodularity and thickening are noted, a process which may involve the vas deferens. There is usually little tenderness, despite considerable involvement of the epididymis. In advanced cases, caseation takes place. The process may then involve the subcutaneous tissues and the skin, causing edema, ulceration and fistula formation. The

testis is invaded late being relatively immune to the tubercle bacillus. Secondary infection may take place giving rise to all the manifestations of acute nonspecific epididymitis.

With respect to diagnosis, the history and presence of tuberculosis elsewhere, particularly in the urogenital tract, should suggest tuberculosis. Proof is obtained by isolating tubercle bacilli in the urine or discharge from a sinus and with surgical exploration by microscopic examination of the tissue. In chronic cases calcification may be noted on x ray of the scrotum. Differential diagnosis must be made between nonspecific epididymitis, coccidioidomycosis and tumor of the testis or epididymis.

If the lesion is not far advanced, chemotherapy and general measures directed to tuberculosis elsewhere in the body may suffice. If there is caseation and particularly if there is scrotal fistula, operation is indicated, excising the vas deferens, epididymis and involved tissues of the scrotum.

NEOPLASMS

Tumors of the epididymis are extremely rare and, in contrast to testicular neoplasms, most are benign. These include angioma, myoma, leiomyoma and dermoid cyst. Examination shows a mass in the epididymis which does not transilluminate. Tumors may be mistaken for tuberculous epididymitis. The precise diagnosis will seldom be made without microscopic examination of tissue. Malignant neoplasms of the epididymis are chiefly adenocarcinoma, although occasionally a sarcoma is present. Since these tumors grow rapidly and frequently invade the testis they may be mistaken for testicular neoplasm. Treatment is similar to that for tumor of the testis i.e., removal of the testicle followed by radiation. The prognosis is usually poor.

MISCELLANEOUS CONDITIONS

A spermatocele consists of a diverticulum of the epididymis (Fig. 10a). It is a single or multilocular cystic structure communicating with the vasa efferentia. It contains a cloudy fluid usually found to be teeming with motile spermatozoa when examined microscopically. The typical spermatocele is a small, cystic, nontender spherical mass which transilluminates usually attached to the upper pole of the epididymis. Usually the condition is asymptomatic and discovered as an incidental finding on examination of the scrotal contents. The vent is not

indicated unless the spermatocele attains considerable size and causes pain in which case excision is indicated. The differential diagnosis lies chiefly between various types of hydrocele or the rare epididymal neoplasm. The cystic consistency of the mass and the characteristic of transillumination establish the diagnosis. Aspiration of the spermatocele yields fluid containing spermatozoa. However this procedure is followed by recurrence and there is the hazard of producing hemorrhage and infection.

TUNICA VAGINALIS TESTIS

The tunica vaginalis testis is a pouch formed by two layers of connective tissue. It is a remnant of the peritoneal prolongation into the scrotum. The visceral layer covers most of the epididymis and is firmly attached to the tunica albuginea testis the parietal layer forms the outer covering, a secretion from the cells of the endothelial lining lubricates the space between the two. When excess fluid accumulates, a hydrocele is produced. Although the processus vaginalis is usually open at birth, it is closed within the first month in most infants.

ANOMALIES

When the processus vaginalis fails to close and remains patent in its course through the

inguinal canal and spermatic cord it predisposes to an indirect hernia and to communicating hydrocele. The diagnosis of an inguinal hernia (Fig. 10b) is made on the basis of physical signs. The scrotal mass is soft, compressible, usually reducible and does not transilluminate. Auscultation may disclose peristaltic sounds or gurgling from intestinal contents. The presence of a communicating hydrocele (Fig. 10c) is not usually distinguished from a hydrocele of the tunica vaginalis testis until at operation a communication with the peritoneal cavity is found.

TRAUMA

Injury of the tunica vaginalis is nearly always caused by a blow to the scrotum. The response of the tunica may be the production of a large serous effusion known as a traumatic hydrocele (Fig. 10d). If the cavity becomes filled with blood it is known as a hematocele. A scrotal hematoma is a collection of blood in tissues outside the tunica. Acute hydrocele is similar to idiopathic hydrocele which will be discussed below. Spontaneous hematocele has been observed in arterial disease such as arteriosclerosis and in blood dyscrasias such as purpura. Traumatic lesions may or may not be painful.

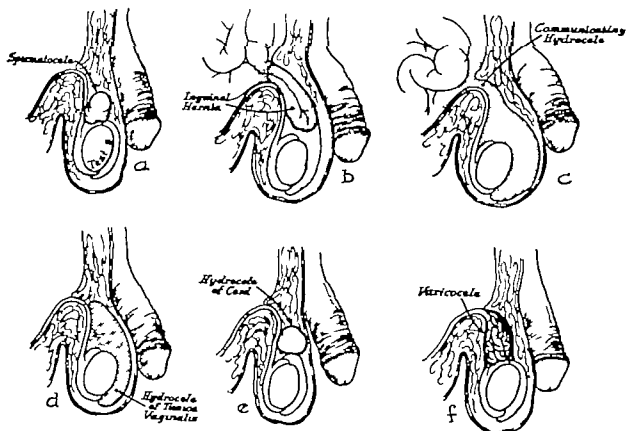


Figure 10 Common lesions causing a mass in the scrotum.

Hematocoele does not transilluminate. In addition, the blood may be under such tension that the surface feels stony hard and heavy findings similar to those in tumor of the testis. The treatment of hematocoele is surgical evacuation of the blood with excision or reflection of the tunica. Usually hematocoele is evident in the history of trauma. In addition to tumor of the testis, differential diagnosis must be made from torsion of the spermatic cord and acute epididymitis.

IDIOPATHIC HYDROCOELE

A hydrocoele constitutes an abnormal accumulation of fluid in the tunica vaginalis testis (Fig. 10d) or in an incompletely obliterated processus vaginalis, known as hydrocoele of the cord (Fig. 10e). Although the lesion is occasionally infectious or traumatic, the cause is usually unknown. Hydrocoele is one of the most common causes of scrotal swelling. Small hydroceles are asymptomatic and go unrecognized by the patient. With large accumulations of fluid the prevailing complaint is of a mass in the scrotum, sometimes with a dragging sensation. Occasionally hydroceles attain such tremendous size as to interfere with walking and sexual intercourse. The diagnosis is made on the basis of a symmetrical, cystic mass which is not tender. Hydrocoele of the tunica vaginalis testis is confluent with the testis which is observed posterolaterally on transillumination of the scrotum. In hydrocoele of the spermatic cord the testicle is felt apart from the cystic mass and in large hydrocoele is displaced to the base of the scrotum.

A hydrocoele of long standing may evoke fibrous tissue or calcification in the wall and fail to transmit light and thus be mistaken for neoplasm or hematocoele. Elephantiasis of the scrotum is easily differentiated because the scrotal edema is bilateral and unaccompanied by a cystic mass within the scrotum.

Small hydroceles require no treatment. Those observed in infants and secondary to epididymitis may recede spontaneously. Whereas aspiration and injection of a sclerosing material has been attended with some success, this method of treatment may lead to infection and hemorrhage and is not recommended. Treatment should be surgical, either excising the parietal tunica vaginalis testis or reflecting it behind the testicle to prevent the reaccumulation of fluid. In addition to yielding a permanent cure, operation affords accurate examination of the scrotal

contents, ruling out any associated hernia, tumor or other abnormality.

SPERMATIC CORD

The spermatic cord is composed of the vas deferens, the artery of the testis, the epididymis, the pampiniform plexus, the lymphatics and the autonomic nervous supply. In addition there may be a patent or partially obliterated processus vaginalis which may lead to inguinal hernia or hydrocoele of the cord.

The vas deferens extends from the epididymis to the inguinal canal and thence retroperitoneally to an ampulla before it joins the outlet of the seminal vesicle to form the ejaculatory duct.

ANOMALIES

Anomalies of the vas deferens consist either of an absence or aplasia of the structure which, if bilateral, accounts for isolated cases of sterility. These are an occasional accompaniment of renal agenesis or hypoplasia.

TRAUMA

Torsion of the spermatic cord (Fig. 11) is produced by an axial rotation and the testicle is destroyed unless the torsion is promptly corrected by operation. As the cord twists, the blood supply to the testicle is diminished or completely cut off. As a rule, gangrene results. If there is not complete impairment to the circulation a few spermatogenic cells survive but atrophy is almost inevitable.

The condition is almost limited to boys

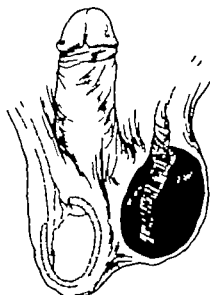


Figure 11 Torsion of spermatic cord causing gangrene of testis and epididymis.

nearing the age of puberty. It may occur in the cryptorchid testis as well as in the normally descended gonad. Although torsion is frequently attributed to a laxness of the tunica vaginalis testis, this is not always present. Torsion may take place within the tunica vaginalis (intravaginal torsion) or outside the tunica vaginalis (extravaginal torsion).

Rotation may be as minimal as a quarter turn, although as many as five complete turns have been seen. Apparently the lesion is produced by a violent contraction of the cremaster muscle. Sometimes there is a history of strenuous physical activity such as running or jumping, but in other instances, torsion develops as the patient lies quietly in bed. Rarely torsion corrects itself spontaneously. Some patients experience recurrent attacks. Since the condition is likely to be bilateral, when operation is performed to correct torsion on one side, it is advisable to also suture the opposite testicle in place.

The typical onset of torsion is manifest by exquisite testicular pain and swelling. Abdominal pain, nausea and vomiting are common accompaniments. Rotation of the spermatic cord leads to elevation of the testicle, a valuable diagnostic sign. When untreated, the local process continues to cause acute symptoms for two or three days after which gangrene ensues with lessening of pain and relative anesthesia of the involved structures. Atrophy takes place over a period of several months.

A valuable diagnostic sign is that the testicle under torsion lies at a higher level than the normal organ. In assessing this sign, it is important to allow for the fact that the left testicle is normally at a lower level than the right. The chief error in differential diagnosis is to mistake torsion for acute epididymitis. In the latter condition the urine usually contains pus cells and bacteria, and physical examination of the scrotum is of considerable help. In torsion it is impossible to make out a sulcus, as one can between an enlarged, tender epididymis and an uninvolved testis. The entire testicle is exquisitely tender and is palpated as one boggy or indurated mass.

Torsion of the appendix epididymis and appendix testis must also be considered in differential diagnosis. These may twist on their pedicles producing a similar but more localized and less violent reaction. Soon after it occurs, torsion of an appendage produces a tender indurated nodule at the upper pole of the testicle; later scrotal edema obscures this sign.

Since examination may be extremely painful in acute conditions it is helpful to infiltrate the spermatic cord at the level of the external inguinal ring with a solution of 1 per cent Xylocaine. This relieves pain and makes it possible to palpate the structures in the scrotum. Aspiration of bloody fluid from the tunica vaginalis testis is a diagnostic sign of torsion. Blood is not present in epididymitis or orchitis. Whenever the diagnosis is in doubt, immediate exploratory operation is indicated. In torsion of the spermatic cord, immediate surgical correction offers the only hope of preserving the testicle. If operation is delayed more than three or four hours atrophy is inevitable. In many cases orchiectomy is necessary because of gangrene of the testicle.

INFECTION

Funiculitis, inflammation of the spermatic cord, is usually a complication of epididymitis or of vasitis. Acute funiculitis causes swelling of the cord with tenderness and induration. In chronic funiculitis the cord may be edematous and increase of connective tissue may suggest a mistaken diagnosis of hernia. Infection of the vas deferens extends upward from concurrent epididymitis below or downward from prostatic infection above. In the latter instance, if vasectomy has been performed, the process ends abruptly at the operative site, preventing extension to the epididymis. In acute vasitis there may be local tenderness, but this is mild in comparison to that in epididymitis and there is no febrile reaction from vasitis as is usual in epididymitis. In chronic vasitis the vas becomes fibrosed, beaded or nodular; the latter is common in tuberculosis.

NEOPLASM

Tumors of the spermatic cord are usually secondary to tumors of the testis. Primary tumors of the vas deferens are extremely rare. A few have been observed similar to those of the epididymis.

VARICOCELE

Varicocele (Fig. 10f) is characterized by elongation, dilation and tortuosity of the veins of the pampiniform plexus usually on the left side. Most varicoceles are idiopathic and occur between the ages of fifteen and thirty-five years. Secondary varicocele results from obstruction to the spermatic vein by intra-abdominal tumors particularly tumors of the kidney. Small varicoceles rarely give rise to symptoms. If large, they may

produce pain in the scrotum. Diagnosis is suspected on inspection, the wormlike configuration of the dilated veins being seen through the scrotal skin. This observation is confirmed by palpating enlarged soft dilated veins within the spermatic cord.

For asymptomatic varicocele treatment is not indicated. When there is discomfort or pain, a snugly fitting scrotal suspensory may provide relief. Operation is indicated for large varicoceles which cause pain. The dilated segment of veins is excised and the testicle suspended by suturing the distal stump to the external inguinal ring.

INGUINAL LYMPHATICS

The inguinal lymphatics warrant consideration since they are intimately concerned with many of the lesions of the penis and scrotum. There are three groups of superficial inguinal lymph nodes which lie above and external to the saphenous opening. A deep group is situated around the femoral vein at the level of the saphenous opening. The lymphatics of the shaft of the penis, the penile skin and scrotum drain into the superficial inguinal lymph nodes; those of the urethra into the deep inguinal and hypogastric nodes and those from the glans penis to the external iliac nodes.

INGUINAL ADENITIS

Inguinal adenitis may be secondary to infections of the penis or scrotum. In an acute process the nodes usually become enlarged and tender and the condition may progress to suppuration. In the chronic form, the nodes are discrete and painless. Suitable treatment of the local primary inflammation of the genitalia minimizes the development of secondary adenitis. Treatment of acute lymphadenitis consists of appropriate antimicrobial therapy, rest in bed and cold application to the affected areas. Chronic adenitis rarely requires treatment. Chan-roidal bubo represents a lymphatic extension from a chancre, usually on the penis. This lesion tends to suppurate and requires surgical drainage.

GRANULOMA INGUINALE

Granuloma inguinale is a superficial ulcerative skin lesion with complicating inguinal adenitis. The etiologic agent is an intracellular monocystic Donovan body transmitted by sexual contact. Although the lesion is prevalent in tropical areas, it has a low incidence in the United States. It is seen more frequently in the southern states and

in the Negro population. The lesion is essentially an indolent granuloma of the skin. There are usually multiple painless lesions. Advancing borders leave dense scars in their wake. Secondary infection is common. Diagnosis is made by finding the Donovan bodies in the smear of the caudate. Treatment consists of administration of streptomycin and tetracycline or their derivatives.

LYMPHOGRANULOMA VENEREUM

Lymphogranuloma venereum (lymphopathia venereum, lymphogranuloma inguinale) is a rare inguinal adenitis produced by a filtrable virus transmitted by sexual contact. Blockage of the inguinal lymphatics may cause elephantiasis of the penis and scrotum. Diagnosis is suggested by inguinal adenitis and fistulas from involved lymph nodes. Confirmation is made by the intradermal Frei test and complement fixation test. Although tetracycline is useful in treatment, surgical excision of the involved areas is frequently required when there is secondary infection.

METASTATIC NEOPLASM

Neoplastic invasion of the inguinal lymph nodes is a common sequela of carcinoma of the penis and of the scrotum. Differential diagnosis between lymphadenitis and carcinomatous invasion can be established only by biopsy since secondary infection is so frequent in carcinoma of the genitals.

Since the lymphatic drainage of the penis and urethra is not only to the inguinal nodes but also by a separate route to the hypogastric and iliac groups of the pelvis, excision of the inguinal nodes is insufficient if radical operation is to be performed for carcinoma of these organs.

INTERNAL GENITALIA

The internal genitalia are the prostate, seminal vesicles and Cowper's glands. Their function is to provide secretions for storage of spermatozoa and to convey them during copulation. It has been suggested that the prostate may produce a hormone, but the evidence is not convincing. In addition to providing a vehicle, seminal plasma furnishes extracellular nourishment for sperm, supplementing the intracellular nutritional reserve. Ninety-five per cent of a total volume of 2 to 4 ml. of spermatic fluid is provided by the prostate and seminal vesicles.

PROSTATE

The prostate (Fig. 12) is a musculoglandular body surrounding the vesical neck.

Through its central portion it transmits the posterior urethra, posteriorly it is perforated by the ejaculatory ducts. These open into the verumontanum on the floor of the prostatic urethra. The normal prostate weighs about 20 gm. and is roughly pyramidal. It lies deep within the pelvic cavity, below the symphysis pubis, being supported anteriorly by the puboprostatic ligaments and inferiorly by the urogenital diaphragm. In the embryo the gland is derived from five epithelial evaginations of the posterior urethra which form into compound alveolar glands and empty into ducts opening into the posterior urethra.

The blood supply to the prostate is from the inferior vesical and internal pudendal arteries. The prostatic veins form plexuses about the sides of the gland and drain into the internal iliac veins. The nerves are derived from the pelvic plexus. The lymphatic drainage is into the nodes lying beside the external iliac and hypogastric blood vessels. There are intercommunications with the lymphatics of the bladder, seminal vesicles, vasa deferentia and rectum.

ANOMALIES

Of chief surgical interest are contraction of the vesical neck, valves of the posterior urethra, hypertrophy of the verumontanum, cysts of the prostate and müllerian

duct cyst. The great danger is urinary obstruction which may cause profound renal damage. The symptoms are similar to those of prostatic obstructions of later life. Treatment consists of surgical excision.

TRAUMA

Because of its protected position the prostate is rarely injured. Injury may be caused by puncture or gunshot wounds and by inexpert urethral instrumentation. In severe injury treatment consists of immediate repair to avoid urethral stricture and permanent damage to the adjacent external urinary sphincter.

INFECTIONS

Infections of the prostate comprise a large portion of the problems encountered in the practice of urology. In view of an intimate association with the urethra and the vasa deferentia, the prostate is prone to infection from either of these sources. Less commonly infection is by way of the blood stream, the lymphatics or by direct extension.

Nonspecific Infection. From a surgical standpoint, the chief importance of the nonspecific infections is their coincidence with obstructive lesions of the prostate. Any obstructive lesion predisposes to infection and,

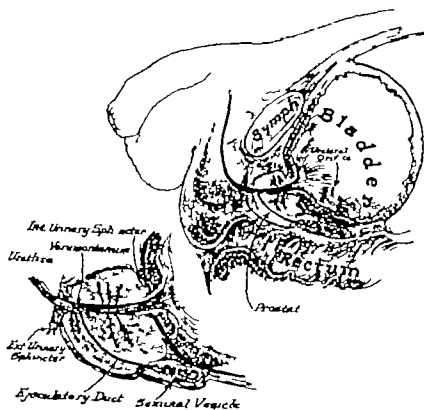


Figure 12 Anatomy and relationships of prostate.

once infection is established, it usually can not be eradicated without first removing the obstruction. Any of the common pyogenic bacteria may infect the prostate either alone or in combination. *Escherichia coli* and other gram negative bacilli are the most common invaders.

Acute prostatitis is an inflammation of the prostatic parenchyma, inevitably complicated by posterior urethritis. Unless there is co-existent obstruction, it may resolve without progression to chronic prostatitis or to abscess. In acute prostatitis frequency and urgency of urination, nocturia and dysuria may be extreme. There may be hematuria and urethral discharge. The patient may complain of perineal aching or rectal or low back pain. Systemic symptoms of fever and, occasionally chills occur. Inflammatory edema may lead to urinary retention. Digital examination reveals an exquisitely tender enlarged prostate, usually firm. Examination of an acutely inflamed prostate should be most gentle. Massage may lead to epididymitis, bacteremia and severe systemic reaction.

Of chief importance in differential diagnosis is acute urinary infection such as cystitis and pyelonephritis. The site of an acute infection is usually revealed by tenderness over the involved organ. Acute congestion of the prostate from retained secretion may lead to symptoms typical of prostatitis. The condition is due to lack of sexual intercourse or to sexual excitement which does not terminate in ejaculation. Since there is no bacterial invasion the patient is afebrile and the prostatic secretion is devoid of pus cells and bacteria.

Treatment of acute prostatitis consists of appropriate antimicrobial agents selected according to the results of microscopic and cultural studies of the urine, urethral discharge and prostatic secretion. Instrumentation other than the passage of a small urethral catheter to relieve urinary retention is contraindicated during the acute phase. Continuous catheter drainage is preferable to repeated catheterization, which is more injurious to the urethra and prostate.

Chronic prostatitis is usually either a sequela to acute prostatitis or is a complication of some prostatic obstruction. The condition may be asymptomatic. A few men complain of an aching pain or fullness in the perineum or of symptoms of mild vesical irritability. There may be a scant urethral discharge. Digital examination of the rectum may reveal a normal, boggy or an indurated prostate. Epididymitis either acute or chronic, may be a complication.

Diagnosis is established by the finding of pus cells, with or without bacteria, in the prostatic secretion. The chief error in differential diagnosis is in mistaking chronic, nonspecific urethritis for chronic prostatitis. The error is avoided by having the patient void prior to massage of the prostate. This removes any secretion from the urethra before prostatic secretion is expressed. Up to 5 per cent pus in the cellular elements of the prostatic secretion is a normal finding. Cystitis may be confused with prostatitis. Diseases of the anus and rectum may cause urinary symptoms and lead to the mistaken diagnosis of prostatitis.

Treatment of chronic nonspecific prostatitis should be directed to any contributory cause such as an obstructive process. Antimicrobial therapy is usually ineffective. Occasionally judicious prostatic massage is helpful. This should be carried out no often-er than once a week or once every two weeks with the most gentle manipulation. Frequent massage injures the prostate and induces acute infection. Massage is contraindicated in the presence of prostatic calculi. The rationale of prostatic massage is that it dislodges cellular exudates which obstruct the prostatic ducts and impede drainage. Sexual intercourse encourages drainage without injuring the prostate.

Prostatic abscess is an occasional complication of acute prostatitis. Sometimes it can be recognized as a fluctuant area on rectal examination. Such an abscess may resolve, if small, or it may rupture spontaneously into the urethra. If not, prosta-tomy becomes necessary. Symptomatic treatment consists of analgesics, hot sitz baths and bed rest. Fluids should not be forced unless an indwelling urethral catheter is in place, because frequent voiding causes irritability of the bladder and urethra.

Tuberculous prostatitis is usually secondary to tuberculosis of the kidney. The symptoms are generally overshadowed by the symptoms of involvement of the urinary tract: dysuria, frequency of urination and hematuria. On rectal examination the prostate is found to be markedly indurated. It may be stony hard, suggesting carcinoma. However in contrast to carcinoma, the condition usually occurs in the young. Differentiation is made by the finding of tubercle bacilli in the urine and the recognition of tuberculosis elsewhere in the urogenital

tract. Tuberculous prostatitis usually responds promptly to chemotherapy. Excision of the prostate and seminal vesicles is no longer indicated.

PROSTATIC ENLARGEMENTS

Since the prostate completely surrounds the posterior urethra, any abnormality of this gland is a potential source of urinary obstruction. Some pathologic conditions are more likely to produce obstruction than others. Lesions such as benign hyperplasia progress slowly. Others, such as acute prostatitis, cause only temporary obstruction and still others, such as prostatic calculi are less prone to cause obstruction. Prostatism is a clinical term which refers to the symptomatic and pathologic complex which results from any obstruction at the vesical neck. The symptoms and diagnostic measures are quite similar for the different lesions. Symptoms fall roughly into four groups:

1. *Obstructive symptoms* The symptoms of mechanical obstruction are caused by encroachment on the vesical neck and by the reaction of the bladder to obstruction. No-

turia is usually the earliest symptom and in most instances serves as a rough index of the stage of a lesion. Less noticeable but occurring with nocturia are diurnal frequency of urination and a diminution in the size and force of the urinary stream. Later there are hesitancy and dribbling on urination, urgency of urination and incontinence. Hematuria may be another symptom of obstruction. The force exercised during difficult urination incites bleeding. With the development of residual urine the patient may note a mass low in the abdomen and have a feeling of incomplete emptying of the bladder. Complete obstruction may supervene.

2. *Symptoms of superimposed infection.* When an obstruction is complicated by acute urinary infection all previous symptoms exacerbate markedly.

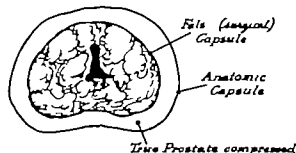
3. *Uremic symptoms* In some instances the symptoms of azotemia, secondary to hydronephrosis, are the first to call attention to a lower urinary obstruction.

4. *Sexual symptoms* Some patients with prostatic enlargement have increased potency in others, it is diminished. As a general

NORMAL PROSTATE



BENIGN PROSTATIC HYPERPLASIA



CARCINOMA OF PROSTATE

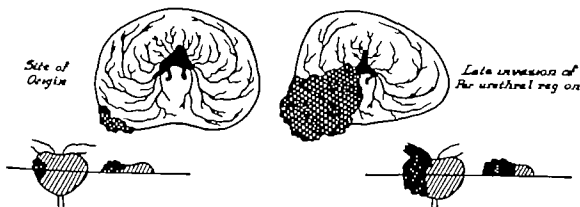


Figure 13. Site of origin of benign prostatic hyperplasia as contrasted with that of carcinoma of prostate.

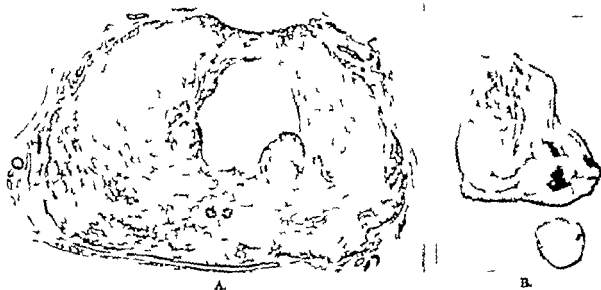


Figure 14 Surgical capsule between benign prostatic hyperplasia and compressed capsule. A, Cross section of midprostatic region showing spheroids of hyperplasia around urethra within true prostate. B, Hyperplasia enucleated from within rim of true prostatic tissue.

rule decreased potency is a more frequent accompaniment of carcinoma of the prostate than of a benign lesion.

Obstructions to the vesical neck impede the outflow of urine leading to back pressure changes in the bladder—hypertrophy of the trigone, trabeculation of the detrusor the formation of cellulæ and diverticula. During an initial stage of compensation, the capacity of the bladder becomes decreased to 250 ml. or less. Later the kidney is destroyed by hydronephrosis caused by compression of the intramural portion of the ureter by the hypertrophied bladder musculature. Later with decompensation, the bladder becomes widely distended, to 1000 ml. or more and overflow incontinence results.

As a part of the study of the patient with prostatic obstruction, kidney function tests are necessary to reveal whether there has been any impairment of renal function. Intravenous urograms are obtained, if renal function is adequate, to learn the condition of the kidneys, ureter and bladder. The after voiding film will show residual urine with out the danger of introducing infection, which accompanies catheterization.

Benign Hyperplasia of Prostate. Of all obstructions which arise from the prostate, benign hyperplasia is the most common, occurring in over 50 per cent of men past the age of fifty and 75 per cent of those past eighty years of age. Although the cause of benign hyperplasia remains in doubt, some hormonal imbalance seems to be either a predisposing or aggravating factor. In con-

tradistinction to carcinoma of the prostate, which arises from the prostatic cortex, benign hyperplasia arises from the glandular acini in the immediate vicinity of the mucosa of the prostatic urethra (Fig. 13). For this reason it is more logically referred to as periurethral adenoma than benign prostatic hyperplasia. It is not a neoplasm, but is an overgrowth of normal glandular and muscular elements. As prostatic hyperplasia progresses the true glands of the prostate are compressed peripherally. Between the two a plane of cleavage develops, known as the surgical capsule, a plane which permits the surgeon to shell out a periurethral adenoma from within the prostate at open operation (Fig. 14).

Benign prostatic hyperplasia is referred to according to the location of hyperplastic glands. Median lobe and lateral lobe enlargements are the most common (Fig. 15). On rectal examination in lateral lobe hyperplasia the gland is symmetrical and elastic throughout. In median lobe hyperplasia the findings may be confined to the vesical side and are established only by cystourethroscopy and cystourethrography. Treatment is surgical, removing the hyperplastic adenoma. Large adenomas are to be removed by open operation, small adenomas by transurethral prostatectomy. In conservative prostatectomy only enlargements within the prostate or at the vesical neck are removed (Fig. 16). The true prostatic tissue is left in place.

Median Bar. Next in frequency of occurrence of benign hyperplasia is median bar (Fig. 15). This lesion develops in a younger

age group than does the former. The lesion is a fibromuscular elevation of the posterior vesical lip which distorts the normally smooth vesicourethral outlet. The bar is separate from the prostate. Although it has been said to be secondary to prostatitis, in most cases of median bar prostatic tissue is normal. When a rim of fibrous tissue completely encircles the vesical neck, the condition is constricture of the vesical neck. This may be congenital or secondary to infection or to prostatectomy. The diagnosis of both these lesions is established by cystourethroscopy.

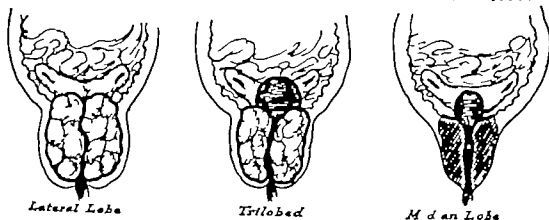
Carcinoma of Prostate. Of the prostatic neoplasms, adenocarcinoma is the most common. It has been estimated that 15 per cent of all men over fifty years of age develop cancer of the prostate. However many carcinomas are small and do not come to clinical recognition. Like most neoplasms, prostatic carcinoma has no known cause. It develops in the cortex of the prostate whereas benign hyperplasia develops immediately around the urethra (Fig. 13). Its progress is so slow that by the time it produces symptoms of obstruction to the outflow of urine, metastasis has occurred in many cases. Lesions amenable to surgical cure are those discov-

ered as incidental findings on routine rectal examination or those that occur with benign prostatic hyperplasia which leads to their discovery. The earliest sign on rectal examination is a stony hard area within the prostate. If it lies superficial to the rectal surface palpation usually reveals irregularity (Fig. 13). In advanced carcinoma the prostate becomes fixed and there is asymmetric involvement of the entire gland (Fig. 13). Typical areas of invasion are to the seminal vesicles, the base of the bladder and the membranous urethra.

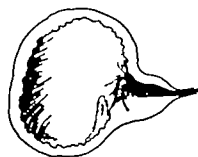
Carcinoma of the prostate spreads both by way of the lymphatics and the blood stream, the latter giving rise to osseous metastasis most frequently in the lumbosacral spine, the bony pelvis and upper portions of the femur. Osseous metastasis causes pain when the body is immobile, whereas lumbosacral pains caused by arthritis usually cease when the patient is quiet. Invasion of perineural lymphatics accounts for pain along the course of the sciatic nerves on the posterior aspects of the thighs.

A peculiar feature of prostatic tissue is the production of a large quantity of enzyme known as acid phosphatase. In the normal

BENIGN HYPERPLASIA OF THE PROSTATE



MEDIAN BAR



CARCINOMA OF PROSTATE

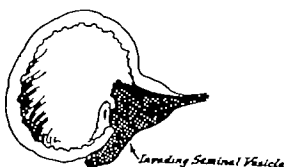
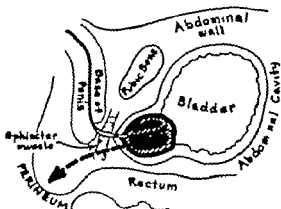
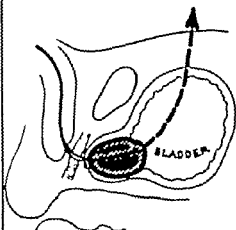


Figure 15 Common types of vesical neck obstruction.

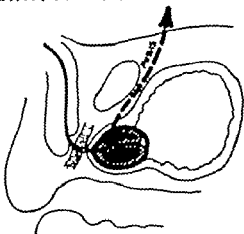
PERINEAL PROSTATECTOMY



SUPRAPUBIC PROSTATECTOMY



RETROPUBIC PROSTATECTOMY



TRANSURETHRAL RESECTION

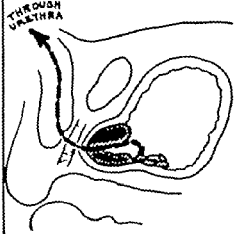


Figure 16 Methods of conservative prostatectomy

prostate most of this enzyme drains into the urethra and is carried off in the urine so that the blood level remains low. When enormous quantities are produced, blood acid phosphatase becomes elevated, indicating metastatic carcinoma of the prostate. Since the blood acid phosphatase in all metastatic carcinomas is not elevated, a negative finding does not rule out dissemination. A source of error in this test is in obtaining blood for examination within forty-eight hours of prostatic massage, a manipulation which will carry the enzyme into the blood stream from the normal prostate.

Differential diagnosis must be made from chronic prostatitis, either nonspecific or tuberculous, with fibrous tissue reaction which may simulate the stony-hard induration of carcinoma. Also areas of calcification or calculi may yield rectal findings suggestive of carcinoma. The ultimate diagnosis, particularly in early cases amenable to surgical cure, is made by biopsy. This is usually best accomplished after perineal exposure of

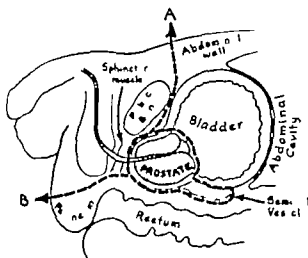
the gland when tissue is examined by frozen section. If the diagnosis proves positive, immediate prostatovesiculectomy is carried out at the same time. In advanced cases the diagnosis usually presents little difficulty. In addition to the typical rectal findings there is frequent elevation of the blood acid phosphatase and osseous metastasis. However even in advanced cases, it is important to establish the diagnosis with certainty before starting treatment. This can be done by needle biopsy through the perineum or by transurethral resection of tissue.

With respect to treatment, the only known cure is by complete surgical extirpation, i.e., total prostatovesiculectomy (Fig. 17). However the outlook in patients with advanced carcinoma of the prostate has been remarkably brightened by hormonal therapy. In 1947 Huggins made the epic making discovery that castration and/or the administration of estrogen favorably influences the course in 80 to 90 per cent of all cases. Estrogen suppresses the function of androgen-forming

cells of the testis, which promote activity of prostatic carcinoma. Although the effect of neoplastic stimulation has been abundantly proved, it has never been demonstrated that androgens initiate carcinoma of the prostate. With removal of androgen stimulation, there is a cessation or regression in many patients to such an extent that the rectal signs of carcinoma entirely recede. Likewise the blood acid phosphatase activity is suppressed and osseous metastasis recedes. Unfortunately although patients may obtain relief for periods of several months or several years, there is a tendency sooner or later for the carcinoma to escape from hormonal control, either with estrogens alone or castration alone, or with both. Apparently the use of both methods is the most advisable.

After escape of the carcinoma from hormonal control, other measures may be applied, such as administration of tremendous dosages of estrogens or the use of adrenal steroid hormones. At this stage androgens occasionally prove beneficial. Neither hypophysectomy nor adrenalectomy has proved its worth in treatment of prostatic carcinoma although both operations have been helpful in isolated instances.

When advanced carcinoma of the prostate does not yield to conservative measures, transurethral resection of obstructing tissue is indicated. It must be remembered that if there is coincident hyperplasia of the prostate, a fairly common association, treatment directed to carcinoma will not relieve the obstruction of benign hyperplasia.



"RADICAL" PROSTATECTOMY

Figure 17 Total prostatovesiculectomy for carcinoma of the prostate, removal of prostate, seminal vesicles and surrounding fascia. A, retropubic route. B, perineal route.

Sarcoma and secondary neoplasms, such as invasion of carcinoma of the rectum, are less common and usually can be treated only by palliative resection and symptomatic treatment.

Prostatic Calculi. Calculi sometimes form in the glandular acini and ducts of the prostate. With growth, they assume the contour of the units in which they lie. They may involve segments on one or both sides, or the entire gland. Large calculi impart a stony hardness to the prostate, observed on rectal examination, occasionally they yield crepitation. Diagnosis is established by demonstrat



Figure 18. X ray showing prostatic calculi which have assumed conformation of prostatic ducts.

ing their presence on x ray of the prostatic region (Fig. 18)

From a clinical standpoint calculi are of threefold significance (1) they may lead to the mistaken diagnosis of carcinoma, (2) they frequently occur in association with obstructive processes as benign hyperplasia and carcinoma of the prostate and (3) calculi themselves may cause urinary obstruction or contribute to infection and thus provide indication for operation. If they are asymptomatic and do not produce urinary obstruction or contribute to infection no treatment is indicated.

SEMINAL VESICLES

The seminal vesicles are a pair of monotubular structures located just above the prostate between the floor of the bladder and the rectum. Joined medially by the ampullae of the vasa deferentia, they unite to form the ejaculatory ducts (Fig. 1). The function of the seminal vesicles is to secrete a tenacious mucus and serve as a reservoir for spermatic fluid. Their muscular wall contracts during ejaculation to expel secretion.

The intimate relationship of the seminal vesicles to the vesical trigone, the prostate, the posterior urethra and the rectum explains why seminal vesiculitis, the condition of most surgical concern, may cause urinary frequency, dysuria and painful defecation.

ANOMALIES

Congenital anomalies of the seminal vesicles usually accompany ipsilateral maldevelopment of the upper urinary tract. They are extremely rare and of little clinical importance.

TRAUMA

Injuries of the seminal vesicles are likewise extremely rare. They are usually a complication of fracture of the bony pelvis. Treatment is the same as for trauma to the prostate.

INFECTION

Inflammation of the seminal vesicles is usually with pyogenic bacteria secondary to infection of the posterior urethra and prostate. Failure to recognize seminal vesiculitis may lead to confusion in explaining obscure symptoms of low backache, perineal discomfort and urinary irritability. Perhaps one important cause of continued difficulty from seminal vesiculitis is occlusion of the ejaculatory duct which prevents escape of infected products.

Acute seminal vesiculitis is readily diagnosed by the palpation of an enlarged tender seminal vesicle, an oblong-shaped, cystic mass above the prostate. In chronic seminal vesiculitis the seminal vesicle becomes indurated and fixed. Diagnosis can sometimes be established by stripping the seminal vesicle and recovering vesicle fluid impregnated with pus cells and bacteria. The treatment is as for prostatitis. In chronic infection, gentle stripping of the vesicles may induce resolution of the process. Occasionally in chronic infection seminal vesiculectomy becomes indicated. Since this operation may produce sexual impotence it should not be lightly considered in young men.

NEOPLASMS

Fewer than thirty cases of carcinoma of the seminal vesicles have been reported. The growth of the neoplasm is so rapid as to make it difficult to determine the site of origin. Secondary invasion of the seminal vesicles by carcinoma of the prostate by way of the perineural lymphatics is quite common. Extension is revealed by rectal palpation.

COOPER'S GLANDS

The bulbomembranous glands of Cowper are paired glands between the layers of the urogenital diaphragm which drain by ducts which empty into the floor of the bulbous urethra (Fig. 1). They provide a clear alkaline mucoid secretion which supplements the spermatic fluid. This secretion apparently alkalizes the urethral channel to prepare the way for expulsion of semen. In the normal state, Cowper's glands are not palpable. When they become infected, they enlarge and, on rectal examination, can be palpated adjacent to and lateral to the membranous urethra. Sometimes pressure over an infected gland yields pus from the urethra.

DIFFUSE ANOMALIES AFFECTING REPRODUCTIVE SYSTEM

SEXUAL IMPOTENCE

Impotence signifies the inability to perform properly the sexual act. This may be manifest by a loss of libido, by the inability to gain or to sustain an erection or by difficulties related to ejaculation. The mechanism of coitus entails a complex coordination of nervous, vascular, muscular and chemohormonal influences. When any or all of these factors are deficient, impotence may de-

velop It has been estimated that impotence affects between 25 to 35 per cent of all adult males.

In 90 to 95 per cent of men complaining of impotence the inadequacy is emotional rather than physical. The practice of with drawal as a means of preventing pregnancy is sometimes the sole cause. Psychogenic influences which lead to impotence are guilt, anxiety or jealousy or of frigidity of the female partner. Common among the neurogenic causes are tabes dorsalis, hemiplegia, multiple sclerosis and other diseases affecting the spinal cord. Damaging both the nerves and vascular supply to the penis are extensive pelvic operations as for carcinoma of the rectum or prostate. Purely vascular causes are coarctation of the abdominal aorta. A chemohormonal cause of impotence is absence of both testes, either congenital or traumatic. Atrophic testes usually provide adequate androgen to establish secondary sex characteristics and to perform coitus despite defective spermatogenesis. In Cushing's disease impotence may result from decreased gonadotropic activity and loss of androgen secretion. Bilateral orchiectomy performed after puberty some times, but not universally produces impotence. The administration of estrogens, as in the treatment of carcinoma of the prostate, almost invariably produces impotence because of the inhibitory effect on the pituitary gland. Patients with pituitary, adrenogenital syndrome do not develop secondary sex characters and are impotent. Impotence may also develop in profound systemic disorders, febrile and cachectic states.

With respect to the local causes of impotence, congestion and infection of the prostate, prostatic urethra and verumontanum tend to produce premature ejaculation, apparently because of mechanical irritation. Carcinoma of the prostate may produce impotence particularly in the advanced stages. It is obvious that almost all lesions of the penis may either cause or contribute to impotence.

In the study of a patient with impotence the examination must be sufficiently complete to differentiate between systemic, local, organic, and psychologic causes, for without accurate diagnosis, treatment cannot be intelligently administered. Cystourethroscopy should be a part of the examination to disclose any abnormality of the vesical neck or posterior urethra. Treatment is directed to the cause. In most young males this is psychiatric. Since sexual problems are

among the most difficult to treat, they usually require more skill than the physician without psychiatric training can give.

INFERTILITY

Fertility is the capacity to procreate. Infertility signifies impairment of a normal capacity to produce offspring. With complete and permanent incapacity the condition is known as sterility. Studies indicate that the male is the sole cause of infertility in 20 to 40 per cent of all barren marriages. The fertility of the male is learned through studies of the spermatic fluid, including the number of spermatozoa, the percentage of abnormal forms and their viability. If sperms are absent or defective, biopsy of the testis is indicated to differentiate between the types of intrinsic deficiency of the germ cells. This study will also reveal cases in which there is normal spermatogenesis and azospermia caused by obstruction to the conductive system.

The causes of male infertility are one or more of the following factors:

1. Deficiencies in the manufacture of germ cells. It has been estimated that 95 per cent of infertile males suffer from intrinsic spermatogenic defects. The germ cells of the seminiferous tubules may be congenitally defective; they may have been damaged by infection as in mumps orchitis or by trauma, as in destruction of the blood supply, or the defect may be chemohormonal, as in hypopituitarism. The cryptorchid testis is almost invariably deficient in spermatogenesis. Although this was previously presumed to be due to damage from increased temperature in the undescended position, the inability to restore normal spermatogenesis by orchiopexy no matter how early in life the operation is performed, suggests that the possible defect may be congenital.

2. Obstructions to the conductive system. The usual cause of this defect is bilateral stenosis due to epididymitis, usually gonococcal. Occasionally there is congenital absence of the vas deferens. This excludes acquired defects such as bilateral vasectomy.

3. The inability to perform the normal sexual act. Obviously the impotent male has difficulty in the delivery of spermatozoa, although his capacity to produce normal spermatozoa may not be impaired. Thus, the patient may be impotent yet fertile or he may be potent yet sterile.

In the clinical study of a patient complaining of inability to reproduce, a careful history should be taken of all possible eti

ologic factors such as mumps orchitis, tuberculosis, deficiency diseases, operations such as repair of inguinal hernia or orchiopepy, trauma to the genitalia and genital infections such as epididymitis. The patient should be questioned about the course of any pregnancies he has induced and about his performance of the sexual act.

Physical examination should be painstaking, seeking out any neurologic cause or endocrine dyscrasia. Careful examination should be made of the genitalia with particular attention to the prostate, seminal vesicles, vasa deferentia, epididymides and testes. In obscure cases cystourethroscopy should be carried out to seek any abnormality of the posterior urethra or verumontanum. The urine and prostatic secretion should be examined for pus cells which would indicate a complicating infection.

The most important step in the study of a patient of suspected infertility is examination of the spermatic fluid. Care should be taken in collection to avoid contamination with chemicals such as those that might be present on a condom. The best specimen is produced by masturbation following four days of abstinence from intercourse. Examination should be made within a few hours after collection to learn of the motility of the spermatozoa. In addition to a total count, the percentage of defective forms should be calculated. Testicular biopsy provides the final information concerning spermatogenesis, giving indication of the degree and type of any impairment.

The results of treatment of infertility in the male are not encouraging. General measures should be instituted to improve the general health. Frequently when infertility is mild, a vacation supplemented by rest and an adequate diet will produce the desired result. Sexual technique should be discussed with the patient. Frequently the timing of intercourse to the period of fertility in the female will prove successful. Intercourse at this time should be carried out after three or four days of celibacy since this helps increase the number and vigor of spermatozoa. In overweight men, a reducing diet should be prescribed. Vitamin D preparations help the liver to inactivate estrogens.

Of the various forms of hormonal therapy thyroid has proved most effective particularly in cases of hypothyroidism. Gonadotropins would theoretically aid the development of the seminiferous tubules, but their use has not proved successful, perhaps because preparations thus far developed are

not sufficiently powerful. Androgens are contraindicated since they depress rather than stimulate spermatogenesis. There is one exception to this rule in that the rebound following administration of testosterone has proved effective in some instances. In this method testosterone is given for three to six months, providing there is no blockage to the conductive system. Following cessation of treatment, a rebound may result in increased spermatogenesis.

Vasovasostomy in cases of previous vasoligation will result in success in 60 to 80 per cent, when the operation is performed bilaterally. Epididymovasostomy if azoospermia is caused by occlusion of the ducts in the globus minor has a very low rate of cure. This operation attempts to divert sperm from the globus minor of the epididymis to the vas deferens. Technically the operation leaves much to be desired and it may be followed by stenosis. The possibility of restoration of fertility in bilateral cryptorchism by bringing both undescended testes into the scrotum is of questionable value even if the operation is performed early in life. After puberty there is no chance of restoration of spermatogenesis.

INTERSEX STATES

These conditions produce external manifestations of both sexes to an abnormal degree. Hermaphroditism makes it difficult or impossible to determine on external inspection the sex of the patient. The entire appearance such as the breasts, distribution of hair, shape of the body and tone of the voice may suggest the sex opposite the one represented by the gonads. Deformities of the external genitalia which throw doubt upon the true sex of the individual should be recognized during childhood. Temporarily is not only disastrous to the emotional balance of the child but may contribute to omission of appropriate forms of therapy.

FEMALE PSEUDHERMAPHRODISM

This condition also known as congenital adrenogenital syndrome, is caused by prenatal hyperplasia of adrenocortical tissue. The patient has normal ovaries, uterus, fallopian tubes and vagina, however the vagina usually communicates with the urethra, which, in turn, opens at the base of an enlarged clitoris that may resemble a penis. The labia are often hypertrophied, simulating a bifid scrotum. The external appearance thus simulates perineal hypospadias with cryptorchism.

In many instances the diagnosis of the true condition rests upon abdominal exploration and biopsy of any gonadal tissue. Gonadal sex can be ascertained also by microscopic study of the cells in a skin biopsy or vaginal or buccal smear estimating the percentage of female cells which have large chromosomes. If 70 per cent or more of the cells contain this chromosome the sex is female. If less than 10 per cent are positive the sex is male. Female pseudohermaphrodites usually adjust best as females. Patients seen in infancy and treated with cortisone may achieve normal female development. If the clitoris remains large excision of this structure is to be considered, as is separation of the urethra from the vaginal tract so that both open on the perineum.

MALE PSEUDOHERMAPHRODISM

In male pseudohermaphrodisism the testicles are developed, but development of the external genitalia is incomplete. Perineal hypospadias with bifid scrotum is a form of male pseudohermaphrodisism. Strictly speaking, even mild hypospadias presents a form of intersexuality. In marked male pseudohermaphrodisism, the external genitalia may closely simulate the female type. Diagnosis is made by observing testicles in the groin, a short vagina and the male sex chromatin in skin biopsy or buccal smear or by surgical exploration. These children are reared most successfully as girls. Since the testicles are sometimes painful and provide a source of embarrassment in later life orchiectomy is indicated. Estrogen therapy is begun at puberty to accentuate the female characteristics.

TRUE HERMAPHRODISM

True hermaphrodisism is extremely rare. It may be manifest as one ovary and one testis on either or both sides or an ovotestis on either or both sides. Surgical correction is directed toward establishing the sex to which the patient seems most inclined, removing the opposite gonadal tissue. Usually the external genitalia can be converted to the one or the other sex by appropriate surgical measures.

HYPOGONADISM IN THE MALE

Since the hormones derived from the testes have little influence on development until after puberty the effects of male hypogonadism first become apparent after puberty. However causative lesions are often congenital and may be discovered in

early life. Sexual infantilism or hypogonadism is identified by a disproportionate smallness of the external genitalia in relation to the general bodily development. It may result from primary testicular hypoplasia or from destructive diseases of the anterior pituitary lobe. There is frequently associated thyroid and/or adrenal deficiency. Pituitary infantilism is commonly known as Fröhlich's syndrome. Turner's syndrome is characterized by testes of normal size, yet histologically spermatogenic cells are arrested and the penis and prostate are hypoplastic. Klinefelter's syndrome is characterized by small testes. Eunuchoidism is caused by bilateral congenital absence or hypoplasia of the testes.

SEXUAL PRECOCITY

Adrenocortical hyperplasia is the most common cause of precocious sexual development in the male. The external genitalia may be noticeably enlarged at birth and at times a mild degree of hypospadias is present. Cortical tumors of the adrenal produce a similar precocity. Since the action of the hormone is only upon the secondary sex characteristics there is a lack of simultaneous spermatogenesis.

READING REFERENCES

- Ambrose, S. S., and Shandalakis, J. E. Torsion of the Appendix Epididymis and Testis. Report of Six Episodes. *J. Urol.* 77:51-58, 1957.
- Baker, R. Management of Benign Prostatic Hypertrophy. *Postgrad. Med.* 19:168-176, 1956.
- Bergman, R. T. Scrotal Swellings. *West. J. Surg.* 66:166-168, 1958.
- Bonner, C. D., and others. Prostatic Serum Acid Phosphatase Level in Cancer of the Prostate. *J.A.M.A.* 164:1070-1076, 1957.
- Barford, C. E., and Barford, E. H. Combined Therapy for Peyronie's Disease. *J. Urol.* 78:265-268, 1957.
- Callonan, F. T., and Wilson, J. F. The Non-venereal Diseases of the Genitals. Springfield, Ill., Charles C. Thomas, 1956.
- Charny, C. W., and Wolgin, W. Cryptorchism. New York, Paul B. Hoeber, 1957.
- Creevy, A. A., and Benzler, F. S., Jr. Fracture of the Penis, Traumatic Rupture of Corpora Cavernosa. *J. Urol.* 78:620-627, 1957.
- Creevy, C. D. Hypospadias. *Postgrad. Med.* 18:86-93, 1955.
- Colston, J. A. C. Differential Diagnosis and Management of the Senile Prostate Showing Twenty Five Years of Progress. *Am. Surgeon* 21:581-589, 1955.
- Colp, O. S. Surgical Correction of Hypospadias. *J. Urol.* 79:279-292, 1958.
- Dean, A. L. Tumors of Penis, Urethra, Scrotum and Testis. In *Urology* edited by M. F. Campbell. Philadelphia, W. B. Saunders Company 1954, pp. 1177-1253.

- Ekstrom, T. and Edsmyr, F. Cancer of the Penis. *Acta chir. Scandinav.* 115:25-45 1958.
- Falkenburg, L. W., Paparo, G. P., and Liang, D. S. F. Adenomatoid Tumor of the Epididymis, A Review and Report of Two Additional Cases. *Am. J. Surg.* 94:509-512, 1957.
- Flocks, R. H. Carcinoma of the Prostate. *Postgrad. Med.* 18:106-114 1955.
- Forshall, I. and Ruckham, P. P. Transposition of the Penis and Scrotum. *Brit. J. Urol.* 29:250-252, 1956.
- Gartman, E. Torsion of the Spermathe Cord in Adult Scrotal Testes, A Review of Eighteen Cases Managed Surgically. *Am. J. Surg.* 94:787-789 1957.
- Getzoff, P. L. Clinical Evaluation of Testicular Biopsy and the Rebound Phenomenon. *Fertil. & Steril.* 6:468-474 1955.
- Gibson, T. E. Tumors of the Seminal Vesicles. In *Urology*, edited by M. F. Campbell. Philadelphia, W. B. Saunders Company 1954. pp. 1170-1176.
- Goold, W. L. Impotence. *M. Times*, 84:302-303, 1956.
- Greene, L. F. and Sutton, H. B. Occult Carcinoma of the Prostate. Clinical and Therapeutic Study of Eighty-three Cases. *J. A. M. A.* 159:1494-1498, 1955.
- Haller, J. R., Schumaker, L. B. and Furness, T. D. Congenital Absence of the Penis. *J. Urol.* 78:155-157 1957.
- Hand, J. R. Treatment of Undescended Testis and Its Complications. *J. A. M. A.* 164:1185-1191 1957.
- Henry, W. L., and others. True Hermaphroditism. *J. Nat. M. A.* 49:312-318 1957.
- Hinman, F. Jr. Interspecificity. *Pediat. Clin. North America* 4:905-924, 1957.
- Hotchkins, R. S. Infertility in the Male. In *Urology*, edited by M. F. Campbell. Philadelphia, W. B. Saunders Company 1954. pp. 733-766.
- Kunkel, R. E. Prostatitis. *Clin. Med.* 4:673-674 1957.
- Lowale, O. S. and Kirwin, T. J. *Clinical Urology*. Baltimore, Williams & Wilkins Company 1956. pp. 141-143.
- MacCollum, D. W., Longino, L. A., and Meier, L. A. Jr. Treatment of Hypospadias. *S. Clin. North America*, 36:1555-1568, 1956.
- McIntosh, H. W. Syndromes of Hypogonadism in the Male. *Postgrad. Med.* 22:103-105 1957.
- Moers, J. M. Interspecificity. *J. A. M. A.* 163:538-542, 1957.
- Nelson, W. O., and Boccabella, R. A. Application of the Sex Chromatin Test. *Fertil. & Steril.* 8:323-326, 1957.
- Obney, N. Hydroceles of the Testicle Complicating Inguinal Hernias. *Canad. M. A. J.* 5:733-736, 1956.
- O'Shaughnessy, E. J., Parrino, P. S., and White, J. D. Chronic Prostatitis—Fact or Fiction? *J. A. M. A.* 166:540-544, 1958.
- Rolnick, D., Cottrell, T. L. C., and Lloyd, F. L. Priapism. Report of Two Cases and Discussion of Treatment. *Illinois M. J.* 112:120-130, 1957.
- Schneiderman, C. Traumatic Rupture of the Testis. *J. Urol.* 78:54-57 1957.
- Scott, W. W. Carcinoma of the Prostate. In *Campbell, M. F. Principles of Urology*. Philadelphia, W. B. Saunders Company 1954. pp. 1153-1160.
- Serd, B. Management of Seminal Vesiculitis. *Illness M. J.* 118:297-300 1958.
- Smith, K. H. Torsion of the Spermathe Cord. *Brit. J. Surg.* 45:280-283, 1957.
- Stauber, W. J. and others. Management of Testicular Tumors. *J. A. M. A.* 166:751-753, 1958.
- Thompson, R. V., and Ash, J. E. Benign Hyperplasia of the Prostate Gland. In *Urology*, edited by M. F. Campbell. Philadelphia, W. B. Saunders Company 1954. pp. 1093-1124.
- Tyler, E. T., and Singher, H. O. Male Infertility—Status of Treatment, Prevention, and Current Research. *J. A. M. A.* 160:91-97 1956.

THE FEMALE REPRODUCTIVE SYSTEM

By JOHN C. BURCH M.D. ROBERT L. CHALFANT M.D.
and HORACE T. LAVELY JR. M.D.

JOHN CHRISTOPHER BURCH, a Tennessean, was educated at Vanderbilt University where he is now Professor of Gynecology. He received an extensive surgical training before pursuing his specialty of gynecology. Doctor Burch is a proponent of the view that there is a close interrelationship between abdominal surgery and gynecology and that diseases do not respect the boundaries of surgical specialties. For this reason, he believes the surgeon must be grounded in gynecology and the gynecologist in surgery.

ROBERT LEE CHALFANT received his medical education at the University of Tennessee and completed his training in obstetrics and gynecology at Vanderbilt University. He is associated in the practice and teaching of gynecology with Doctor Burch.

HORACE THOMAS LAVELY JR. graduated in medicine from the University of Pennsylvania and holds an appointment on the Gynecological Teaching Staff of Vanderbilt University.

Individualization of the Patient. This presentation of the diseases of the female reproductive system follows a clinical plan. It starts with the individualization of the patient according to her needs for her various functions. The guiding principle is the conservation of useful function. This is the cardinal rule of the physiologic approach to pelvic surgery. Its mastery is the distinguishing characteristic of those surgeons whose

patients are happy and satisfied with their treatment.

A little story illustrates this type of individualization of the patient. Three uteri are on the pathologist's table. They are all most identical. Several myomas of golf ball size distort the regularity of their contour. Each weighs about 175 gm. The first came from a young woman thirty-two years of age. This patient came to her physician for

- Ekstrom, T. and Edsmyr, F. Cancer of the Penis. *Acta chir. scandinav.* 115:25-45 1958
- Falkenburg, L. W., Paparo, G. P., and Liang, D. S. F. Adenomatoid Tumor of the Epididymis, A Review and Report of Two Additional Cases. *Am. J. Surg.* 94:509-514, 1957
- Flocks, R. H. Carcinoma of the Prostate. *Postgrad. Med.* 18:106-114 1955
- Fordhall, I. and Ruckham, P. P. Transposition of the Penis and Scrotum. *Brit. J. Urol.* 28:250-252, 1956
- Gartman, E. Torsion of the Spermathe Cord in Adult Scrotal Testes, A Review of Eighteen Cases Managed Surgically. *Am. J. Surg.* 94:787-789 1957
- Getzoff, P. L. Clinical Evaluation of Testicular Biopsy and the Rebound Phenomenon. *Fertil. & Steril.* 6:465-474 1955
- Gibson, T. E. Tumors of the Seminal Vesicles. In *Urology*, edited by M. F. Campbell. Philadelphia, W. B. Saunders Company 1954 pp. 1170-1176.
- Gould, W. L. Impotence. *M. Times*, 84:302-305 1956
- Greene, L. F. and Simon, H. B. Occult Carcinoma of the Prostate. Clinical and Therapeutic Study of Eighty three Cases. *J.A.M.A.* 158:1494-1498, 1955
- Haller, J. R., Schumaker, L. B. and Furness, T. D. Congenital Absence of the Penis. *J. Urol.* 78:155-157 1957
- Hand, J. R. Treatment of Undescended Testis and Its Complications. *J.A.M.A.* 164:1185-1191 1957
- Henry, W. L., and others. True Hermaphroditism. *J. Nat. M. A.* 49:212-218 1957
- Hinman, F. Jr. Intersexuality. *Pediat. Clin. North America* 4:905-924, 1957
- Hotchkiss, R. S. Infertility in the Male. In *Urology*, edited by M. F. Campbell. Philadelphia, W. B. Saunders Company 1954 pp. 793-766
- Kunkel, R. E. Prostatitis. *Clin. Med.* 4:973-974 1957
- Lowley, O. S. and Kirwin, T. J. *Clinical Urology*. Baltimore: Williams & Wilkins Company 1956, pp. 141-143.
- MacCollum, D. W., Longino, L. A., and Meier, L. A., Jr. Treatment of Hypospadias. *S. Clin. North America*, 36:1535-1563, 1956.
- McIntosh, H. W. Syndromes of Hypogonadism in the Male. *Postgrad. Med.* 22:103-105, 1957
- Morris, J. M. Intersexuality. *J.A.M.A.* 162:538-542, 1957
- Nelson, W. O., and Boccabella, R. A. Application of the Sex Chromatin Test. *Fertil. & Steril.* 8:337-338, 1957
- Obney, N.: Hydroceles of the Testicle Complicating Inguinal Hernias. *Canad. M. A. J.* 75:733-735, 1956.
- O'Shaghtunsey, E. J., Parrino, P. S. and White, J. D. Chronic Prostatitis—Fact or Fiction? *J.A.M.A.* 160:510-514, 1956.
- Rolnick, D., Cottrell, T. L. C., and Lloyd, F. A. Priapism. Report of Two Cases and Discussion of Treatment. *Illinois M. J.* 112:120-130, 1957
- Schnecklerman, C. Traumatic Rupture of the Testicle. *J. Urol.* 78:54-57 1957
- Scott, W. W. Carcinoma of the Prostate. In *Campbell, M. F. Principles of Urology*. Philadelphia, W. B. Saunders Company 1954, pp. 1125-1160.
- Seld, B. Management of Seminal Vesiculitis. *Illinois M. J.* 118:297-309, 1958.
- Smith, K. H. Torsion of the Spermathe Cord. *Brit. J. Surg.* 45:280-283, 1957
- Staubitz, W. J. and others. Management of Testicular Tumors. *J.A.M.A.* 160:751-758, 1958.
- Thompson, R. V., and Ash, J. E. Benign Hyperplasia of the Prostate Gland. In *Urology*, edited by M. F. Campbell. Philadelphia, W. B. Saunders Company 1954 pp. 1095-1124.
- Tyler, E. T., and Singher, H. O. Male Infertility—Status of Treatment, Prevention, and Current Research. *J.A.M.A.* 160:91-97 1956.

amount of blood is so slight as not to produce discoloration. In such circumstances, the discharge appears whitish. A leukorrheal discharge may become bloody and a bloody discharge leukorrheal.

The principal conditions causing leukorrhea are trichomoniasis, endocervicitis, neisserian infection or gonorrhea, endocrine changes, thrush and cancer. When arranged in this order they spell the key word TENETC.

In making a differential diagnosis of the cause of leukorrhea, the starting point is the history. From a good history a fairly accurate diagnosis can often be made. The first point to settle is whether the patient really does have an abnormal discharge. The appearance of a little mucus at ovulation, or a slightly increased moisture preceding or following menstruation is physiologic. If the discharge is noticeable at all times or is of sufficient amount to stain the underclothes, it must be considered abnormal. Some idea can be obtained as to the amount of the discharge by inquiring if it is necessary to wear a napkin, or by finding out how many times a day it is necessary to change the underclothes. If it is not necessary for the patient to change her underclothes, knowing the size of the spot on them may be helpful.

Circumstances surrounding the onset of the symptoms give some help. Trichomoniasis, for example, seldom occurs before puberty but is common after puberty. Moniliasis may occur at any age and is especially common during pregnancy. It occurs frequently in association with diabetes and following the use of antibiotics. It is a clinical rule to always think of the gonococcus as a cause of discharge before puberty.

TRICHOMONIASIS

The patient complains of either discharge or some form of irritation such as itching, burning or stinging. In consistency the discharge is thin and its amount is variable. The color is yellowish green, little bubbles contained within the discharge give it a foamy appearance. The odor is disagreeable and somewhat characteristic. Dyspareunia is not voluntarily mentioned by the patient but is frequent. On physical examination, the findings vary according to the severity of the infection. In the acute stage there is marked vulvitis and vaginitis. Multiple red, punctate spots occur throughout the upper vagina, especially in the fornices. When marked, these produce a characteristic ap-

pearance referred to as "strawberry vagina." Small erosions occur on the exocervix. An eccentric erosion not touching the os, is rather characteristic. Changes in the hydrogen ion concentration of the vagina are suggestive. Usually the pH reading is 5 or above using Nitrazine paper as an indicator. The diagnosis depends upon finding trichomonads in the secretion. If present, they are easily identified by placing a little of the discharge on a slide, mixing it with a drop or two of saline solution, and covering the mixture with a coverglass and examining it under a microscope.

Donné first described *Trichomonas vaginalis* in 1836. Not until quite recently was its importance as a cause of vaginitis appreciated. Recent inoculation experiments made possible by the development of pure cultures of the organism, clearly establish it as a cause of vaginitis. However there are still some who believe that the associated organisms, especially streptococci, are of greater importance in the production of symptoms. The *Trichomonas vaginalis* is a distinct species, different culturally and morphologically from the *Trichomonas buccalis* and the *Trichomonas fecalis*.

The *Trichomonas vaginalis* inhabits the male and female urogenital tracts. In the male, its habitat is the preputial sac and prostate; in the female, the bladder, urethra, urethral glands, ducts of Bartholin's glands and the vagina. Trichomonads do not invade the endocervix. There is increased evidence that sexual intercourse is a factor in the transmission. However in an individual case the explanation of the infection is often difficult. Formerly a good deal of attention was directed toward the rectum and rectal contamination was thought to be one of the chief factors in transmission. Recent studies do not support this view. Contaminations from infected instruments, bath water, douche nozzles, towels and hands are of course possibilities. Many women harbor the organisms and yet never have any of the symptoms of vaginitis. However if the biochemical balance of the carrier's vagina becomes disturbed, infection may result. Alkaline douches, profuse alkaline cervical discharges and infections which disturb the normal vaginal flora are all precipitating causes.

In the treatment of trichomoniasis the first step is to restore the normal acidity of the vagina. This is accomplished by reducing the amount of alkaline discharge from the cervix and by increasing the acidity of the

a premarital examination. The tumor was found and the uterus removed. The next was from a woman, aged forty-two years, who was the mother of four children. She was anemic from excessive menstruation. The last was found in a woman of sixty-five years who sought examination because of a fear of cancer. She had no symptoms.

In the first woman, the useful function of reproduction could have been conserved by myomectomy or perhaps, simply by observation. As a result of the loss of this function, the operation has changed the course of her life from normal to abnormal. To her the operation was a tragedy. The second patient probably had no further use for her reproductive functions. She had a disabling condition and to her the operation is a blessing. In the third woman, there is little justification for the operation because small, asymptomatic fibroids in a postmenopausal woman generally cause no trouble. Their malignant potential is hardly as great as the mortality of the operation.

Now each of these women had an obviously diseased uterus—of sufficient degree perhaps to satisfy an exacting tissue committee. Yet one operation was an unnecessary mutilation, another just plain unnecessary and the third, thoroughly justified. The story proves that the surgeon must select a plan of treatment which is suitable for the pathologic process and for the needs of a particular woman. Now how is this done? Is there a method of reasoning which can be applied to all problems involving the female reproductive system?

There is such a method and in its simplest form it consists of four steps.

1. Consider the pathologic condition in relation to its effect on function. Function is a broad term and includes specifically, the general functions of life and health (both mental and physical) and the pelvic functions of sex, reproduction, ovarian activity, menstruation and support.

2. Define the needs of the particular patient for each of these functions.

3. Determine the effect of all proposed forms of treatment in terms of these functions.

4. Lastly put the data together and make the best possible deal, in regard to the pathologic process, for the particular patient.

Those who use this approach study disease from the functional standpoint. In that which follows the effect of the pathologic condition on function is emphasized so that it can be compared with the effects of the

various forms of treatment. In studying patients, attention should be given to the need of the individual patient for her functions.

Clinical Approach to Gynecology is the diseases of the female reproductive system, there are three main symptoms and one outstanding physical sign. Discharge, pain and bleeding are the symptoms. The presence of a mass is the physical sign. When a symptom or sign is encountered, the first step in diagnosis is the enumeration of its possible causes. To illustrate this process, consider abnormal vaginal bleeding. The five common causes of this bleeding are cancer, abortion, fibroid tumor, ectopic pregnancy and endocrine dysfunction. The first letter of these disorders are C-A-F-E-E. Although the word "cafee" is not in the dictionary it is nevertheless useful as a key word or mnemonic. The student thinks of a bleeding problem as a "cafee" problem and this immediately sets up the differential diagnosis. A good many hundreds of students have used the "cafee" scheme in the diagnosis of abnormal vaginal bleeding, and it has been so helpful and practical that key words and a key sentence are now used for discharge, pain, bleeding and mass. The key sentence is "TENETC COPS CAFE PREMIO". True the sentence is a little odd, but its oddness makes an impression which serves to better stamp it on the memory.

DISCHARGE

Leukorrhea means any whitish discharge from the female genitalia. It is commonly known among women as the whites. Normally there is a secretion from the uterus, the vagina and Bartholin's glands, but these secretions are not enough to call the attention of the patient to their presence. Under conditions of sexual excitement, the secretions may be marked in amount just before and immediately following the menstrual period, a slight increase in secretions may be noticed. This is physiologic. The whitish discharge may contain a small amount of blood and this gives it a brownish character. In conditions producing a bloody discharge there may be periods in which the

Translated, TENETC means trichomonads, endocervicitis, neisserian infection, endocrine disorder, thrombosis and cancer. COPS refers to pain of cervical, organic, peritoneal and somatic origin. CAFE represents bleeding due to cancer, abortion, fibroid tumor, ectopic pregnancy and endocrine causes. PREMIO is the mnemonic for masses. These are pregnancy, retroversion, enlargement, undiagnosed, myoma, inflammatory disease, ectopic pregnancy and ovarian masses.

as the discharge becomes less, the squamous epithelium pushes back toward the os and blocks off the openings of the glands with the subsequent formation of the familiar nabothian cysts of the cervix. A flare-up of the endocervicitis leads to a renewal of the process and the erosion cycle repeats itself many times. The histologic pictures in such a cervix are often bizarre. The pathologist must exercise the greatest care in differentiating them from early intraepithelial carcinomatous changes. One of the important features of an erosion is that the histologic changes may be very significant in the subsequent development of cancer.

An area of erosion is often seen in virgins and is congenital in origin. In the normal process of development, the squamous and columnar epithelium meet at the external os, but sometimes the squamocolumnar junction is well beyond the os. The chance of cancer development in congenital erosion is less than in acquired erosion.

A cervical laceration of some degree always follows childbirth. Inevitably infection occurs in the fresh laceration and healing is by granulation. A well healed laceration is of little significance. Persistence of the endocervicitis, however, causes poor healing and erosion. A widening of the os allows the endocervix to roll out on the external surface; this is ectropion. It occurs frequently in association with large bilateral tears. The importance of lacerations is in their causal relationship to endocervicitis and to erosion of the cervix. A laceration has the same clinical significance as endocervicitis and erosion unless it involves the internal os and so predisposes to abortion.

Stricture of the cervix is not uncommon in endocervicitis. It may result from the disease, or scarring from treatment by cauterization, conization or radium application. It causes insufficient cervical drainage and the stasis within the canal inevitably leads to infection.

Leukorrhea is the principal symptom in chronic endocervicitis. Its characteristics vary according to the stage of the disease and the amount of infection. It bears a similarity to the nasal discharge of the common cold. First, the discharge is watery then thick and purulent and, lastly clear with a high degree of mucus. When the concentration of mucus is high, the discharge is viscous. It emerges from the vaginal orifice, not as an even flow but at irregular times in the form of masses or globs. The degree to which it is colored yellow depends upon

the amount of pus. Generally the discharge is nonirritating. However as previously noted, its alkalinity changes the biochemical composition of the vagina and predisposes to trichomoniasis. In mixed infections in which *Trichomonas* is one of the organisms responsible the discharge has the irritating characteristics of that due to trichomoniasis.

Dyspareunia and pain on manipulation of the uterus occur if the infection extends into the parametrial tissues. Unless this extension takes place, local symptoms such as low abdominal pain and low backache are more often coincidental to rather than caused by the endocervicitis. Extragenital symptoms which might be related to a focal infection are not generally relieved by the treatment of endocervicitis except in the cases of systemic gonorrhea and chronic posterior urethritis. Many urologists believe that endocervicitis has a causal relationship to chronic posterior urethritis.

In treating endocervicitis it is necessary to remember that the endocervix has two important physiologic functions. Its mucus serves as a medium for migration of the sperm and its sphincteric action prevents the premature onset of labor. If the sphincter is destroyed by high amputation, a condition is produced which prohibits a woman from carrying a pregnancy to full term. If the source of a normal amount of cervical mucus is destroyed, an impediment to conception occurs.

As in most conditions the treatment of endocervicitis is both medical and surgical. The specific endocervicitis of gonorrheal origin responds dramatically to penicillin therapy. Dill has recently described an interesting nonsurgical method for the treatment of nonspecific endocervicitis. At the time of ovulation, the patient is started on a regimen of 1 mg. of stilbestrol daily. This increases the mucous flow from the cervix. At the same time, 1 gm. of sulfadiazine is given three times a day. This treatment is continued for fifteen days.

The surgical treatment of endocervicitis consists of a variety of procedures, ranging from simple cauterization to the use of total hysterectomy in exceptional cases. Local treatment is contraindicated when the disease is acute, in the presence of chronic gonorrhea, and until the possibility of cancer is excluded. In treating endocervicitis, it is necessary to remember that the associated erosion is a result of the endocervicitis and it cannot be cured permanently until the endocervicitis is cured. Generally this is best

vagina. One of the most commonly employed methods of increasing the acidity of the vagina is a mild acid douche. Vinegar in the proportion of 1 ounce to a quart of water is often used. Perhaps 1 teaspoonful of lactic acid to a quart of water is preferable. Most of the suppositories now in use contain lactose which is readily converted to lactic acid by the Doderlein bacillus.

Among the various trichomonacides none is absolutely specific. Perhaps the most commonly used is Flóraquin, which contains diodoquin, boric acid and carbohydrates. One suppository inserted night and morning, with the use of an occasional acid douche is a very satisfactory form of treatment. Devegan contains acetarsone, boric acid and hydrolyzed carbohydrates. The recommended dose is two to four tablets inserted daily. Sulfa creams have a wide range of applicability and are fairly effective. Picric acid preparations in the form of suppositories of silver picrate have found favor with many clinicians. Milibis is the newest of the trichomonacides and, while expensive, it seems to be highly potent. Vioform is a very useful and well tolerated preparation. All of the trichomonacides must be used for a period of several weeks and should be continued during the menstrual period. The oral administration of Trithion, one tablet three times daily for ten days has been helpful in the resistant cases.

Recurrences are common and should stimulate a search for the focus of infection. Most commonly this focus is in the paraurethral glands, the bladder or in the urogenital tract of the husband. The best treatment for the infected paraurethral glands is to destroy them by electrocoagulation.

ENDOCERVICITIS

Cervicitis can involve the entire cervix, the endocervix or the external cervix. Endocervicitis is the important lesion, however, because it serves to maintain the disease on the external cervix. Since the etiologic basis of all three types of cervicitis is the same and since the endocervicitis is usually the solution to the problem of discharge, it seems appropriate to emphasize this by considering the other forms of cervicitis largely as subsidiary diseases.

Endocervicitis may be caused by any of the pathogenic bacteria which may be in the vagina, childbirth infections with or without lacerations, secondary infections occurring after cervical trauma from any cause such as surgery or the insertion of a stem pessary.

gonorrheal infection, syphilis, tuberculosis.

Acute Endocervicitis. In the acute stage of endocervicitis, the tissues are red and edematous. Microscopic examination shows the familiar picture of acute inflammation. The discharge causes maceration and degeneration of squamous epithelium around the external os. In endocervicitis following childbirth, there is danger of a lateral spread to the parametrium by way of the lymphatics. Ultimately such infection can spread to the pelvic peritoneum, tubes and ovaries. In gonorrheal infection the spread is upward to the tubes. Syphilis of the cervix is largely a disease of the exocervix. The cervix is often the site of the primary lesion. However, a typical chancre rarely occurs. Usually the lesion is insignificant and frequently missed. Secondary lesions of the cervix are frequent. The ulcers are grayish white with a yellow base. If there is a zone of normal tissue between the os and the cervix, it adds to the suspicion. Tuberculosis of the cervix is rare, but when it occurs infection is almost always present in the endometrium and the tubes.

Usually the symptoms of acute endocervicitis are minimal, except when they are overshadowed by the symptoms of the spread of the disease. Then they may be violent, but most of the time there is only mild pelvic discomfort, fever, low backache and some malaise. There is always a purulent mucoid discharge in variable amounts.

The diagnosis is easy and is made by finding the signs of acute inflammation. Especially characteristic is pointing of the cervix and the presence of an acute mucopurulent discharge. Unless there is another obvious cause of the infection, gonorrhea is always a possibility. To overlook it invites a crippling spread of the disease to the tubes.

Since the advent of the antibiotics, treatment is easy and effective. Penicillin is the agent of choice.

Chronic Endocervicitis. In chronic endocervicitis there is often a red, granular appearance of the tissue surrounding the external os. This is an erosion and it is a significant lesion. The alkaline secretions from the site of chronic endocervicitis bathe the epithelium surrounding the external os. Maceration of the squamous epithelium results and this allows the columnar epithelium of the endocervix to replace the squamous epithelium of the exocervix. The endocervical epithelium in the area of erosion forms glands and, in the course of time,

the vulva, dysuria and slight leukorrhea are the principal symptoms. These are not uncommon among women and may disappear in three to four days. Many women ignore these symptoms. Others seek advice. When they do, it is important for the physician to make the diagnosis before the disease spreads to the tubes. This occurs most generally at the time of the period.

In the acute phase, the vulva is hyperemic. Swelling and edema of the urethral mucous membrane everts the tissue so that the orifice appears red and pouting. The periurethral glands are red and prominent. The ducts of Bartholin's glands appear as red macules (Sänger's macules). A thick, mucopurulent discharge exudes from the inflamed cervix. Palpation of the urethra may reveal thickening. Stripping the urethra from above downward expresses pus from the urethral orifice. Closer inspection reveals infection in the glands on the floor of the urethra. The ducts of the two largest glands (Skene's glands) open just inside the orifice. Infection in Bartholin's glands causes swelling and tenderness. A normal Bartholin gland is hard to palpate, but the infected gland is thickened and hard. When abscess is present, the pain is intense and there is marked swelling and tenderness of the affected side of the vulva. The cervical os is reddened and congested, and this redness and congestion blends gradually into the normal cervical tissue. The exudate is thick and purulent. Tenderness on manipulation may be present and variable in degree.

As the infection subsides, the disease becomes chronic. The urethra may be permanently affected and some describe the feel of the chronically infected Skene's glands as bristle-like. Dilation of the urethral orifice affords visualization of their reddened openings. Both Bartholin's glands may be enlarged. Cysts of the glands are common. The redness around the cervical os subsides, but frequently evidence of infection persists in the form of a mucopurulent discharge from the cervix.

The absolute diagnosis of gonorrhea is possible only by identifying the organism from smears and cultures. Even with this method, it is oftentimes impossible to find the organism in patients with known cases. In a recent series of 538 women named as sex contacts by men with acute gonorrhea it was possible to confirm the diagnosis by smear and culture in only 20.6 per cent. A presumptive diagnosis is warranted following exposure if the clinical picture of acute

or chronic gonorrhea develops. It is almost certain if there has been salpingitis.

Success in the treatment of gonorrhea in the female depends upon eliminating infection in the sexual partner. To do this often requires extreme tact. Gonorrhea responds to the sulfas and antibiotics. In the adult, the dosage of sulfadiazine is 1 gm given four times a day for five days. Penicillin, Aureomycin, Terramycin and streptomycin are effective. Of these penicillin is generally preferred. A single dose of 300 000 units given intramuscularly for three days, or Aureomycin or Terramycin, 250 mg., given orally four times a day for five days usually effects a cure. All forms of therapy should be repeated until the patient is asymptomatic. The criterion for cure is to obtain three negative smears and cultures, taken at weekly intervals. In children over ten years of age, penicillin therapy is the same as for adults. For younger children the dosage may be reduced proportionately.

Abscesses will not respond to the above therapy and must be incised. The most common abscess is that of Bartholin's gland. In opening these abscesses, it is best to make the incision just inside the mucocutaneous border. In a cyst of Bartholin's gland, excision or marsupialization is generally necessary. To the uninitiated it is tempting to carry out the treatment in the office with the patient under local anesthesia. Of course this can be done, but it is far better to do it in the operating room with the patient under general anesthesia as the large venous sinuses of the vulva bleed copiously. Infected Skene's glands and paraurethral crypts are easily destroyed by inserting a small wire and touching it with the electrocoagulating unit. An ordinary cautery serves nicely if the duct will admit a probe.

ENDOCRINE DISORDERS

Endocrine Vaginitis (Senile Vaginitis). Estrogen causes marked changes in the vaginal mucosa by increasing the blood supply and stimulating the epithelium to an orderly proliferation and cornification. As a result the vagina becomes resistant to infection and trauma. When the estrogen secretion ceases, the epithelium becomes thin and prone to infection. Frequently little petechial-like areas with superficial ulceration form. They resemble somewhat the strawberry lesions in *Trichomonas vaginalis* vaginitis. Scrubbing the vagina, as in preoperative preparation, makes them especially prominent. In the course of time, marked

accomplished by surgical means. There are five methods for the removal of the endocervix: cauterization, electrocoagulation, high frequency conization, surgical conization of the endocervix, cervical amputation.

The nasal-tip cautery is excellent for destroying the infected endocervix and the associated erosion. After exposing and cleansing the endocervix, the hot cautery incises the mucous membrane from the internal os to the external os and continues out over the erosion to the normal mucous membrane. Several such incisions complete the operation on each lip of the cervix. When nabothian cysts occur the cautery punctures them. Too vigorous treatment causes pain and as the tissues slough annoying bleeding can occur. Strictures may form from the agglutination of opposing raw surfaces. For these reasons it is best to carry out the procedure in stages, allowing the incisions in one lip to heal before making new ones opposite them.

Electrocoagulation overcomes many of the disadvantages of the cautery. The construction of the endocervical electrode prevents any damage to the opposite side of the canal. This is important in avoiding strictures. If the individual treatments cover less than half the circumference of the canal strictures cannot form. After cessation of the endocervical discharge the erosion will heal when cauterized.

The high-frequency cutting current excises the endocervix in a thorough fashion. A special electrode removes the infected endocervix and the erosion in one cone-shaped piece. The wound remains open and healing occurs by granulation. Bleeding, which sometimes is annoying, occurs during the healing period in a small percentage of patients. Often strictures form unless prevented by dilation. A plastic procedure similar to the Sturmdorf operation, overcomes some of the disadvantages of the method. Hemiconization at intervals of six weeks is simple and is a more practical procedure for office use. It should be done only by those experienced in the method.

A scalpel can remove as much tissue as does the conization tip. Sturmdorf's technique is best. With a tenaculum grasping the anterior and posterior lips of the cervix within the area of erosion, the incision circumscribes the eroded area and dissects back the vaginocervical mucous membrane for a distance of 2 to 3 cm. Next, the scalpel comes out the entire gland-bearing area of the endocervix. Care is taken to preserve the

musculature. Coning becomes an amputation when too much muscle is removed. Failure occurs when infected glands are left in situ. Finally a double, inverting suture on each lip pulls the loose vaginocervical cut into the cavity and covers most of the raw area. The Sturmdorf operation can and does give excellent results. However it can be difficult and it is not as popular now as it was in the past.

Amputation of the cervix in the treatment of uncomplicated endocervicitis is rare nowadays, as it predisposes to abortion and cervical dystocia. It is mainly reserved for the removal of the prolapsed cervix as part of the Manchester operation.

In very exceptional cases, total hysterectomy is the solution to the problem. Such patients always have other indications for hysterectomy in addition to their endocervicitis.

NEISSERIAN INFECTION (GONORRHEA)

Before the days of the antibiotics, gonorrhea caused more suffering and disability among women than did any other disease. Today the disease occurs much less frequently. Coitus transmits the infection and the customary incubation period is three to eight days. In adult females the gonococcus attacks the urethra, periurethral glands, Skene's glands, Bartholin's glands and the cervix. The discharge is purulent and in the acute stage may be loaded with organisms. Abscesses may form in Bartholin's glands. Later, after subsidence or rupture, the gland becomes either fibrosed or cystic. The cervix and urethra are the foci from which the infection spreads. Cystitis is common, but pyelitis is rare. The endometrium readily overcomes the organism, but the tubes usually succumb to the infection with all of the distressing sequelae of salpingitis.

In female children, the disease is very contagious and, until recent times, it was present in most institutions for their care. The vaginal mucous membrane of a child does not resist the infection. Resistance is linked to the changes produced in the mucous membrane by the ovarian hormones at the menarche. In children, vaginitis and cervicitis are the common lesions. Salpingitis rarely occurs. The disease becomes chronic with alternating periods of exacerbation and remission. It finally disappears at the menarche.

Lower genital tract gonorrhea is insidious because the symptoms are few and mild. A little temporary itching, a mild irritation of

the vulva, dysuria and slight leukorrhea are the principal symptoms. These are not uncommon among women and may disappear in three to four days. Many women ignore these symptoms. Others seek advice. When they do, it is important for the physician to make the diagnosis before the disease spreads to the tubes. This occurs most generally at the time of the period.

In the acute phase the vulva is hyperemic. Swelling and edema of the urethral mucous membrane everts the tissue so that the orifice appears red and pouting. The periurethral glands are red and prominent. The ducts of Bartholin's glands appear as red macules (Sanger's macules). A thick, mucopurulent discharge exudes from the inflamed cervix. Palpation of the urethra may reveal thickening. Stripping the urethra from above downward expresses pus from the urethral orifice. Closer inspection reveals infection in the glands on the floor of the urethra. The ducts of the two largest glands (Skene's glands) open just inside the orifice. Infection in Bartholin's glands causes swelling and tenderness. A normal Bartholin gland is hard to palpate, but the infected gland is thickened and hard. When abscess is present, the pain is intense and there is marked swelling and tenderness of the affected side of the vulva. The cervical os is reddened and congested, and this redness and congestion blends gradually into the normal cervical tissue. The exudate is thick and purulent. Tenderness on manipulation may be present and variable in degree.

As the infection subsides, the disease becomes chronic. The urethra may be permanently affected and some describe the feel of the chronically infected Skene's glands as bristle-like. Dilation of the urethral orifice affords visualization of their reddened openings. Both Bartholin's glands may be enlarged. Cysts of the glands are common. The redness around the cervical os subsides, but frequently evidence of infection persists in the form of a mucopurulent discharge from the cervix.

The absolute diagnosis of gonorrhea is possible only by identifying the organism from smears and cultures. Even with this method, it is oftentimes impossible to find the organism in patients with known cases. In a recent series of 538 women named as sex contacts by men with acute gonorrhea it was possible to confirm the diagnosis by smear and culture in only 20.6 per cent. A presumptive diagnosis is warranted follow-

ing if the clinical picture of acute

or chronic gonorrhea develops. It is almost certain if there has been salpingitis.

Success in the treatment of gonorrhea in the female depends upon eliminating infection in the sexual partner. To do this often requires extreme tact. Gonorrhea responds to the sulfas and antibiotics. In the adult the dosage of sulfadiazine is 1 gm. given four times a day for five days. Penicillin, Aureomycin, Terramycin and streptomycin are effective. Of these penicillin is generally preferred. A single dose of 300,000 units given intramuscularly for three days or Aureomycin or Terramycin 250 mg., given orally four times a day for five days usually effects a cure. All forms of therapy should be repeated until the patient is asymptomatic. The criterion for cure is to obtain three negative smears and cultures taken at weekly intervals. In children over ten years of age penicillin therapy is the same as for adults. For younger children the dosage may be reduced proportionately.

Abscesses will not respond to the above therapy and must be incised. The most common abscess is that of Bartholin's gland. In opening these abscesses, it is best to make the incision just inside the mucocutaneous border. In a cyst of Bartholin's gland, excision or marsupialization is generally necessary. To the uninitiated it is tempting to carry out the treatment in the office with the patient under local anesthesia. Of course this can be done, but it is far better to do it in the operating room with the patient under general anesthesia, as the large venous sinuses of the vulva bleed copiously. Infected Skene's glands and paraurethral crypts are easily destroyed by inserting a small wire and touching it with the electrocoagulating unit. An ordinary cautery serves nicely if the duct will admit a probe.

ENDOCRINE DISORDERS

Endocrine Vaginitis (Senile Vaginitis). Estrogen causes marked changes in the vaginal mucosa by increasing the blood supply and stimulating the epithelium to an orderly proliferation and cornification. As a result the vagina becomes resistant to infection and trauma. When the estrogen secretion ceases, the epithelium becomes thin and prone to infection. Frequently little petechia-like areas with superficial ulceration form. They resemble somewhat the strawberry lesions in *Trichomonas vaginalis* vaginitis. Scrubbing the vagina, as in preoperative preparation, makes them especially prominent. In the course of time marked

atrophy and contraction may occur and sometimes the anterior and posterior walls agglutinate causing complete or partial obliteration of the vagina. The discharge is serous in character and most often brownish in color. Slight bleeding occurs and this must be differentiated from that due to the other causes of postmenopausal bleeding. Burning is common and mild itching not infrequent. Dyspareunia is frequent. The epithelium is thin and flat and the rugae are atrophic to the point of nonrecognition.

Even the severely atrophic mucous membrane responds to estrogen therapy. The dosage required is 0.5 mg. of conjugated estrogens given once daily by mouth, for three to four weeks. Sometimes this treatment confuses the picture by causing bleeding. For this reason, vaginal suppositories are preferable. The dosage for suppositories is 2000 I U estrogens or 0.5 mg. of stilbestrol or benzestrol given daily for a week or ten days. Estrogen in this amount inserted once a week provides a satisfactory maintenance dose. Suppositories such as Flor aquin or Devegan containing a carbolydrate aid in re-establishing the normal bacterial flora.

Endocrine-Cervical Hypersecretion. Estrogen causes increased secretion from the endocervix and this occurs normally at the time of ovulation. In ovarian failure there are derangements in ovarian function, causing a continuous high level estrogen secretion. The discharge is colorless, consists of thin mucus and has no odor. Usually it is fluid in consistency but may be a little stringy at times. It is nonirritating. Many times an associated *Trichomonas vaginalis* infection masks the character of the discharge. Symptoms are few, the main ones being a sense of wetness and the presence of a discharge on the clothing. Unless the stimulus to an abnormal estrogenic secretion is caused by therapeutic agents, medical treatment is unsatisfactory. To obtain satisfactory cure, a partial obliteration of the hypersecreting glands in the endocervix is required.

THRUSH INFECTIONS

Thrush is a mycotic vaginitis. The responsible organism is usually *Candida albicans*. Pruritus is the chief complaint of most women with thrush. Oftentimes it is distressing to the point of distraction. The patient is in real misery. Dyspareunia and dysuria are frequently present but are rarely mentioned by the patient. The discharge is variable in amount, being scanty more often

than profuse. It is thick in consistency and tends to adhere to the vulva and comes away like tiny bits of cottage cheese. Sometimes it is described as buttermilk like. The disease can occur at any age and in all classes of women, but it is especially common in the pregnant woman, the diabetic and in those who have recently received antibiotics. In the individual patient, it is practically impossible to state the method of transmission. *Candida albicans* is isolated so frequently from the mouth, skin and stool that infection from these sources is always a possibility. It can occur from coitus or from contamination of the hands, douche nozzles or instruments. Many women who harbor the organisms are asymptomatic. In these women symptoms develop only when there is a change in hydrogen ion concentration in the vagina resulting in a shift to the acid side. A high intake of sugar, use of acid douches and acid contraceptive jellies and destruction of the normal vaginal flora by antibiotics are all predisposing factors. In severe cases, the entire vulva becomes fiery red. Such an appearance is highly suggestive of underlying diabetes. Lesser degrees of vulvitis are the rule. The vagina is inflamed and contains patches of thickened, whitish exudate. In chronic infections, the mucous membrane of the vulva is thin, dry and glazed. The skin around the vulva is thickened and whitish in appearance. The hydrogen ion concentration tends to the acid side around 4.5 or lower. Finding the organisms confirms the diagnosis. For this purpose, wet smears are satisfactory. A drop of the discharge is placed on a slide and diluted with two or three drops of 10 per cent sodium hydroxide solution; on examination the organisms are readily found if present.

The first consideration in the treatment of thrush is to eliminate the possibility of diabetes, either frank or latent. It is amazing how many sufferers from this type of infection have been using vinegar douches. These of course should be eliminated. The reliable treatment is to swab the vagina with gentian violet. It is supplied in 1 to 2 per cent aqueous solution, but even in such concentration chemical irritation is not unusual. The treatment should not be repeated for three or four days. The newer preparation of Gentia-Jel or Gentersal has much to offer to those who rely upon gentian violet. Mild iodine douches have found great favor with many practitioners. 8 cc. or 2 tea spoonfuls to half a gallon water is the

usual prescription. The simplest and easiest form of treatment is the instillation of a mixture of 1 ounce of sodium borate and 4 ounces of glycerin. Two teaspoonfuls of this mixture are instilled into the vagina with a bulb syringe every other night. Most cases will respond to this form of treatment. Propion Gel is an effective agent as an alternative treatment. The most recent therapy is the intravaginal use of the antifungal antibiotic Mycostatin. One or two tablets inserted daily for seven to fourteen days produce excellent results.

CANCER

Discharge is an early symptom of all forms of genital cancer. At first the discharge is represented simply by a slight increase over the normal secretions and generally the patient overlooks it. When the surface epithelium over the growth sloughs, there is an abrasion and from this exudes a serous discharge. Infection adds to the normal weeping of the tissue. As the growth opens up the smaller blood vessels the discharge becomes brownish. Frank bleeding occurs as larger vessels are involved. Contact bleeding from examination, coitus, douches or downward displacement of the cervix against the posterior vaginal wall, as in straining, is common at this stage. As the process continues, the mass of sloughing and devitalized tissue increases. The vaginal bacteria attack it and the discharges become putrid. Cancer of the cervix produces a discharge which often follows such a pattern. In cancer of the body of the uterus, the discharge usually remains serosanguineous and relatively nonodorous for a rather long time. A clear continuous serous discharge from the cervix in a postmenopausal woman always arouses suspicion of cancer of the fallopian tubes.

PAIN

In order to interpret painful symptoms arising from the female reproductive tract, one must understand the mechanisms of pelvic pain. Much ill-advised surgery has been due to a lack of this understanding. For example, one sixth of all women entering a large clinic had pelvic pain. One-fourth of these had normal pelvic organs and the pelvic pain was not relieved by surgery. All too often the pain is attributed to the ovary. One investigator checked 1320 ovaries removed because of pain and found 993 to be normal.

The interpretation of pelvic pain also requires an appreciation of many varying factors. First there is its inconstant nature. Then the threshold for pain varies with different individuals and at different times in the same person. Much of this variation is due to the emotional reaction of the individual. Another difficulty is the various descriptions which patients give of their pain and its characteristics. A good history should include the usual information as to onset, duration, location and quality of the pain and, in addition, should note the relationship of the pain to such functions as menstruation, defecation, urination, coitus and ovulation.

Some understanding of the major causes of pelvic pain is essential to accurate history taking and examination. For this purpose four different types of pain should be distinguished according to the site of origin. These are cerebral, organic, peritoneal and somatic. The mnemonic for pain, therefore, is COPS. In using this classification, it should be clearly understood that mixtures of the various types of pain are common. In many patients with organic disease there is a cerebral component. In inflammation of an organ, the peritoneal component may be prominent and at times completely dominant. Understanding comes only when we are able to recognize and evaluate the various components.

CEREBRAL PAIN

Pain in the pelvis is often the result of an emotional conflict. It is common but difficult to interpret. Some psychiatrists believe it may be associated with the presence or absence of some kind of sexual activity, either imaginary or actual. Most of the patients with pain due to emotions also have easily recognized neurotic symptoms. Many times there is a tendency to use the symptoms as an evasion mechanism in a particular situation. Fatigability, palpitation, depression, insomnia, poor appetite and irritability are present either singly or in combination. The causative mechanism of pelvic pain of emotional origin is generally considered to be the result of a contraction of blood vessels in the affected part.

ORGANIC PAIN

In general, there is nothing specifically characteristic about pain from the various pelvic organs. Perhaps the best example of organic pain is the labor pain, but the cir-

circumstances under which it occurs and its time-intensity relationship are its characteristic aspects, rather than its quality.

Pure visceral pain unassociated with peritonitis, occurs either as poorly localized pain in the region of the diseased viscus or as a more sharply localized pain in some area of the body surface supplied by the same neural segment. When the pain stimulus is severe adjacent neural segments may be involved. Injection of procaine in the zone of reference will sometimes abolish the pain when the stimulus is mild, but if the stimulus increases this procedure becomes ineffective.

Observations made during operation with the patient under local anesthesia show that the wall of the uterus is rather insensitive. A uterine incision causes little pain. The endometrium is sensitive especially so in the region just above the internal os. The fallopian tubes are always very sensitive.

Neurosurgical operations for the relief of pelvic pain show the importance of the sacral parasympathetics. Interruption of the sympathetic pathways by resecting the superior hypogastric plexus (*presacral neurectomy*) relieves uterine pain but has little effect on cervical or bladder pain. Pain impulses from the uterus enter the cord from the twelfth thoracic vertebra to the second lumbar vertebra. The zone of reference is the suprapubic region and the lower back.

The fallopian tubes are always very sensitive. Pain from the inner third of the tube utilizes the uterine pathway while that from the outer two-thirds utilizes the ovarian pathway which runs with the ovarian vessels to the aortic plexus. Impulses from the ovary enter the cord over a broad area, from the tenth thoracic vertebra to the first lumbar. Their ordinary zone of reference is the suprapubic region, the loin and thigh. The ovaries are practically insensitive. Pricking them with a needle and handling them with a forceps are almost painless. Distending the capsule of the ovary by injecting saline solution causes only mild pain. Twisting of an elongated mesovarium causes severe pain, referred to the umbilical or subumbilical region.

Clinical localization of the origin of a pain arising from the pelvic viscera cannot be made by the characteristics of the pain or from its zone of reference. Lewis calls attention to the similarity of visceral pain and somatic pain in quality and in segmental distribution. Abdominal wall tenderness and muscular rigidity are not conclusive of vis-

ceral pain. Allowable conclusions can only be based on the consideration of the entire picture of pain, its localization, time-intensity curve, the constitutional reaction, or circumstances under which it occurred, its relation to organ functions and the demonstration of visceral disease.

PERITONEAL PAIN

Anatomically the peritoneum has little or no nerve supply. It is very sensitive as a result of the rich innervation of the underlying tissue. Pain impulses from the peritoneum go by way of the intercostal nerves supplying the particular area. Inflammation of the parietal walls causes pain, tenderness and rigidity in the affected area. Hyperalgesia of the skin is frequently present. In diagnosis it is necessary to look upon pain caused by the irritation of the parietal peritoneum as the surface outcropping of underlying disease. This indicates only the extent to which the disease is in contact with the parietal peritoneum and does not indicate either the extent or degree of the primary disease. An extensive and serious inflammation may be so situated that only a very slight amount of the area involved is in contact with the parietal peritoneum. This is not infrequent in diseases originating in the fallopian tubes or in the sigmoid colon. On the other hand, some disease processes are so intimately related to the parietal peritoneum that the signs rather accurately indicate the extent and degree of the process. Usually preceding the development of the peritoneal phenomena are the signs and symptoms of organic disease. Cramplike pain may be transmitted from a diseased viscus to the umbilicus, subumbilical region and the lower back. The constitutional reaction and the abdominopelvic examination usually supply the information necessary to relate all of the phenomena to the primary disease.

SOMATIC PAIN

Pain in the abdomen should direct attention to a visceral origin. Almost universally the tendency is to overlook other causes, and especially the somatic structures of the abdominal wall, as the basis of pain impulses. In doing so we forget that the parietes have a far richer nerve supply than do the viscera and are far more sensitive. Certainly the seriousness of visceral disease and its potential danger have much to do with this attitude. Everyone realizes that pain can be felt over the abdominal viscera as a

result of herpes zoster and the possibility of basal pleurisy with irritation of the intercostal nerves is excluded in every case of suspected appendicitis. As a matter of fact somatic pain is far more common than visceral pain. Failure to understand it has led to missed diagnosis resulting in needless surgery and bad results.

In Lewis' studies on pain he found that the injection of the interspinous ligaments with hypertonic saline solution produces pain similar to the pain of visceral disease. Injection of both recti below the navel produced a pain similar to that of intestinal colic. Carnett stressed the importance of somatic pain many years ago and his views are only now beginning to receive the attention of those interested in the female reproductive system.

According to Judovitch and Bates, the most common and clear-cut type of somatic pain is that of segmental neuralgia. By definition, this is a painful or tender area within one or more sensory skin segments. Etiologically it results from any disease process, toxic absorption or mechanical disturbance which irritates the roots, ganglia or trunks of the spinal nerves. As a result its point of origin is in or close to the vertebrae. Tenderness of the parietes is a major sign in segmental neuralgia. Pinching and pin pricking reveal superficial tenderness. Pressure against bony structures or against contracted muscle and bimanual palpation of the abdominal wall reveal deep tenderness. The diagnosis depends upon the demonstration of tenderness in the paravertebral region or along the rib margins corresponding to the level of the tender anterior distribution. Segmental tenderness rarely if ever occurs as a result of a disease of the pelvic viscera. Tenderness does occur from pelvic visceral disease as a result of involvement of parietal peritoneum overlying the viscera. But this tenderness is local, not segmental, and usually is associated with local pain and many times with some degree of rigidity.

The severity of the pain of segmental neuralgia varies from mild to severe. Most frequently it is mild and is described as a nagging or aching pain or as a soreness. Pain on motion, radiating from the anterior distribution to the back, has diagnostic significance. On vaginobdominal examination, the tenderness may be so great as to confuse the examiner. To clarify the picture, the pressure of the abdominal hand should be decreased slowly. If the pain diminishes and

the vaginal hand can palpate the viscera without pain somatic pain should be suspected. The direct pressure of the intra-vaginal fingers against the sacrum or the side walls of the pelvis is often very painful if the parietes are sore. Palpation of the body of the uterus presses the tender abdominal wall against the uterus supported by the intravaginal fingers and thus causes pain. To determine if the uterus is painful or if the pain is in the overlying abdominal wall the intravaginal fingers are slipped anterior to the uterus and the overlying structures are palpated bimanually. This maneuver can be utilized to palpate a large area of the lower abdominal wall. When the examiner can be reasonably sure that the bladder and intestines are not between the external and internal fingers and that peritonitis is absent this sign is of great value in the diagnosis of somatic pain.

The treatment of segmental neuralgia depends upon finding the cause of the nerve irritation. Many times this is the result of a defect in body posture such as is present in scoliosis, lordosis and ptosis of the lower abdomen. Occasionally an inequality of leg length is the cause. From time to time atrophy of the spinal muscles from old poliomyelitis is noted. Unfortunately in many circumstances it is impossible to find the cause.

In treating the patient for segmental neuralgia one should remember that most of the time pain is of a mild degree. The emotional reaction resulting from the uncertainty and anxiety as to the cause and origin of the pain is in many instances the real cause of the patient seeking relief. For this reason, explanation and reassurance are often all that are necessary. Simple corrective exercises to strengthen the muscles of the lower abdomen and to improve the body posture are always helpful. In severe cases, procaine injections of the affected nerve often produce surprisingly good results.

DISMENORRHEA

Dysmenorrhea, or painful menstruation is one of the most common types of pain confronting the physician. It has been estimated that approximately 30 per cent of menstruating women are subject to either primary or secondary dysmenorrhea. Division of dysmenorrhea into primary and secondary categories is purely an arbitrary one. Primary dysmenorrhea designates painful menstruation unassociated with any demonstrable pelvic pathologic process, in contradistinction

tion to secondary dysmenorrhea which is due to pathologic states within the pelvis such as endometriosis, pelvic inflammatory disease, fibroids or fixed retroflexion. Obviously the treatment of secondary dysmenorrhea should be directed toward the correction of any existing pelvic disease. The problem of dealing with primary dysmenorrhea is a much more difficult one. Indeed it presents one of the most challenging problems in the practice of gynecology. To refer back to the mnemonic the pain of primary dysmenorrhea may be entirely cerebral or entirely organic or any combination of the two. In most cases there is a liberal amount of both factors present and each must be handled appropriately.

Characteristically the pain of dysmenorrhea is cramping in nature and usually quite severe—in many respects simulating labor pains. It may precede the onset of menstruation and its duration may be from a few minutes to several days. Frequently there are associated systemic complaints of nausea, vomiting, headache and backache. Generally the pain of dysmenorrhea does not make its appearance for some several months or years after the menarche. This is considered to be due to anovulatory cycles. When the cycles become ovulatory the dysmenorrhea commences. Following pregnancy the pain is less severe in the majority of women. Also many women tend to outgrow their cramps. In others they persist in the same or in an intensified degree. The exact etiologic basis of primary dysmenorrhea is unknown. Certainly abnormal uterine contractions are known to be a factor.

Treatment of dysmenorrhea falls into three categories: psychotherapy, medical management and surgical treatment. It probably is safe to say that nowhere in medicine is the psychologic evaluation of a patient more important to the eventual successful management. Even after this is done it is sometimes difficult to tell immediately how much of any given patient's pain is functional and how much is organic. It usually requires several interviews and several courses of therapy with various medications to establish the exact relationship. Two important points to include in the psychotherapy of a patient with what is felt to be a fair amount of functional dysmenorrhea are reassurances as to the normalcy of her pelvic organs and careful explanation that uterine contraction is a normal reaction to a physiologic process.

The medical management of dysmenor-

rhea is often effective. If combined with appropriate psychotherapy, probably 70 per cent of patients can be relieved to a point where the dysmenorrhea is tolerable. A variety of analgesics and antispasmodics has been used. A combination of aspirin (5 grains), phenacetin (5 grains) and Propadrine hydrochloride ($\frac{1}{4}$ grain) given at four hour intervals is helpful. Great discretion should be used in prescribing codeine. Other opiates should not be given. Endocrine therapy also may be used and in many cases is extremely helpful. Perhaps its chief value is the establishment of the organic nature of the pain. It is a well-known fact that if ovulation is suppressed, the bleeding which follows is painless. This is the basis for a regimen which is frequently employed as a diagnostic measure. It consists of the administration of stilbestrol, 1 to 2 mg. daily beginning on the first day of menstruation and extending for twenty-one to twenty-five days. Stilbestrol suppresses ovulation during the current cycle and the absence of pain establishes the cycle's anovulatory nature. Androgens also have been used in the treatment of dysmenorrhea. The exact mode of their action is uncertain, but here again it probably is related to a change in the mechanism of ovulation or a decreased formation of corpus luteum hormone. An effective method is to administer 10 to 30 mg. of methyltestosterone daily from the seventh to fourteenth day of the cycle.

Surgical methods for relief of dysmenorrhea consist of dilation of the cervix, with or without insertion of a stem pessary and presacral neurectomy. Dilation of the cervix often produces a remission but rarely cures dysmenorrhea. In our experience, the percentage of success with dilation and curettage has been appreciably increased by the insertion of a coil spring type of stem pessary. This remains in place for a period of two menstrual cycles. However, stem pessaries are not without danger and endocervicitis is an absolute contraindication to their use.

The decision to perform presacral neurectomy on any patient for the relief of dysmenorrhea can be arrived at only after a careful evaluation of the patient and the determination that the dysmenorrhea is uterine in origin and disabling in nature. In well-selected patients and with a thorough operation, the cure rate will be satisfactory.

Doyle recently proposed a simplified method of interrupting the sensory parasympathetic fibers to the uterus and the

sensory sympathetic fibers to the fundus. Both sets of fibers run in the terminal 2.5 cm. of the uterosacral ligament. To denervate the uterus, it is only necessary to cut the uterosacral ligament. The method appears to have many advantages, but it still lacks widespread clinical confirmation.

BLEEDING

The analysis of abnormal genital tract bleeding poses many interesting problems. There is much truth in the saying, "He who knows bleeding knows gynecology." The first problem is to determine if the bleeding is really abnormal. Contrary to popular belief, menstruation does not always occur with clock-like regularity. As a matter of fact, many women exhibit a considerable variation from month to month and it is only when these variations exceed rather wide limits that we are justified in labeling the bleeding as abnormal. In taking a history, therefore, it is necessary to consider the normal variability of the process as well as the normal for that particular woman.

Onset. The great majority of women begin to menstruate between the twelfth and fifteenth years of life. The outside limits of normal are from eleven to sixteen years of age.

Interval. The normal intermenstrual interval is twenty-eight days, plus or minus seven days. There is a good deal of variation among different women and from cycle to cycle in the same woman.

Duration. The normal duration of flow is five days plus or minus three days. In the same woman the length of the period is fairly constant, but it varies among different women.

Amount. The amount of blood lost at each menstrual period also varies widely in different women and in the same woman at different periods. The number of napkins gives a rough idea of the amount of blood lost. During the first two or three days, the flow is usually more profuse and thereafter gradually tapers off.

Character. Under normal conditions, the menstrual discharge is of a dark color and free from clots. When the flow becomes profuse, it is redder and contains clots.

Pain. In 70 per cent of women, menstruation is a painless process, the remainder exhibit some habitual discomfort of a widely varying degree. The onset of pain, or a change in its character has great diagnostic significance in bleeding problems. In

the early stages of cancer for example, bleeding is painless. In abortion the bleeding is definitely painful. If a fibroid tumor is present there may be gradually increasing pain. In ectopic gestation pain is almost always present, while in the common forms of endocrine bleeding pain is generally absent. Hence careful attention to the relationship of pain and bleeding may reap a rich diagnostic reward.

Associated Symptoms. There are many symptoms associated with bleeding which will be encountered from time to time. The most constant is a dull heaviness or dragging in the pelvis. Occurring with this may be headache, nausea and vomiting, depression, nervousness and tension states. Usually these symptoms are not disabling. After determining the characteristics of the patient's normal cycle and the date of the last menstrual period, the change in the patient's menstrual flow is noted as regards interval, duration, amount, character and relationship to pain. Often the patient cannot give exact dates, but even without them a fairly accurate picture of the abnormal bleeding can be obtained. In such cases, a calendar is a great help as the women can readily note thereon the approximate intervals, durations and amounts.

CANCER OF THE CERVIX

Even though the cause of cancer is unknown, much can now be done to control the disease and it is possible that still more can be accomplished in the future. Three-fourths of all genital tract cancer in women occurs in the cervix, a site which is readily accessible for study and examination. Next to cancer of the breast, cancer of the cervix is the most common in the female. It accounts for 10 per cent of the total cancer deaths. Its common occurrence, deadly nature, and ready availability for observation have stimulated an ever increasing number of fruitful investigations.

Cancer Control. In 1900 the cure rate was practically zero. Today it ranges from 35 to 45 per cent and tomorrow it could readily be 70 per cent. Cancer control depends on the elimination of predisposing factors and the diagnosis of the disease in its preclinical form. When cervical cancer can be recognized by clinical means, the chances for cure have already begun to diminish.

Life insurance statistics indicate a drop in the death rate of cancer in females. This is

largely due to a marked decline in the death rate from cancer of the female generative tract.

Predisposing Factors. The predisposing factors present some interesting points of information. The disease is more common in the lower income group than in the upper income group. Next, it is very rare in virgins and occurs only occasionally in the single. Marriage definitely increases its incidence. Those individuals who marry young are the most susceptible. Multiple marriages seem to be of some significance. Prostitutes have more than their share of the disease. All of this indicates that an active sex life is a factor of importance. This naturally leads to pregnancies and a higher incidence of chronically inflamed and lacerated cervixes. Chronic cervicitis has often been described as the breeding ground of cancer and most gynecologists believe that these cervical lesions are potentially dangerous as a future source of cancer. So far all of this evidence incriminates sexual intercourse but this may not be the whole story, as the Jews and Moslems have a very low incidence of the disease. Members of both of these races practice circumcision. Negroes on the other hand, have a very high incidence of cancer. This may be attributed to poor penile hygiene or to a racial factor.

In our enthusiasm to control the disease we should not try to eliminate a function which is a biologic and psychologic necessity but rather we should concentrate on the susceptible individual or groups in our efforts to discover the cause. Needless to say the elimination of cervical irritation is of great importance not only in ruling out a potential source of danger but also in directing the attention of physician and patient to the cervix. The data on circumcision and penile hygiene are suggestive. The wide adoption of circumcision and good penile hygiene can do no harm and may be a step in the right direction.

Histologic Changes and Metastasis. The basal layer of the normal cervical epithelium consists of several layers of deep-staining cells. Squamous cell carcinoma originates from these cells. At first there may be basal cell hyperactivity with an area of proliferation six to ten cells deep. Gradually the process involves the entire epithelium. In well marked cases, there is complete loss of the normal stratification of the epithelium. The cells vary in size and shape, and mitoses as well as nuclear irregularities, are present. The cells are identical with malignant cells.

But invasion, the most important characteristic of malignancy appears to be lacking. To establish the fact that it really is lacking requires a careful examination of many specimens from the cervix. Often this extensive study reveals invasion. Many times, in clear-cut invasive carcinoma, there is an area of preinvasive carcinoma at the margins of the growth. From a therapeutic standpoint, therefore preinvasive carcinoma must be considered as invasive until proved otherwise.

There have been a number of reported cases in which it was possible to trace the sequence of events from basal cell hyperactivity to frank invasive carcinoma. In one of our cases, described by Jones, the changes extended over a thirteen year period. The transition to a malignant state may be a slow process. Preinvasive carcinoma reaches the peak of its incidence in women at the age of thirty-eight years. Invasive carcinoma in those forty five years of age. On the other side of the picture it has not been determined that preinvasive carcinoma always progresses to invasive carcinoma. At times regression occurs and this is especially likely following the termination of a pregnancy. Once the process acquires sufficient growth momentum to become invasive it loses forever its capacity to undergo spontaneous regression.

Cancer of the cervix spreads by three methods. The first is by continuity or direct extension. This type of spread begins primarily with involvement of the cervix. The growth then extends laterally in any of three main directions—the paracervical tissue, the paravaginal tissue or along the uterosacral ligaments. The second method is by the lymphatic channels. This type of metastasis is embolic in nature. The most common nodes involved in our experience are the obturator hypogastric, and the external and common iliac nodes. The paraaortic node has not been frequently involved in the earlier cases. As shown by operative results, the lymph node involvement of stage I lesions is approximately 18 to 20 per cent, in stage II lesions 30 to 35 per cent. In the more advanced lesions, the percentage of involvement increases. The third method of spread of carcinoma of the cervix is by the blood stream. This usually is a late occurrence in the disease. Carcinoma of the cervix is restricted within the limits of the pelvis for a relatively long time before distant metastasis by the blood stream occurs.

Classification and Grading. The size of

the tumor is closely related to the degree to which it metastasizes and its curability by radiation or surgery. In order to compare the results of treatment, it is necessary to consider patients in whom the growths are approximately the same size. For these reasons therefore it has been necessary to establish a uniform descriptive system. The following has been adopted by the League of Nations Health Organization and is now used universally. It is sometimes referred to as the International Classification.

Stage 0 Noninvasive carcinoma confined to the epithelial margins of the cervix.

Stage I The carcinoma is strictly confined to the cervix.

Stage II The carcinoma infiltrates the parametrium on one or both sides but has not involved the pelvic wall. The carcinoma infiltrates the vagina but does not involve the lower third. The carcinoma involves the endocervix and has spread to the corpus.

Stage III The carcinomatous infiltration of the parametrium has involved the pelvic wall on one or both sides. On rectal examination, no cancer-free space is found between the tumor and the pelvic wall. The carcinoma involves the lower third of the vagina. Isolated metastases are palpable on the side wall of the pelvis, irrespective of the extent of the primary growth.

Stage IV The carcinoma involves the bladder as determined by cystoscopy or by the presence of a vesicovaginal fistula. The carcinoma involves the rectum. The carcinoma has spread outside the true pelvis.

For most practical purposes adenocarcinoma of the cervix may be considered to behave as does squamous cell carcinoma. Because of its origin within the cervical canal, it presents a little more diagnostic difficulty and because of its location it is a little more likely to spread laterally early in the disease. In spite of these minor differences, the results of treatment are about the same in the two groups.

Both squamous cell carcinoma and adenocarcinoma of the cervix exhibit marked differences in their histologic characteristics. Some squamous cell carcinomas consist of cells which are very highly differentiated and closely resemble the cells of the normal cervix. In others, the cells are completely anaplastic and have no resemblance to normal cervical cells. As a result of these differences in the appearance of the cells of different tumors, various systems of histologic gradation have come into being. The two classifications in common use are those of

Martzfloff and Broders. Martzfloff used the three cell types of the normal cervical epithelium as the basis of his classification.

1. *Spinal cell type* representing chiefly the external layer of cells. These are the most benign of the epidermic carcinomas and are classified by Broders as grade I.

2. *Transitional cell type* consisting of cells resembling the middle layers of the cervical epithelium. These cells are less differentiated than the spinal cells and are more malignant. Broders places tumors consisting of these cells in grade II.

3. *Spindle cell or basal cell tumor*. In this group the cells resemble those from basal layers, the so-called stratum germinativum. These are the most undifferentiated and anaplastic of the cervical cells and are the most malignant type. Broders classifies tumors of this type as grade III or IV.

Transitional cell tumor (grade II) is the most common, with the spinal cell (grade I) next in frequency and the spindle or basal cell (grade III) the least common.

The cell types are important and are thought by many pathologists to be of significance in the prognosis and selection of treatment. But one should bear in mind that there is a marked variation in all types and in different portions of the same tumor. Rarely does a tumor at any one spot consist entirely of one clear-cut cell, but rather is a mixture of several types, one of which usually predominates. A single biopsy may give an erroneous impression of the tumor as a whole. All of these factors must be considered if histologic grading is to be of any significance.

Symptomatology. Cervical cancer in its curable stage presents few if any symptoms. As the disease progresses to the incurable stage the classical symptoms of discharge, bleeding and pain make their appearance. Patients, as well as physicians, must learn that to await the appearance of symptoms is to diminish the chances of cure. In the typical case discharge is usually the first symptom. It is often mild and frequently unnoticed until bleeding occurs. Many times the first bleeding follows intercourse. This postcoital bleeding is a very suggestive symptom and always calls for investigation. Other forms of contact, such as with a douche nozzle or by the insertion of a diaphragm, or the downward descent of the cervix against the posterior wall of the vagina as a result of straining, may cause bleeding. Bleeding implies a break in the surface covering of the growth and, follow-

ing this infection occurs with an increase in the amount and character of the discharge. The discharge usually is serous or serosanguineous at this stage and gradually begins to develop an odor which in advanced cases can only be described as stinking. The discharge is in many respects similar to that emanating from a chronic leg ulcer. The amount of odor roughly parallels the degree of slough. As sloughing proceeds the process opens larger vessels, the bleeding becomes more profuse and often is continuous. In late stages of the disease it can exanguinate a patient very rapidly. The bleeding is not related to the normal menstrual cycle but is superimposed and depending on circumstances it can and does mimic all the varieties of abnormal menstruation.

Pain, as such, does not occur in the early stages of the disease but there is a strange awareness of something not right which patients often describe as occurring early. This is usually labeled as discomfort. Later as the growth extends and involves the nerves the pain becomes severe. Especially characteristic is pain in the low back and pelvis with radiation into the back of the upper thighs.

Diagnosis. The first step in examination is to visualize the cervix and take smears from it and the vaginal pool. When these are stained, a competent cytologist can state if cancer is present or absent in a high percentage of cases. This technique first described by Papanicolaou and Traut, depends on the exfoliative nature of cancer cells and its recognition. Unfortunately the interpretation of the smear is not so easily learned, is time consuming and requires very experienced personnel. For this reason it is difficult to use on a routine basis. But where it has been used, the results have been worth while. It is especially valuable in the detection of the preinvasive and the early invasive cases.

The great value of the Papanicolaou technique is that cells from all portions of the cervix and uterus can be examined on a single slide. It is a diffuse test, while biopsy is focused on a few spots. The great promise of the Papanicolaou technique is in the routine detection of early cancer. At the present time, it is best to regard it as purely a screening test. In all patients having a suspicious or positive report, biopsy should be performed. To our way of thinking, the colposcope affords important advantages in selecting the areas for biopsy. With this instrument it is possible to examine the

cervix with magnifications of $P \times 20 \times$ and many changes invisible to the naked eye are apparent. Preinvasive carcinoma and invasive carcinoma present well-defined characteristics.

Preinvasive carcinoma may present no recognizable signs on visual examination. In some cases the bright red color of an erosion changes to a paler saffron-like color. Dilatation of the surface veins over this area is suggestive. Painting the cervix with compound tincture of iodine stains the normal squamous epithelium a mahogany brown. Abnormal areas do not absorb the stain. This is known as Schiller's test. It delineates the abnormal areas and it is from these areas that we must take our biopsy specimens. In early cases a single biopsy may miss the lesion in perhaps half the subjects. At least four pieces of tissue should be taken from the circumference of a cervix. In taking biopsy specimens it is helpful to remember that the significant changes are in the epithelium. It is easy to remove the epithelium from the hard, unyielding stroma by taking a superficial bite with the biopsy instrument. The correct technique is somewhat like that which would be used in attempting to take multiple pinch grafts from the cervix. When the disease becomes invasive the tissue appears friable and granular. It bleeds readily at the slightest touch. A probe passes easily into the growth until it reaches the uninvolved cervical stroma.

Advanced cancer of the cervix is not difficult to recognize. It may be either a cauliflower-like growth, an infiltrating growth or an ulcerated growth. Everted growths form cauliflower-like lesions which are soft, friable and bleed easily. Infiltrating growths grow beneath the mucous membrane and into the stroma. For this reason the cervix is hard and has a suggestion of nodularity. Ulcerating lesions can result from either an infiltrating or a cauliflower growth. The latter is the more frequent cause.

Treatment. Cancer of the cervix yields only to surgery, irradiation or a combination of the two. To use either successfully requires skill and experience with the disease. The selection of patients suitable for each form of treatment varies from clinic to clinic. Ruth Graham believes it possible to determine the response of a given patient to radiation by cytologic study and thereby to select the appropriate form of treatment. The confirmation and perfection of her method would go a long way toward solving the problems of treatment. I that th

each patient must be treated with respect to her functional needs, the stage and grade of the lesion and the abilities and experience of the physician. In this disease, the latter counts as in no other pelvic condition.

Since preinvasive carcinoma occurs in a comparatively young age group, the patients often have greater functional needs than do those suffering from invasive carcinoma. In addition, the long, latent period and the likelihood that the disease will not explode overnight, make the problem a little less pressing. For these reasons, the physician can assume the calculated risk of observation or local treatment in exceptional cases, provided of course he has done all necessary in his judgment to exclude invasion. This can best be accomplished by surgical conization of the cervix. By following a conservative course in these cases, he may allow the patient to satisfy her reproductive desires. Such a course however does entail risk and is not to be entered upon lightly. When the patient's desire for reproduction is not so important, the consensus is to proceed immediately with total hysterectomy. In doing this operation, the parametrial clamps should be outside the cervical fascia. In order to place the clamps properly to avoid uterine injury, it is necessary to expose these structures. A generous portion of the vagina adjacent to the cervix should be removed with the specimen. This operation is considerably more difficult than is routine total hysterectomy. Te Lindé describes it as a modified radical hysterectomy. If indicated, the ovaries can be safely conserved.

In our clinic, we use a modified Stockholm technique in radiation therapy for carcinoma of the cervix. Basically it is a combination of treatment with radium and supplementary deep x-ray radiation. More recently we have used cobalt radiation. The application of radium is the most important part of the treatment and usually is given in two applications, separated by a period of three weeks. The radium is applied in the vagina, about the cervix and in the uterine cavity. No radium is placed in the lower 0.5 cm. of the endocervical canal. The vaginal radium is applied by means of three tubes—one in each lateral vault and one posterior to the cervix in a transverse position. These tubes are placed in this position to radiate the areas of direct extension, that is, the parametrial tissue, the paravaginal tissue and the uterosacral ligaments. These tubes are incorporated in a dental compound which holds the three radium containers firmly in position in the cervix. The intrauterine

radium extends the entire length of the uterine cavity. The dosage varies from 3000 to 4000 mg. hours for each treatment according to the size of the lesion, the width of the vagina and the length of the uterine cavity.

Deep x-ray or cobalt therapy may precede the use of radium in those patients in whom the lesion is bulky and infected or extends down the vaginal mucosa. Deep therapy through two perineal fields can be helpful in cleaning up the markedly infected lesions. The filtration used in the application of radium is equivalent to 1 mm. of platinum. It is generally accepted that a tumor dose is 5000 r. This technique will deliver approximately 6000 to 7000 gamma r at the point called 1/2 cm. lateral from the center of the cervix and 2 cm. beneath the mucous membrane on the lateral vaginal vault. At the area on the pelvic wall in the region of the obturator nerve the average dosage is approximately 1200 to 1500 gamma r by this method of application. It is obvious that the majority of the tissue lateral to point A receives an insufficient tumor dose, and deep radiation therapy is necessary to increase the amount of radiation in the parametrium and on the pelvic wall. It is rarely possible however to achieve a total dosage of more than 3500 to 4000 r in the typical patient in the region of the obturator fossa. The deep x-ray therapy is given through two anterior and two posterior portals. The total skin dose varies from 3200 to 4000 r to each field. With cobalt therapy a tumor dose of 3500 to 4500 r can be given to the parametrial and pelvic wall areas. The skin reaction is minimal and nausea and general lized discomfort are less. Heyman and Kottmeier of Stockholm were the originators of this technique. They were pioneers in the development of radiotherapy for carcinoma of the cervix. In their patients treated between 1936 and 1943, at the end of five years they had a relative apparent recovery rate of 69.2 per cent in stage I, 51.4 per cent in stage II, 34.4 per cent in stage III and 11.3 per cent in stage IV.

In the early 1900's, before the advent of radiotherapy, surgery offered the only hope for cure of cancer of the cervix. True, it was a slim hope, but some patients were cured. At this time surgery was in its infancy. Anesthesia was poor, transfusions unknown, wound healing not understood and there were no antibiotics. It was also before the days of the early diagnosis of cervical cancer and most of the cases were advanced. In spite of all this, surgeons cured

a fair number of patients with cervical cancer. But there were many disappointments: long hours at the operating table, fearful hemorrhages and shock, convalescence was long and difficult. *Fistulae were frequent.* In 1904 Abbe, a New York surgeon, began to treat cervical cancer with radium. The results were miraculous. No wonder surgeons turned to the promise of radiotherapy.

Of course, radiotherapy had its disappointments too and they were somewhat similar to those experienced in surgery. But the ease of treatment and the good palliation encouraged even the most skeptical. As soon as the new method was perfected, results would be better. However, in the course of time the cure rate reached a plateau. This was about the time that surgery was really developing. It was only natural that surgeons again interested themselves in cancer of the cervix. In this country Lynch, Taussig and, above all, Meigs led the surgical revival. So far the surgical results have been superior and somewhat better than the best from radiotherapy. But comparisons are not valid because the surgical cases represent a selected group. Therefore the question of the superiority of surgery or radiation is not settled. Probably it cannot be settled for a long time by statistical means. Most agree that surgery is ideal for many early cases. For the rest, radiation is best. To solve the problem we must discover solid indications for each type of treatment.

Our personal reaction to this situation is a double barreled technique—preliminary radiation followed by radical hysterectomy with bilateral pelvic lymphadenectomy. So far our results are encouraging to us but there are not enough cases to warrant positive recommendations to others.

In considering the problem of the treatment of cancer of the cervix, it is easy to see the tree and to overlook the forest. Both radiation and surgery can cure cancer of the cervix. We know that the stage of the lesion influences the cure rate more than does the form of treatment. Of course perfecting either form of treatment will increase the number of cures. But the best results come from early diagnosis for with early diagnosis either surgery or radiation can eliminate the mortality of cancer of the cervix.

CARCINOMA OF THE CORPUS UTERI

Today carcinoma of the corpus uteri is being recognized more frequently than ever

before. It is stated that the ratio of incidence of carcinoma of the corpus to carcinoma of the cervix uteri is approximately 8:1. In our material this ratio is approximately 3.5:1. An increase in the life span is an important factor in this increased incidence of the disease.

In America the disease is rare before the age of forty. 25 per cent of cases occur before age fifty while 75 per cent of cases occur after this age. The peak of the incidence curve is in the mid-fifties. The probability of a woman developing the disease is 15 per thousand at age forty-five, and this decreases to about 7.5 per thousand at between sixty-five and seventy. In any series of cases the high percentage of multiparous women is striking; in some series this is as high as 40 per cent. Some surgeons have suggested the presence of prolonged ovarian activity as a factor in the pathogenesis of carcinoma of the corpus.

A hereditary influence seems to occur in a significant number of histories. There are records of the disease occurring in identical twins with an onset at the same approximate time. From an economic standpoint the number of private patients is significantly greater than in cancer of the cervix. Often times the women are large and obese. One authority has described them as burlesque. In Corcadens's cases, the women with cancer of the endometrium weighed on an average of eighteen pounds more than those with cancer of the cervix. There is more than an ordinarily close relationship with diabetes. Palmer gives the amazing figure of an incidence of 167 times that in women of the same age in the general population.

Novak stresses the relationship between postmenopausal endometrial hyperplasia and the development of carcinoma of the body of the uterus. While this opinion is not unanimous the evidence is suggestive. Kottmeier puts greater emphasis on the association with endometrial polyps than with endometrial hyperplasia. The age of the menopause lends credence to this suggestion. In women who do not develop cancer of the endometrium, only 8 per cent menstruate beyond age fifty while in those who do 35 per cent continue their periods beyond the fiftieth year.

The chief symptom of carcinoma of the uterus is usually some form of abnormal uterine bleeding. In women who have not passed the menopause this is characterized by intermenstrual bleeding. Only a very small percentage of preclimacteric bleeding

is due to cancer of the endometrium, but when the common causes of bleeding are eliminated the incidence rises to about 10 per cent. In the postmenopausal woman the bleeding may vary from a small amount of spotting to an excessive type of flow. Slightly more than a third of all women having bleeding a year or more after the menopause will be found to have carcinoma of the endometrium. An increased vaginal discharge may also be noted. Early in the disease the discharge is odorless later as the tissue begins to degenerate it may be foul. At intervals, this discharge is blood tinged. Pain is not a frequent symptom. However most patients do complain of a vague lower abdominal or pelvic heaviness or discomfort.

The efficiency of vaginal smears in the diagnosis of this disease is less than in cancer of the cervix. As in cervical cancer a positive smear must be confirmed by tissue study. For this purpose, as well as in the differential diagnosis of the disease endometrial biopsy is useful. This is an office procedure and, with experience, one can sample the endometrium almost as thoroughly as with a conventional curettage. The method is not applicable to the woman with a rigidly stenotic cervix or to the extremely unstable woman. Dilation and curettage is reserved for these women, as well as for those in whom endometrial biopsy does not give a clear-cut answer.

For curettage Heyman emphasized the fractional technique. It should be done in the following manner: (1) curettage of the area around the external os, (2) curettage of the endocervical canal without passing the internal os, (3) measuring the length of the uterine cavity, (4) careful dilation of the cervix, (5) examination of the fundus of the uterine cavity with polyp forceps, (6) curettage of the fundus and the corpus and examination of the uterine cavity with forceps and the curet. This method of curettage is most important in the differentiation between carcinoma of the body of the uterus and carcinoma of the body of the uterus and endocervix. This differentiation is of significant importance, both in the prognosis of the disease and in determining the form of therapy.

Carcinoma of the corpus uteri is an adenocarcinoma arising from the endometrial glands. The gross lesion varies from a small circumscribed one to a diffuse polypoid growth which fills the entire uterine cavity.

Microscopically the tumor also shows much variation. In the well-differentiated lesion, the glandular pattern is still present,

but as the degree of differentiation becomes less solid masses of malignant cells replace the glands. Mitotic figures and nuclear changes are prominent.

Hertig was one of the first to investigate the carcinoma in situ phase of adenocarcinoma of the corpus uteri. Histologically he bases his diagnosis on the appearance of endometrial glands composed of large cells with abundant clear eosinophilic cytoplasm. In addition to this the nuclear membranes are irregular and arranged in irregular palisades. Cellular disorientation, variation in size and stratification are common findings. This contribution is a real step forward in the control of carcinoma of the body of the uterus.

The principles of treatment for carcinoma of the uterus depend upon the clinical characteristics and the method of spread of the disease. When the diagnosis is made by tissue biopsy we first apply intracavity radium by the multiple packing technique. A total dosage of 4000 to 5000 mg. hours is given, depending on the size of the uterus and the number of applicators used. This technique gives more uniform radiation through the entire uterine cavity. The number of uteri with residual carcinoma has been greatly reduced by this form of therapy. Operation then follows the radiation therapy in approximately six weeks. It is generally believed that age, obesity, hypertension and diabetes often complicate the treatment. Hence as a matter of necessity the condition of the patient determines the extent of the operation. Under ideal circumstances the operation is a radical hysterectomy, bilateral salpingo-oophorectomy and bilateral pelvic lymphadenectomy. If this seems too much of an operation for the patient the surgeon must do a lesser operation, consisting of a wide extrafascial removal of the uterus and a portion of the upper vagina. Some surgeons speak of an operation of this magnitude as a semiradical hysterectomy. In our hands, it pretty closely approximates the original Wertheim type of operation. It is definitely a more difficult operation than is the ordinary total hysterectomy. This latter operation may occasionally have to suffice in the very old or the very sick, but it is not a sound operation for the removal of any form of uterine malignant lesion.

Of importance is the postoperative use of radium in the vagina. Approximately 5 per cent of the patients will develop recurrences in the vaginal cuff. A lesser per cent will develop metastatic lesions in the peri-

urethral area. The use of prophylactic vaginal radiation postoperatively will reduce the incidence of these complications. This radium application is usually given in the form of a large bomb which fills the vaginal cavity. The dosage varies somewhat according to the size of the vagina. The average dose is 1500 to 1800 mg. hours. We do not use deep x-ray therapy postoperatively except in patients in whom ovarian metastasis or obvious metastatic spread has occurred beyond the limits of the uterus. In these patients and in those in whom the spread is extensive and surgical treatment is not feasible x-ray or cobalt therapy is given to two anterior and two posterior portals. In the group in whom operation is not undertaken the deep therapy is preceded by a relatively large dose of radium applied within the uterus by the packing technique. Application is usually divided into two doses at three to four week intervals.

Today the over all expected five-year survival rate in all patients with carcinoma of the body of the uterus is 50 to 60 per cent. In patients in whom operation is clinically possible the five year apparent survival rate rises as high as 70 to 80 per cent.

It is important to remember that approximately 5 per cent of the malignant lesions of the body of the uterus are sarcomatous in nature and that the majority of these arise in myomas. Sarcoma of the endometrial stroma is seen occasionally. The therapy is primarily surgical however preoperative use of radium within the uterus of patients with endometrial sarcomas is of benefit.

Rare cases of carcinosarcoma have been reported in the literature. It is our opinion that many of these lesions turn out to be an undifferentiated type of adenocarcinoma when differential stains are employed.

ABORTIONS

Abortion is one of the five major causes of abnormal uterine bleeding, as designated by the mnemonic C-A-F-E-E. The term "abortion" is defined as the expulsion of the products of conception from their site of implantation before the period of fetal viability. In some areas, the term is applied to fetal loss up to the twenty-eighth week, in other areas, as in Tennessee, the term abortion is applied to fetal loss up to the twentieth week of gestation.

It has been estimated that approximately 18 to 20 per cent of all pregnancies terminate as abortions. This incidence is somewhat higher than the usual hospital incidence of

10 to 15 per cent because of the large number of unreported or unrecognized cases. More than 750 000 abortions occur in the United States each year.

A number of adjectives have been used in order to describe the various phases of an abortion. Abortions may be divided into the following categories: threatened abortion, inevitable abortion, incomplete abortion, complete abortion and missed abortion.

Threatened Abortion. Threatened abortions most often occur in the first trimester of pregnancy. The expected times of the second and third menstrual periods are the periods of danger. The one constant symptom is uterine bleeding. The amount may vary from minimal spotting to a flow that is equal to the flow of a normal menstrual period. The color of the flow may vary from a dark brownish stain to the bright red of fresh bleeding. Frequently a brownish stain will precede the onset of a more profuse flow. The presence of abdominal pain is variable. When pain is present it is of the visceral type characteristic of the contracting uterus. It is intermittent and not constant. This pain must not be confused with the short lasting, sharp catchlike pains present in both inguinal regions in the first trimester.

In addition to the usual signs of pregnancy the significant finding is a closed cervix without apparent shortening. The blood in the vagina varies in amount and color according to the rate and duration of the bleeding. Clots may be present if the flow is profuse.

Inevitable Abortion. This form of the moving abortion picture shows definite evidence that fetal loss is imminent. The bleeding is usually greater in amount and the blood redder than in threatened abortions. Usually the bleeding is constant but varies in amount as fresh pieces of placenta separate. Clots indicate profuse bleeding. Owing to the fact that blood tends to accumulate in the upper vagina, there may be periods in which the uterine bleeding is fairly heavy and the external bleeding only slight. When the patient bears down, as with urination, the vagina is emptied. Patients describe these sudden gushes of blood from the vagina as flooding spells. Lower abdominal cramps are more prominent than in the threatened abortion and are similar to labor pains. These make the patients so extremely uncomfortable that they often refer to an abortion as being more painful than labor.

All the signs and symptoms of pregnancy are present. The uterus is enlarged accord-

ing to the duration of the pregnancy. Cervical effacement is definite. The external os may be dilated and frequently will admit one finger. On inspection, the membranes may be seen to bulge in the dilated os. Sometimes the membranes may be ruptured and the fetus is seen presenting at the os.

Incomplete Abortion. This type of abortion shows a variable picture. The history may be short or long, from a few hours to several months. As pregnancy proceeds the chorionic villi attach themselves more firmly to the uterine wall. This firm attachment predisposes to incomplete separation of the placenta resulting in an incomplete abortion. Prior to the eighth week, the placenta separates easily but after the eighth week the attachment is so firm that incomplete separation is the rule.

Pain, bleeding and the passage of the products of conception are the chief symptoms. The pain is often severe and similar to a labor pain in all respects. Bleeding is profuse, sometimes to the point of exsanguination. Transfusions are frequently necessary. The passage of the fetus or portions of the placenta may be masked by the passage of clots. Patients frequently mistake clots for tissue and almost invariably describe them as "like chicken liver." For these reasons the physician is wise to instruct the patients to save everything passed.

In the chronic cases there is a history of bleeding and cramping followed by a more or less painless flow which varies in amount from a continuous bloody discharge to a moderately profuse flow. Occasionally the flow will become foul in odor which indicates degenerating and infected tissue.

The size of the uterus may conform to the duration of the pregnancy, especially if the examination occurs shortly after the onset of signs and symptoms. But as the process continues and as portions of the conceptus pass, the uterus becomes smaller and firmer. Early in the process, the cervix is open and frequently a piece of placenta lodges in the os. Later the cervix shows a lesser degree of dilatation and no evidence of placental tissue.

Complete Abortion. The uterus has expelled the entire conceptus in complete abortions. Because of the weak attachment of the chorionic villi, complete abortions occur mostly before the eighth week. The history is of cramping, bleeding and the passage of tissue. By the time the physician sees the patient, the cramps have ceased and the bleeding is minimal.

The cervix shows moderate dilatation with no visible tissue. The bleeding has largely ceased and there is a serosanguineous discharge from the os. The uterus is slightly enlarged, contracted and firm.

Mixed Abortion. In mixed abortion the conceptus remains in the uterus after the death of the fetus. The history reveals the development of the signs of pregnancy followed by their regression. A most suggestive sign is the involution of the breasts. After the death of the fetus reactions to pregnancy tests become negative. At times the history is hard to interpret and the diagnosis is difficult.

The physician who is following the patient will note the failure of the uterus to grow. If the patient is seen some time after the death of the fetus the uterus will be smaller than expected at that particular stage of gestation. It may have a very soft, boggy feeling. The cervix will be closed. A dark brown discharge may be seen coming from the os. Sometimes the discharge may have a foul odor.

Treatment. In abortions the choice of treatment is and always has been difficult. The old axiom that an accurate diagnosis is the basis for sound treatment does not always apply in abortions. An examination is not without danger. It may introduce infection or stimulate the contractions of the uterus. It might even cause a threatened abortion to progress to an inevitable abortion. There is a natural reluctance of the physician to hasten or stimulate a threatened abortion or to do anything which a patient might even think caused her to abort. For all these reasons physicians are inclined to treat expectantly and without examination, these patients with the symptoms of a threatened abortion. This is a good plan but it is easy to overdo. If the symptoms do not abate in twenty-four to forty-eight hours or if they increase in severity an examination is in order. In making the examination, gentleness and observance of a sterile technique should be practiced.

If the cervix is closed, the abortion is still only threatening and conservative treatment must be continued. On the other hand, if there is a shortening of the cervix with dilatation or a bulging of the amniotic sac, the condition is considered to be an inevitable abortion. In the opinion of many physicians this diagnosis is justification for the institution of therapy designed to stimulate the uterus. In many hospitals, such treatment would cause severe repercussions. However

If the physician is convinced of the inevitable nature of the process and the ineffectiveness of his treatment, he may abandon those measures considered useful in threatened abortion and leave the outcome to nature.

As the process proceeds the uterine contractions rupture the membrane and extrude the fetus and portions of the placenta. Under these circumstances it is proper to administer drugs to stimulate the process or if necessary to complete the emptying of the uterus by surgical means.

In every patient with a threatened abortion bed rest is indicated as an initial procedure. To quiet the patient and to stop the contractions barbiturates and opiates are useful. Care should be given to the bowels so that the patient need not strain unnecessarily during evacuation. Large doses of vitamin C and the flavonoids have been used favorably. The use of hormones is popular but controversial. They will not harm and may be of value. Stilbestrol is the most widely used. The dosage varies according to the severity of the symptoms and the beliefs of the physician. In one plan of treatment 25 mg. of stilbestrol is given hourly until the bleeding is controlled. The administration of progesterone is a rational form of therapy and perhaps useful if the drug is given in a dosage of 100 to 150 mg. per day. Very recently, Norlutin 10 to 30 mg. daily has been used with success. There have been some observations indicating an effect of the progesterone-like compounds on the secondary sex characteristics. So far this suggestion has not been confirmed. Such therapy is however very expensive. Chorionic gonadotropins are also being used and are perhaps of value.

In patients who have had incomplete abortions of short duration and with mild symptoms one may use an intravenous Pitocin drip to stimulate the contractions and complete the abortion. In patients with more severe symptoms, surgical interference is necessary. Often the blood loss is severe and transfusions are lifesaving. Antibiotics are always indicated.

In the operating room after gentle dilation of the cervix, the finger placental forceps and curet are used to remove the remains of the conceptus. In so doing, the operator may be alarmed by a very brisk hemorrhage. This results from open sinuses in a noncontracted uterus. In such circumstances uterine massage, a firm pack and the use of Ergotrate and Pitocin will quickly control the bleed-

ing. The pack is removed in twenty-four hours.

In the missed abortion, conservatism is indicated. The use of estrogens over a seven- to ten-day period is frequently followed by the passage of the intrauterine contents. This form of therapy may be repeated several times. Practically all patients will empty their uteri if given sufficient time. In the very occasional uterus which does not empty surgery is necessary.

FIBROIDS

The terms myoma fibroid, fibromyoma and leiomyoma are used interchangeably. From an anatomic point of view fibromyoma is preferable.

Fibroids are the most common pelvic tumors and occur in 20 per cent of women over thirty years of age. They are less frequent in those between twenty and thirty years of age but do occur in this group. New growths do not appear after the menopause, but previously existing tumors may rarely increase in size or undergo degenerative processes which make surgical interference necessary. It is well known that Negro women are more susceptible than white women.

In fibroids, the percentage of fibrous and muscle tissue varies. The cut surface is firm and pale when fibrous tissue predominates. A redder or pinkish color indicates an increase in the relative amount of smooth muscle.

All fibroids originate in the myometrium. Traditionally they are described as intramural, subserous and submucous.

Subserous tumors grow out from their original intramural position and come to lie under the visceral peritoneum. As the tumor continues to grow, it may become pedunculated. At times its blood supply may be inadequate and it may develop a secondary blood supply from an omental adhesion. Sometimes the original pedicle spontaneously separates and the tumors only attachment is to the omentum. Under such conditions it is known as a wandering fibroid. Pedunculated subserous fibroids may twist on their pedicles and this may require immediate attention. Tumors which grow out between the leaves of the broad ligament are intraligamentary fibroids. Because of their proximity to the uterine arteries, they are well nourished and may grow rapidly. Intraligamentary fibroids push the arteries and veins outward on their surface; at times they may displace the ureter.

A submucous myoma grows toward the uterine cavity and comes to lie under the endometrium. When submucous tumors become pedunculated they tend to act as a foreign body causing uterine contractions and dilatation of the cervix. Degenerative changes, infection and bleeding, (the latter often marked) are common with these tumors. Tumors which maintain their original intramural position may grow to good size and, in so doing, elongate and distort the endometrial cavity. The growth of such a tumor increases the square area of the endometrial cavity and so increases its menstruating surface. Menorrhagia therefore is common.

Mucoid degeneration follows prolonged circulatory inadequacy. Calcification occurs in tumors of long standing. It is especially common after the menopause. Necrosis occurs from interference with the blood supply. Its causes are a twisted pedicle or circulatory changes occurring during pregnancy or during the puerperium. A special form of necrosis (red degeneration) occurs frequently in pregnancy. Its exact cause is unknown. The red color results from hemolysis. Cystic degeneration also results from necrosis. Local liquefaction of tissue causes the appearance of the cystic spaces. The incidence of sarcomatous degeneration varies between 0.41 per cent and 1.02 per cent. A rapidly growing tumor indicates the possibility of a sarcomatous change. Some believe that cancer of the endometrium develops as frequently in the normal as in the fibroid uterus. Others hold that fibroids predispose to adenocarcinoma of the endometrium. Most of the cases in which the two conditions occur are in patients exhibiting postmenopausal bleeding.

Sterility, low fertility and a tendency to abortion and premature labor are common in women with fibroid tumors. Pain resulting from a degeneration or a twisted pedicle excites uterine contractions and so terminate the pregnancy. At term, the tumors may cause malposition and interfere with the force of the uterine contractions or obstruct the delivery. In the puerperium, there is an increased risk of postpartum hemorrhage and puerperal infection. From a statistical standpoint, fibroid tumors certainly impose an added risk to the pregnancy, but in spite of this the great majority of women with fibroid tumors go through pregnancy and labor satisfactorily. In evaluating the possible effect of a fibroid on a pregnancy, size is the first consideration. With large tumors there may not be enough room in

the abdomen for both the tumor and the baby. The position of the tumor is also of much importance. A small tumor low in the pelvis may block labor while the same tumor arising from the fundus is of little significance. Pedunculated tumors are very liable to twist during pregnancy.

Most fibroids are asymptomatic and require no treatment. Bleeding is the most troublesome symptom. The mechanism is not always the same. As mentioned large tumors increase the menstruating surface of the endometrium. Since intramural tumors are just under the endometrium, some believe they cause bleeding, as a result of pressure on the veins. A submucous myoma acts as a foreign body within the uterus and bleeding results from pressure necrosis of the uterine endometrium in apposition to the tumor. Usually, however, bleeding occurring in conjunction with fibroid tumors is from none of these causes but is the result of associated ovarian failure. If this fact can be established it may alter the decision as to the treatment. The possibility of the simultaneous presence of adenocarcinoma and fibroids increases as the patient passes through the menopause. In postmenopausal women with fibroids and bleeding, the chances are that the fibroid is incidental and that some other cause such as adenocarcinoma of the endometrium is present. In a small minority of postmenopausal women, fibroids which did not bleed during menstrual life migrate to a submucous position and cause bleeding.

Pain from fibroids may be felt in varying degrees and in many forms. The pain resulting from a twisted pedicle may be severe. Degeneration causes pain and sometimes these pains are severe. Dysmenorrhea is frequent, it increases in severity when it has been present before the advent of the fibroid. When the masses are heavy, there may be a sense of weight in the pelvis. Bladder pain may result from inability of the bladder to expand. Pain from pressure on the rectum is rare. Fibroids can cause dyspareunia, especially if the tumor is impacted in the cul-de-sac. Sometimes large abdominal tumors are painful during intercourse.

Fibroids produce few general symptoms unless the bleeding has been severe. Severe secondary anemia was not uncommon before the days of modern surgery and, in such patients, myocardial degeneration was a frequent occurrence. In patients with large tumors, there is sometimes a diminution of the vital capacity.

In the treatment of any given fibroid four

courses are open expectant treatment, radiation therapy, myomectomy and hysterectomy.

Many fibroids require only expectant treatment. Observation should be considered when the tumor is asymptomatic, smaller than the size of a uterus that has been gravid for three months and the diagnosis is clear cut. It must be ascertained that the tumor is a fibroid and not an ovarian cyst. If observation is elected, coexisting malignant disease in the cervix and endometrium must be considered and its possibility eliminated by appropriate examinations. The rationale for observation is greater the nearer the patient is to the menopause.

In fibroids associated with bleeding, expectant treatment is also permissible under the above conditions. But in such cases the indication for the exclusion of malignant disease is more urgent than in the asymptomatic cases. To exclude the possibility of cancer of the endometrium it is necessary to make a study of the endometrium from all parts of the endometrial cavity. The classical method is dilation and curettage. A properly performed suction biopsy of the endometrium by the original technique or one of its many modifications often yields as much or more endometrium as does the conventional dilation and curettage. It has the advantage of being an office procedure and hence can be applied in a greater number of patients. In doubtful cases it can always be supplemented by dilation and curettage. If the endometrial study reveals the changes characteristic of an ovarian failure, it certainly is permissible to try hormone therapy before resorting to surgery or radiation.

Radiation destroys the reproductive ovarian and menstrual functions. It does not abolish sex function but impairs it in the same way as does age. The classical contraindications to irradiation are any fibroid occurring in a woman under forty years of age, unless surgical treatment is contraindicated. If the diagnosis is questionable and the fibroid cannot be differentiated with certainty from an ovarian cyst, if the fibroid is larger than a fetus of three and one-half months, the presence of an old complicating pelvic inflammatory disease, a complicating ovarian neoplasm, a pedunculated submucous fibroid, a submucous fibroid, or a large pedunculated subserous fibroid when there is a history of rapid growth, when abdominal pain is present, if some other condition requires surgical relief.

Some surgeons appear satisfied with the

results of radiation, but in general the tendency of most is away from radiation because of its failure in the occasional patient and because of its destructive effect on function as well as the retention of the malignant potential of the uterus. The occurrence of malignant change seems a little higher in those patients having a fibroid in whom bleeding was present. In spite of this tendency radiation is a valuable method and in a small number of patients can be used to advantage when surgery appears too risky.

Myomectomy is the operation of choice when it is necessary to save the uterus for reproduction, or for sentiment. It is a remarkably successful operation and is applicable to a much higher percentage of patients than is generally believed. It can be practically bloodless by using a catheter as a tourniquet around the cervix and clamping each infundibulopelvic ligament with rubber shod intestinal clamps. The risk is about the same as for hysterectomy. There is some chance of recurrence. Bonney, in 293 cases, had a recurrence in nine or 2.3 per cent. He attributed his success to a very thorough removal of all fibroid seedlings. Others report a higher recurrence rate. In Bonney's opinion the seeds of fibroids are laid down most commonly between the ages of twenty-eight and thirty-three years. In some patients this occurs much earlier. After the age of thirty-three the chances of new seedlings diminish, and after thirty-six they are practically nonexistent. In Bonney's series of those women married and desirous of children, 88 per cent conceived after the operation.

In patients requiring surgery in whom the reproductive function is not important, hysterectomy is the treatment of choice. After the operation ovarian function is about the same as before. The sex function is unchanged and the local condition has been removed. If the operation is restricted to those patients in whom it is properly indicated, it produces the most satisfactory results.

ECTOPIC PREGNANCY

In extrauterine pregnancy, more commonly called ectopic pregnancy, the implantation of the ovum is outside the uterine cavity. The tube is the most common site, most frequently in the ampulla, next, in the isthmus and, lastly, in the interstitial portion of the tube. In rare cases the implantation is in the abdominal cavity or on the ovary.

In general, any obstruction to the passage

of the fertilized ovum through the tube favors the development of ectopic pregnancy. Theoretically the ovum could be too big to pass through the tube. In this connection an adhering membrana granulosa, rapid cell division and growth of the ovum during external migration (transperitoneal migration of the ovum to the contralateral tube) could be mentioned. Recent reports mention a psychogenic mechanism producing tubal constriction through an autonomic dysfunction. Actually the common cause is obstruction in the tube and this results from salpingitis. Since the advent of the antibiotics, partially occluded tubes with resulting ectopic pregnancy are the most common causes. Inflammation produces adhesions of tubal folds with pocket formation, strictures and destruction of cilia. External adhesions interfere with peristalsis and produce disturbances of motility. There are also congenital causes, such as double lumina, accessory ostia ending in blind sacs and tortuous infantile tubes. All of these are etiologic factors. Rarely a fibroid will obstruct or constrict the tube in its interstitial portion.

There is no well formed decidua in the tube and for this reason the tube is anatomically unsuited for nidation. In ectopic pregnancy when the ovum comes to rest it burrows into the tubal mucosa as it does in the endometrium but, in the absence of a protecting decidua, it comes to rest directly on the muscle layer adjacent to or surrounded by large blood vessels. It quickly erodes into the vessels and hemorrhage occurs. Sooner or later the tube ruptures either internally producing so-called tubal abortion, or externally with hemorrhage into the free peritoneal cavity.

By its endocrine action, the growing trophoblast causes the development of a uterine decidua and an enlarged, soft uterus. When trophoblastic activity from the uterus declines, the decidua begins to degenerate with a resulting flow usually reddish brown in color. Sometimes the decidua separates cleanly as a complete or partial cast of the uterine cavity. In the ovary the corpus luteum of pregnancy forms and functions throughout the life of the trophoblast. The trophoblast secretes the anterior pituitary like substance which initiates the above changes and is the basis for the biologic tests for pregnancy. The reactions to these tests remain positive as long as the trophoblast lives and remains in contact with the maternal circulation.

Ectopic pregnancy occurs in the white in

the ratio of one to 190 pregnancies and in the Negro in one to 130. The Negro's higher incidence results from the greater frequency of previous tubal inflammation. Other etiologic factors of importance are a history of abortion especially induced and of previous treatment for primary sterility.

The clinical picture of the disease varies greatly. There are two types: the classical in which the shock is immediate and profound and the atypical in which the symptoms develop more slowly.

Making the diagnosis of the classical type of ectopic pregnancy is easy. Typically the patient is a young woman who has had a delayed or missed period and develops a sudden severe lower abdominal pain with moderate vaginal bleeding. She collapses and faints. When examined, she presents a shocklike appearance, is very pale and has a low blood pressure. Such a picture suggests an intra-abdominal hemorrhage, the commonest cause of which is ectopic pregnancy.

In such patients four symptom groups stand out: the signs and symptoms of pregnancy, the pain, the vaginal bleeding, the evidence of internal bleeding. In the atypical patient these are not always readily recognizable and a determined search must be made to establish their presence or absence. The common error is to overlook entirely the possibility of ectopic pregnancy in the atypical case. The remembrance of the possibilities embodied in the C-A-F-E-E mnemonic eliminates this. Ectopic pregnancy is a diagnostic possibility which must be considered in all patients manifesting the symptoms of pain and bleeding. Another major difficulty in diagnosis is that the symptoms and signs vary according to the stage of the disease—that is, before rupture or after rupture.

Before rupture the usual signs and symptoms of pregnancy may or may not be present. As many as 30 per cent of the patients may have missed no period. The reaction to the pregnancy test is positive in the majority of the patients. Severe pain is not present. Sometimes there is unilateral discomfort. Vaginal spotting or scanty bleeding is common. The uterus is slightly enlarged with deep tenderness on the affected side and a tender soft mass may be present. Sometimes the ovary can be palpated as a structure separate from the mass. Fever and leukocytosis are absent. There are no constitutional signs of hemorrhage.

During rupture, pain is pronounced and is sharp, lancinating and unilateral. When a pain is followed by a period of relief as in

courses are open expectant treatment radiation therapy myomectomy and hysterectomy

Many fibroids require only expectant treatment. Observation should be considered when the tumor is asymptomatic, smaller than the size of a uterus that has been gravid for three months and the diagnosis is clear cut. It must be ascertained that the tumor is a fibroid and not an ovarian cyst. If observation is elected, coexisting malignant disease in the cervix and endometrium must be considered and its possibility eliminated by appropriate examinations. The rationale for observation is greater the nearer the patient is to the menopause.

In fibroids associated with bleeding, expectant treatment is also permissible under the above conditions. But in such cases the indication for the exclusion of malignant disease is more urgent than in the asymptomatic cases. To exclude the possibility of cancer of the endometrium it is necessary to make a study of the endometrium from all parts of the endometrial cavity. The classical method is dilation and curettage. A properly performed suction biopsy of the endometrium by the original technique or one of its many modifications often yields as much or more endometrium as does the conventional dilation and curettage. It has the advantage of being an office procedure and hence can be applied in a greater number of patients. In doubtful cases it can always be supplemented by dilation and curettage. If the endometrial study reveals the changes characteristic of an ovarian failure it certainly is permissible to try hormone therapy before resorting to surgery or radiation.

Radiation destroys the reproductive, ovarian and menstrual functions. It does not abolish sex function but impairs it in the same way as does age. The classical contraindications to irradiation are any fibroid occurring in a woman under forty years of age unless surgical treatment is contraindicated if the diagnosis is questionable and the fibroid cannot be differentiated with certainty from an ovarian cyst, if the fibroid is larger than a fetus of three and one-half months, the presence of an old complicating pelvic inflammatory disease, a complicating ovarian neoplasm, a pedunculated submucous fibroid, a submucous fibroid, or a large pedunculated subserous fibroid, when there is a history of rapid growth, when abdominal pain is present, if some other condition requires surgical relief.

Some surgeons appear satisfied with the

results of radiation, but in general the tendency of most is away from radiation because of its failure in the occasional patient and because of its destructive effect on function, as well as the retention of the malignant potential of the uterus. The occurrence of malignant change seems a little higher in those patients having a fibroid in whom bleeding was present. In spite of this tendency, radiation is a valuable method and in a small number of patients can be used to advantage when surgery appears too risky.

Myomectomy is the operation of choice when it is necessary to save the uterus for reproduction or for sentiment. It is a remarkably successful operation and is applicable to a much higher percentage of patients than is generally believed. It can be practically bloodless by using a catheter as a tourniquet around the cervix and clamping each infundibulopelvic ligament with rubber shod intestinal clamps. The risk is about the same as for hysterectomy. There is some chance of recurrence. Bonney in 293 cases, had a recurrence in nine, or 2.3 per cent. He attributed his success to a very thorough removal of all fibroid seedlings. Others report a higher recurrence rate. In Bonney's opinion, the seeds of fibroids are laid down most commonly between the ages of twenty-eight and thirty-three years. In some patients, this occurs much earlier. After the age of thirty-three the chances of new seedlings diminish, and after thirty-six they are practically nonexistent. In Bonney's series of those women married and desirous of children, 38 per cent conceived after the operation.

In patients requiring surgery in whom the reproductive function is not important, hysterectomy is the treatment of choice. After the operation, ovarian function is about the same as before. The sex function is unchanged and the local condition has been removed. If the operation is restricted to those patients in whom it is properly indicated, it produces the most satisfactory results.

ECTOPIC PREGNANCY

In extrauterine pregnancy more commonly called ectopic pregnancy, the implantation of the ovum is outside the uterine cavity. The tube is the most common site—most frequently in the ampulla, next, in the isthmus, and, lastly in the interstitial portion of the tube. In rare cases, the implantation is in the abdominal cavity or on the ovary.

In general, any obstruction to the passage

vives to term a short false labor occurs the placenta degenerates and the fetal circulation ceases. If the condition is recognized in time an operation may yield a living child.

A definite diagnosis of extruterine pregnancy calls for operation as soon as possible. Patients who have undergone a serious intra-abdominal hemorrhage present real surgical emergencies. As soon as blood replacement has been instituted, the patient is taken to the operating room. In the absence of an anesthesiologist, ether is the anesthetic of choice in our opinion. A left paramedian incision is made rapidly. The peritoneum is seen to have a darkish color due to the blood underneath and, when it is opened black blood literally gushes forth from the wound. The operator's hand rapidly explores the pelvis and locates the mass at the point of rupture. A long clamp is used to grasp the uterine end of the tube and utero-ovarian ligament. Within this clamp is held the anastomosis between the uterine and ovarian vessels. Laterally another clamp is used to grasp the ovarian artery. In very extreme cases, the infundibulopelvic ligament is the most readily accessible site for occluding this artery. However this means sacrificing the ovary on the affected side. In ordinary circumstances, blunt dissection separates the tubal mass from the ovary and the clamp is placed on the ovarian artery in the mesosalpinx. As the hemorrhage ceases, the patient rapidly improves. The removal of the tube can then proceed leisurely. It is well to remove all of the clotted blood which is readily accessible. In closing the wound, hemostasis receives particular attention.

In an abdominal pregnancy the placenta poses a special problem. If the child is alive, there must be an active circulation between the mother and the placenta. Since the site of the placental attachment cannot contract as in the uterus, hemorrhage will be severe. To avoid this, the placenta is left in situ. In patients in whom thrombosis of the vessels at the placental site has already occurred, the placenta can be safely removed.

The mortality rate in ectopic pregnancy under modern conditions is about 0.5 per cent in white women and substantially higher in colored women. Following ectopic pregnancy the chance of a second pregnancy in the opposite tube is about one in twenty. The chance of subsequent pregnancy is about even, but a fair number will terminate before viability. The chance of a living child has been given as one in three.

ENDOCRINE DISORDERS DUE TO OVARIAN FAILURE

An endocrine disorder resulting from ovarian failure can cause any type of menstrual disturbance. Two such different symptoms as menorrhagia and metrorrhagia often have ovarian failure as a common cause. In order to appreciate either of these symptoms we must understand the cause—ovarian failure.

The ovary has three known functions—the production of ova, estrin and progesterone. Ovarian failure is the inability to fulfill one or all of these functions in a normal manner.

There are two forms of ovarian failure—intrinsic or primary and extrinsic, or secondary. Primary or intrinsic, ovarian failure results from aplasia, destruction by neoplasms, surgical removal, inflammation and aging. In secondary or extrinsic, ovarian failure the primary cause is outside the ovary and is either in the pituitary or mediated through the pituitary. Diseases affecting the ovary indirectly by means of the pituitary are those which affect the thyroid gland, diseases in other endocrine glands such as the pancreas or adrenal and constitutional diseases such as tuberculosis, or malnutrition.

Ovarian failure follows a rather constant pattern. First there is a diminution in progesterone production with normal or slightly diminished estrin production. Next, the progesterone production falls with estrin production continuing in a slightly diminished cyclic fashion or in an acyclic fashion. In the latter the level of estrin production may never reach a value equal to the top or the bottom values of the normal curve but continues with only slight fluctuations at some level in between. In such subjects, the monthly excretion of estrin may be greater than in the normal cycle. This is so-called hyperestrinism. Lastly as the failure progresses, estrin production declines and gradually reaches the menopausal level. There are thus three degrees of ovarian failure—progesterone deficiency with mild estrogen deficiency, progesterone failure and estrin failure. These are respectively known as first, second and third degrees of ovarian failure.

In general the symptoms of ovarian failure are progressive in the older groups. In the younger age groups, compensation or remission may occur. Both primary and secondary ovarian failure exhibit a diversified group of symptoms which are identical

colic it is significant. Vaginal bleeding is present and the discharge is a brownish red without clots. Intra-abdominal bleeding occurs, recurring or increasing with each pain paroxysm. Fever and leukocytosis are present. Shocklike symptoms occur frequently. The examination often reveals an exquisitely tender mass to one side or behind the uterus.

Following rupture there is usually an attack of agonizing, lancinating pain on one side accompanied by an internal hemorrhage and followed by nausea, vomiting, fainting, collapse and shock. The pulse is rapid and feeble, the skin pallid and the breathing shallow. Air hunger and anxiety may be present. The veins are collapsed. The abdomen is distended and very tender. In the cul-de-sac, there is extreme tenderness and a mass of clotted blood. Sometimes the latter is difficult to recognize on account of the tenderness and the rather soft nature of the cul-de-sac. Rectal examination helps a great deal and should never be omitted. In patients with massive hemoperitoneum, shoulder pain due to irritation of the diaphragm may be present. Later painful defecation may be noticed and once in his lifetime the examiner may see a bluish black discoloration around the navel (Cullen's sign).

The demonstration of an intra-abdominal hemorrhage establishes the diagnosis with reasonable certainty. The blood findings, cul-de-sac puncture, colpotomy and culdoscopy are all important and helpful.

After a large intra-abdominal hemorrhage there may be little immediate change in the blood picture. But, in time dilution of the circulating blood occurs with a fall in the hemoglobin value, packed cell volume and red blood cell count. In diagnosis the degree of anemia does not parallel the clinical signs of hemorrhage. The important fact is that the blood findings are slowly progressive. A progressive lowering of the hemoglobin value on repeated determinations is therefore, very significant. Another differential point is that the degree of anemia is generally greater than can reasonably be explained on the basis of the vaginal bleeding.

Examination with the patient under ether is a great aid to diagnosis. Often the tenderness is so extreme that a satisfactory examination is impossible in the unanesthetized patient.

A cul-de-sac puncture, using an aspirating needle has been most helpful and safe in our experience. It not only serves to estab-

lish the fact that blood is present, but sometimes reveals pus and thereby excludes the possibility of the presence of ectopic pregnancy. Now and then a small amount of blood is obtained which might have come from the extraperitoneal tissues. Such blood is red and clots readily in the syringe. Blood from an intra-abdominal hemorrhage is dark and, when squirted onto a piece of gauze, there are numerous little clots. Under the microscope the cells are seen to be crenated and rouleaux formation does not occur.

Posterior colpotomy also determines the presence of blood in the cul-de-sac and allows the entrance of an examining finger. Some continental surgeons consider this a most valuable aid in diagnosis.

Culdoscopy, a visual method, has found favor with some and certainly has merit in the doubtful cases. When blood is present, inspection is difficult or impossible and, therefore in patients having an intra-abdominal hemorrhage culdoscopy offers little advantage over simple puncture. Perhaps its greatest value is in revealing the true nature of the disease process when ectopic pregnancy is not present.

Dilation and curettage can give valuable information in doubtful cases. The finding of a well marked decidua without chorionic elements is presumptive evidence of ectopic pregnancy. Before employing dilation and curettage it is wise always to consider the possibility of intrauterine pregnancy since it coexists with ectopic pregnancy in a small percentage of cases.

Ovarian pregnancy is a rare form of extra-uterine pregnancy. The symptoms, signs and treatment of ruptured ovarian pregnancy are essentially the same as those of ruptured tubal pregnancy. Hemorrhages from other causes can occur from the ovary but a severe intra-abdominal hemorrhage of ovarian origin always raises the possibility of an ovarian pregnancy. Microscopic examination of the ovary reveals chorionic villi and this confirms the diagnosis.

Primary abdominal pregnancy with implantation on a peritoneal surface does occur. A secondary form occurs when a live embryo escapes by rupture or abortion from its primary location in the tube or ovary and implants itself on the peritoneum. Such embryos usually are abnormal and die long before term. They undergo degeneration or lithopedion formation. As a result of the latter many bizarre circumstances arise. In those rare instances in which the fetus sur-

useful clinical asset to all who perform abdominal surgery.

In third degree ovarian failure the hot flash is the most important and characteristic of the neurogenic symptoms. At first it occurs only a few times daily and is limited to the head and neck. Later it becomes more frequent and may involve the whole body. Often during sleep, the flash produces so great a sensation of heat that the bed clothes are removed to be followed in a short time by chilling, and awakening. The incidence of hot flashes is fairly well paralleled by the amount of follicle-stimulating hormones in the urine. Following estrin administration the follicle-stimulating hormones decline and the flashes are not so frequent. Thus, the level of excretion of the follicle-stimulating hormone is influenced by the level of estrin. An excess excretion of follicle-stimulating hormone is perhaps the most valuable test in the diagnosis of primary ovarian failure.

Sweating is frequently associated with hot flashes and often causes the patient much discomfort. Smothering is another frequent symptom and always arouses a suspicion of ovarian failure. Many other neurogenic symptoms occur from time to time. Among these are headache, fatigue, cold sensations, tingling and tinnitus.

Ovarian failure causes all types of menstrual disturbances. As a general rule, the minor disturbances of menstrual interval and flow occur in first degree failure. The severe types of bleeding are seen in the second degree failure. Infrequent menstruation and amenorrhea are characteristic of third degree failure. It is well established that there is no absolute correlation between the degree of failure and the type of symptoms.

In second degree ovarian failure, the endometrium is generally hyperplastic and often the glands are irregularly dilated, giving a Swiss-cheesecake appearance to the microscopic section. This glandular cystic hyperplasia, or Swiss cheese hyperplasia, is a very important characteristic of second degree ovarian failure and usually is associated with marked irregularities of the interval and of the flow. Not infrequently, bleeding is preceded by a short period of amenorrhea and this often gives rise to the suspicion of an abortion. Confirming this belief is a marked softening and slight dilatation of the cervix. Lacking is the pain which is so characteristic of abortion. The fact that painless bleeding is the rule with ovarian failure is important in diagnosis.

The general appearance of the patient is normal when primary ovarian failure occurs after adolescence. In those in whom the failure occurred before or during adolescence there is a rather definite tendency toward an increase in the length of the arms and legs and a span—the distance from finger tip to finger tip of outstretched arms—which is greater than the height. The abnormal length of the extremities results from the gonads' inability to exercise its effect on epiphyseal closure. This disproportion of the extremities to the trunk can also occur in women with anterior pituitary dysfunction and in normal women who reach sexual maturity somewhat late. If it is associated with a menstrual disturbance it is a sign of great value especially so if the disturbance dates from adolescence. In the adolescent with primary ovarian failure, the muscular system is frequently deficient and there is a tendency toward leanness which is difficult to overcome by treatment. The blood pressure is often low, the systolic pressure ranging from 90 to 100, while the pulse frequently is rapid. The breasts may be underdeveloped and the uterus has a small body and a long cervix. This gives a ratio of the cervix to the body of the uterus which is greater than the normal cervix body ratio of 1:2.

In third degree failure there is atrophy of the vulva and vaginal mucous membrane and the trophic state of these parts gives a good idea of the amount of estrogen available to the genitalia. A more accurate estimate of the hormones available to the genital tract is obtained by studying the endometrium. In primary ovarian failure, a low estrin level causes an increase in the level of follicle-stimulating hormones and this is considered to be a specific diagnostic test.

The diagnosis of primary ovarian failure is made in two steps. First, the presence of an ovarian failure is determined and, secondly, this is demonstrated to be primarily ovarian. The first step is comparatively easy. Either a functional menstrual disturbance, significant trophic changes in the vagina or changes in the histologic structure of the endometrium are all certain indications of a lowering of ovarian function. The second step consists in determining if the failure is primary or secondary. This is somewhat more difficult and often involves problems in general medicine and endocrinology. From a practical standpoint, the single most important differential test is the determination of the follicle-stimulating hormone.

in respect to those symptoms arising from a lack of ovarian activity. The secondary form, however, also exhibits in addition the symptoms of the primary pituitary disease or of other diseases which result in a decreased pituitary function. Secondary ovarian failure, therefore, has all of the features of the primary form but, in addition, has grafted thereon the symptoms of another endocrinopathy.

The symptoms of ovarian failure fall into five groups—sexual, reproductive vaginal, nervous and menstrual.

Ovarian failure is a consideration in all problems relating to sexual behavior in the female. However it rarely is the chief factor. Ovarian hormones are an indispensable factor in the development of the normal female. However in spite of normal ovaries the psyche and sex impulse may be and often are distorted by psychologic and environmental factors. Hormones also sensitize the parts and the psychic centers, but a normal sex experience can occur in previously normal women who have been deprived of their sex hormones by bilateral oophorectomy. In spite of this it is obvious that the hormones do play a role because sexual activity and response are not the same in the older woman as in the young. The evidence seems to indicate that a lack of sex hormones, in previously normal women, produces changes in libido and response somewhat similar to that occurring from aging.

Other necessary factors which must be considered in relating ovarian failure to a sexual symptom are the normalcy of the parts; the absence of pain, the proper functioning of the nervous system and the proper psychic and sexual conduct of the male.

Normal ovarian function is necessary for normal ovulation and for the proper development of the endometrium. If the development of the endometrium is faulty as in first degree failure, there is an increased likelihood of abortion, low implantation of the placenta and placenta previa. In second and third degree ovarian failure there is no ovulation and consequently sterility results. The development of a well marked progestational endometrium is the most practical and accurate method of determining ovulation in the human being. The endometrium is best obtained by the biopsy method just before the onset of menstruation. This in addition to a test of tubal patency and a determination of the sperm count, is indispensable in any study of sterility.

At puberty the vaginal mucous membrane acquires an increased resistance to infection as the result of estrogenic stimulation. The application of this fact is useful in the treatment of certain discharges. As the patient reaches the menopause the vagina gradually undergoes a process of involution during which the epithelium loses its rugae, becomes thinner and is more prone to infection. Obliteration of the fornices and narrowing of the introitus gradually occur as the process continues, symptoms of stinging and burning occur with the development of discharge (senile vaginitis). Not infrequently there are adhesions between the anterior and posterior walls and, sometimes, the process goes on to almost complete vaginal obliteration. Bleeding is a common accompaniment of senile vaginitis and this is perhaps the most common form of postmenopausal bleeding. It should never be forgotten however that the presence of senile vaginitis does not exclude the possibility of cancer of the body of the uterus. Many endometrial cancers are overlooked because of the simultaneous occurrence of the two diseases.

The nervous symptoms of ovarian failure are both psychogenic and neurogenic. They vary in their intensity according to the personality of the patient and the severity of the failure. Most women with a well balanced personality have relatively few symptoms even from complete failure. Others suffering with various personality defects have a variety of unpleasant and often severe symptoms from only mild failure. In such women, many of the symptoms have a quality which suggests the diagnosis of a primary psychoneurosis and the evaluation of such symptoms can be extremely difficult. Often it is best to reserve judgment until the results of specific therapy can be observed or the psychopathologic state can be demonstrated. Symptoms remaining in the face of adequate therapy and unexplained by a careful general examination can reasonably be considered as psychogenic in origin.

Psychosomatic symptoms pointing to an abdominal viscus are frequent and confusing. Pain is present and frequently leads to an ill-advised operation. In one series of cases of ovarian failure 43 per cent of the patients had had one or more abdominal operations. Most of these patients were unrelieved and they again sought and found further surgical intervention. At long last, their troubles were attributed to adhesions. The recognition of such cases is a highly

conjugated estrogens (Premarin) is given intravenously and the dose is repeated every eight hours until the bleeding is checked. The first two or three doses should be supplemented by the intramuscular injection of 20 to 50 mg of progesterone in order to insure a more complete desquamation of the abnormal endometrium when withdrawal bleeding occurs then cyclic therapy follows as in the previous plan.

Dilation and curettage is often used to establish the diagnosis and to produce temporary hemostasis in bleeding which is refractory to treatment. In a fair proportion of women, the operation produces a remission of symptoms and no further treatment is necessary. However this is not the rule and in a period of several months the symptoms recur. For this reason we usually follow the curettage with endocrine treatment.

Hysterectomy is frequently used in the treatment of endocrine bleeding, but in many cases the surgeon does not know the bleeding is of endocrine origin until he finds what he thinks is a "bleeding fibroid," far removed from an endometrium exhibiting glandular cystic hyperplasia. For the deliberate use of hysterectomy the bleeding should be severe endocrine treatment unavailable or impractical and the reproductive function of little or no value. The advantages of hysterectomy are the definitive nature of the cure with a low mortality and morbidity the preservation of the patient's remaining ovarian function, which is often adequate for a normal sense of well being, and the preservation of normal sexual function.

Its disadvantages are those incident to any operation, as well as destruction of the reproductive and menstrual functions. The psychologic effect of hysterectomy has been much discussed and it is clear that when the patient does not willingly accept her postoperative state, a bad result is inevitable. Fortunately most patients with severe bleeding gladly accept their postoperative state and are extremely pleased with the operation.

Radiation by externally applied x ray or by internally applied radium, is useful for those bad risk patients in whom the reproductive and ovarian functions can be sacrificed. There are still some gynecologists who prefer radiation to hysterectomy for endocrine bleeding. It is certainly much less an ordeal and does have its advantages. Its disadvantages are its destructive effect on the ovarian and sex functions. For example

following radiation a patient of forty two years of age rapidly reaches a sexual age of perhaps sixty two years. This does not necessarily mean that intercourse is unpleasant or that orgasm is impossible but it is not like it was. Radiation owes its curative effect on endocrine bleeding to the abrupt transformation of partial ovarian failure to complete ovarian failure. Following this there may be severe nervous symptoms. In overcoming these by hormonal means there is always the possibility of inducing worrisome bleeding from the uterus. Further the cancer risk of the retained uterus seems to be greater.

THE MENOPAUSE

This is a primary type of ovarian failure normally occurring at the end of the reproductive period. It occurs as a normal physiologic change but has all of the signs and symptoms of primary ovarian failure. Some women have severe symptoms, but in most the symptoms are mild and require no treatment. The nervousness incident to the menopause comes at a time when a woman's responsibilities are often greatest. In addition, it focuses the mind on the aging process and its consequences. To many it appears that life can never be the same. Actually, the years after the menopause can be and frequently are the best of a woman's life. Reassurance and perhaps mild sedation are usually all that are necessary. In some patients estrogenic therapy may be indicated. Symptoms which persist after adequate estrogenic therapy cannot rightfully be attributed to the menopause and must be explained and treated on some other basis.

RELAXATIONS

Defects in the pelvic supporting structures present as anterior vaginal relaxation or cystocele posterior vaginal relaxation or rectocele and uterine descent or prolapse. These lesions may occur separately or in any combination or degree. Each of these lesions may be classified according to the degree of relaxation present. For instance, in uterine prolapse if the cervix lies within the vagina it is called first degree if the cervix protrudes from the vagina it is second degree if the uterus lies outside the pelvis it is third degree prolapse.

Certainly the trauma incident to repeated childbirth is the most important etiologic factor. However certain individuals appear to have relaxation after only mild trauma

To be sure many problems in differential diagnosis arise. For example in a patient with a sexual disturbance ovarian failure is only one of several causes and the possibilities of these must be excluded. Likewise in patients with bleeding, one must first make a differential diagnosis of the cause of bleeding.

The successful treatment of ovarian failure depends upon individualization of the patient and the determination of her functional needs. An accurate diagnosis, elimination of constitutional disease and the intelligent choice of endocrine surgical or radiologic treatment.

In extrinsic or secondary ovarian failure the treatment consists of measures directed at the primary cause as well as at the ovarian failure. In hypothyroidism with secondary ovarian failure for example the treatment would be that of hypothyroidism plus that of ovarian failure.

The treatment of ovarian failure at the present time is largely a substitutional form of therapy. A total or partial deficiency of either or both of the ovarian hormones is corrected by prescribing the deficient hormone in an amount approximately equal to the deficiency at the correct time in the cycle. Under normal conditions a drop in the amount of estrin and progesterone or of progesterone initiates menstruation, while a rise in the level of estrogen terminates menstruation. This is known as the withdrawal theory of menstruation and, while it does not completely explain the phenomena, it is a most valuable therapeutic tool as it enables us to start and stop the flow under most circumstances.

Generally endocrine amenorrhea is a symptom of second or third degree ovarian failure. In second degree failure there is an adequate acyclical estrin production with a total lack of progesterone. According to the withdrawal theory it is only necessary to produce a rise and fall in progesterone to induce menstruation. This is easily accomplished by the administration of 20 mg. of progesterone for three to five days, or of Norlutin, 10 to 20 mg., for five days. Cessation of the medication causes a fall in progesterone and initiates menstruation. In third degree failure there is a total lack of progesterone and an inadequate estrin secretion. Menstruation can be induced by first raising the hormonal level by the intramuscular injection of 20,000 I.U. (2 mg.) of a microcrystalline suspension of estrogenic substances and 20 mg. of progesterone for a

period of five or more days. By the oral route 5 mg. of Premarin and 10 mg. of Norlutin are often effective. When the medications are discontinued, the hormonal level falls and menstruation follows. Should the period fail to appear larger doses of hormones and longer periods of treatment should be used in subsequent cycles.

For abnormally long menstrual cycles, treatment is started on the seventeenth day after the onset of bleeding and consists of raising the hormonal level by the daily oral addition of 1.25 or 2.5 mg. of conjugated estrogens and 40 mg. of anhydrohydroxyprogesterone (Pranone)—orally active progesterone derivative—for a period of seven days. The fall in the hormonal level following cessation of treatment will usually induce menstruation at or near the normal time.

Abnormally short menstrual cycles may be offset by additive therapy. For example, in patients with twenty-one-day cycles, 1.25 or 2.5 mg. of conjugated estrogens plus 40 mg. of anhydrohydroxyprogesterone are given orally daily from the fourteenth to the twenty-fourth day after the onset of the period. The usual fall in the hormonal level at the seventeenth or eighteenth day will be delayed by the addition of these hormones thus prolonging the interval.

In severe menorrhagia or metrorrhagia, the flow can usually be controlled by the daily injection of 25 mg. of testosterone propionate and 20 mg. of progesterone for five days. The addition of 20,000 I.U. (2 mg.) of a microcrystalline solution of estrogenic substances adds to the efficacy of the therapy. At the completion of therapy the bleeding will either be greatly diminished or will have ceased. After a latent period of two to four days, the hormonal withdrawal following the cessation of the injections induces a normal menstrual period with a desquamation of the previously abnormal endometrium. This sometimes is spoken of as a medical curettage. Following this, Hembles cyclic therapy is in order: 1.25 to 2.5 mg. of conjugated estrogens is given from the fifth to fourteenth day after onset of the withdrawal bleeding, or the onset of the normal period and this is increased to 2.5 or 5 mg. from the fifteenth to twenty-fourth day. This is supplemented by the addition of 40 mg. of anhydrohydroxyprogesterone or 10 mg. of Norlutin given orally from the seventeenth to twenty-fourth day.

In patients with very severe bleeding requiring immediate hem 20 mg. of

HYSTERECTOMY

Certainly no discussion of diseases of the female genitalia would be complete without mention of the operation which unfortunately is so often necessary in combating these diseases. This operation is of course, hysterectomy.

Thus far the main purpose has been to present the various symptom complexes which arise in the field of gynecology and to proceed from there to a probable diagnosis. The functional approach and individualization have been emphasized. In many of the diseases the most logical treatment is hysterectomy. But one should remember that there are no absolute indications for hysterectomy. While it is true that in any series of hysterectomies one may list the conditions for which the operation was performed, the reverse—that is, that these conditions are indications for hysterectomy in every case—is not true.

This discussion will be limited to two types of hysterectomy—total abdominal and vaginal. In situations which require the abdominal approach, the total operation is the one of choice. Situations do arise in which the subtotal operation is necessary but any one familiar with the total operation will rarely find a situation in which the subtotal is necessary. Radical hysterectomy is regaining considerable popularity in the treatment of cancer of the cervix and endometrium.

Total abdominal hysterectomy will be the operation of choice in a large percentage of women in whom removal of the uterus is indicated. It is axiomatic that to perform this operation properly the surgeon must have a thorough knowledge of the anatomy involved. Too often however this knowledge is lacking, as the all-too-frequent ureteral, vesical and rectal complications testify. Perhaps the most useful knowledge is that of the supporting structures of the uterus. Visualize the uterus and vagina as relatively flexible structures immobilized within the pelvis by ramifications of the endopelvic fascia arising from the lateral ground bundles. Remember also that these layers of fascia decussate to envelop the bladder and rectum. The operation should be performed in such a fashion that each step makes the succeeding one easier. The supporting structures of the uterus are, therefore attacked from above downward, allowing the uterus to be mobilized upward.

The ability to perform a vaginal hysterectomy well is essential to the armamentarium of every pelvic surgeon. Frequently the

presence of associated vaginal relaxation, or uterine descensus makes this the method of choice. Here again the operation proceeds by a successive attack on the supporting structures but in the reverse order.

A knowledge of the effects of hysterectomy on the general and pelvic functions is of paramount importance in the selection of subjects for the procedure. What is the effect of hysterectomy on life? We know that it can be carried out with minimal mortality. Collected reports indicate that under good conditions the mortality is from two to five per 1000. Even this small figure is improved by the cancer prevention in those who have the operation. The general health of the patient improves in proportion to the degree of disability existing prior to the operation. Only in poorly selected patients and those suffering from severe complications is the general health impaired. The pelvic functions of menstruation and reproduction are lost. In most patients, this is a welcome relief but the loss will be a severe liability in certain women if they are not properly conditioned. Ovarian function is probably not affected if the ovarian circulation is not embarrassed. The sexual function is generally unimpaired and, occasionally is improved. Vaginal support is not greatly affected by hysterectomy as the intrinsic support of the vagina is not disturbed. Certainly any coexisting vaginal relaxations should be repaired at the time of hysterectomy.

PELVIC MASSES

In the diagnosis of pelvic masses, the usual methods apply. After taking the history and making the examination, the physician integrates the data and considers the most logical possibilities. In the differential diagnosis he considers all reasonable possibilities and excludes as many as possible by further examinations and special studies. By this process, he is able to arrive at a working diagnosis in most cases. In some patients the diagnosis may still be uncertain and an exploratory operation may be necessary.

Arriving at a working diagnosis of masses presumably arising from the female internal genitalia can often be easy and straightforward, if the following approach is used. The first question to be answered is: Is the mass genital or extragenital? In answering this question, it is well to remember that a diffuse abdominal swelling has five possible causes. They are sometimes spoken of as the five F's. They are fat, fluid, feces, flatus

and this must be attributed to some defect in the tissues themselves. Occasionally these lesions occur as congenital defects and complete prolapse in a maiden lady is sometimes seen.

The symptoms of vaginal relaxation will vary depending on the degree and character of the relaxation. One of the commonest complaints is a sensation of pressure, or bearing down, in the pelvic region. Patients frequently volunteer the information that they feel as though their insides are coming out. This symptom is usually worse on standing. Patients with rectocele complain of protrusion of the posterior wall of the vagina and inability to defecate unless the rectocele is manually reduced. Those with cystocele and urethrocele also complain of protrusion from the vagina. Varying degrees of frequency dysuria, urgency and incontinence may be present.

Incontinence must be carefully evaluated, as it may require special attention. We have to know exactly what the situation is. Does the patient leak urine only occasionally, on severe straining? Does she require protection because of frequent leakage on moderate straining? Does urine leak continuously when she is in the erect position? Often there is associated posterior urethritis and the symptoms of this condition should be identified before proceeding with repair. Otherwise the patient may be dissatisfied because of persistence of symptoms which were attributed to relaxation, but which were in reality due to urethritis.

Patients with prolapse may complain of a low backache or bilateral low abdominal pain due to tension on the intra-abdominal supporting structures. Commonly the presenting complaint is of a gradually enlarging protrusion from the vaginal outlet. In long standing cases bleeding and ulceration occur from pressure. At times these lesions may simulate malignant disease and require biopsy although carcinoma and prolapse seldom coexist. At times the prolapse may be so severe as to produce urethral obstruction and hydronephrosis. The hydronephrosis usually regresses when adequate drainage is established, which should, of course, be accomplished before operative repair is attempted.

The pathologic state of these conditions is difficult to discuss because of the complex anatomy of the pelvic fascia. However this anatomy must be understood before the defects can be properly repaired. Perhaps the best discussion of this matter is given by

Pelham and Amreich who describe the connective tissue ground bundle. This lateral structure splits to envelop the bladder, vaginouterine canal and rectum with supporting fascia. It is defects in these fasciae which produce the relaxations described above.

In order to simplify the discussion, the anatomic similarity of a prolapse and a post-operative ventral hernia should be visualized. In the ventral hernia, a peritoneal sac protrudes through a defect in the transversalis fascia, rectus muscle and rectus fascia. In prolapse the structures involved are remarkably similar to those in the ventral hernia. The triangular ligament corresponds to the anterior rectus fascia, the levator ani muscles to the rectus muscles and the pelvic fascia to the transversalis fascia. Therefore, in prolapse there is a peritoneal sac containing the uterus protruding through a defect in the pelvic fascia, levator muscles and the triangular ligament.

The final result of both prolapse and ventral hernia is a protruding mass containing a hernial sac and covered by squamous epithelium. Surgical cure of these lesions follows identical lines. In hernia, the structures are dissected out, the sac is removed and closed, the transversalis fascia sutured, the muscle is brought together and the rectus fascia closed. In prolapse the same principles are used. The dissection of the structures exposes the defect in the pubocervical fascia under the cystocele and in the rectal fascia, over the rectocele. The removal of the sac often requires vaginal hysterectomy as the apex of the sac corresponds with the circumferential attachment of the peritoneum to the uterus. The peritoneal portion of the uterus is part of the contents of the sac, while the extraperitoneal portion acts as a hard wedge to push aside the structures in front of the advancing hernia. After the peritoneum is closed, the cardinal and uterosacral ligaments are approximated, then the defect in the pubocervical and rectal fasciae is closed—this corresponds to closure of the fascial layer in the repair of a ventral hernia. Suturing the levators together restores the muscular component of the perineum. The triangular ligament is the fascial part of the perineum comparable to the anterior rectus fascia and suturing of this layer completes the operation except for skin closure. When preservation of sexual function is important, the procedure is complicated by the necessity of maintaining an adequate vaginal outlet.

fingers are brought together and placed just in front and above the os. The right hand is fully pronated and presses the uterus against the fingers of the left hand the uterus is thus caught between the palmar surfaces of the fingers of both hands and can be palpated easily and accurately. When the impact of the uterus against the anterior abdominal wall is not visible and the anterior surface of the lower uterus cannot be felt by the vaginal fingers, palpation is started at the promontory of the sacrum and by a series of dipping motions it is continued downward until the abdominal fingers transmit an impulse to the fingers beneath the cervix, then the fingers of the vaginal hand are rearranged and the uterus is palpated as has just been described. If the uterus cannot be felt posteriorly in the cul-de-sac or found anteriorly a partially filled bladder is suspected and the patient is catheterized before it is assumed that the uterus cannot be outlined.

Is the mass adnexal? Palpation of the uterus separate from the mass and the exclusion of an extragenital mass definitely identify a lateral mass as adnexal. The corollary that palpation of an ovary and the exclusion of an extragenital origin identify a mass as uterine is also true except in certain rare instances of ectopic gestation, hydatid cysts and discrete tubal masses.

It is impossible to palpate the normal tube. To palpate the ovary the vaginal fingers are placed in the lateral vault of the vagina and pressure with the vaginal hand is applied firmly inward toward the sacrum and outward toward the side wall of the pelvis, while the abdominal hand is pressed down, so that the tips of the abdominal fingers come close to the tips of the vaginal fingers. In this position the tips of the fingers partially encircle the ovary if it is in the normal position and, if the ovary is not felt, the same relative position of the fingers is maintained while the hands move slowly downward toward the pubis. By so doing, the fingers pass over the ovary and can feel it if the fingers are fairly closely approximated.

With uterine masses one must differentiate between those conditions designated by the first four letters of our mnemonic PREMIO. These are pregnancy, retroversion, enlargement and myoma. If the mass is adnexal, the differentiation is between the conditions designated by the last three letters of the mnemonic. These are inflammation, ectopic pregnancy and ovarian

mass. Many times it is impossible to state definitely if the mass is uterine or adnexal. In such a situation one must make a differentiation between the various uterine and adnexal masses. In making this differentiation the history, the constitutional reaction, the findings in the blood and urine and the characteristics of the mass are considered. This information together with the condition of the cervix and the position of the mass, its size, shape, consistency, mobility and tenderness, usually gives the data necessary for establishing the diagnosis.

In pregnancy the cervix is soft and as the pregnancy advances toward term it gradually becomes effaced. In abortion, the cervix is soft open and may contain blood clots or pieces of placenta. In ectopic pregnancy, there may be slight softening of the cervix, but this is generally not well marked. Endocrine bleeding causes a softening and dilatation of the cervix similar to that occurring in abortion.

A central position of a mass suggests a uterine origin, a lateral position indicates an ovarian origin. However it is not unusual for an ovarian cyst to present in the midline and for a uterine tumor to be felt in a lateral position. Every now and then an experienced examiner may feel a lateral mass which has the characteristics of an ovarian cyst. At operation no cyst is found and the uterus is the site of a normal pregnancy. The palpatory illusion of a cyst resulted from the fact that the relaxed uterine isthmus allowed the fundus to fall into a lateral version. When the uterus contracted, it returned to the former midline position. Any of the genital masses can present in the cul-de-sac. The most common is the retroverted fundus.

The size of a mass is of some diagnostic importance, especially if considered in relation to the history. Perhaps, in a suspected pregnancy there is a two months period of amenorrhea with a uterine mass the size of a four and one-half months gestation. This would indicate that the suspicion of pregnancy was incorrect or that there was some additional cause of uterine enlargement such as a myoma. In ovarian cysts, size has considerable diagnostic significance. The extremely large cysts, for example are almost always of the pseudomucinous variety. In young women with menstrual disturbances, cysts less than 4 cm. in diameter are usually follicle cysts. Ectopic gestation and pelvic inflammation form masses which are sometimes large enough to present supra-

and fetus. The usual physical examination of the abdomen elicits the data obtained by inspection, palpation, auscultation and percussion. The pelvic examination extends the scope of the examination and adds much useful information. When abdominal fluid is encapsulated, and the lower pole of the mass can be felt in the pelvis the tumor is almost certainly ovarian in origin. Overdistention of the urinary bladder simulates a pelvic tumor. If one recognizes the possibility and catheterizes the bladder mistakes will not occur. When free fluid is present it can often be related to a pelvic mass such as an ovarian carcinoma or to secondary carcinomatous nodules. An adnexal mass with free fluid suggests the possibility of tuberculous peritonitis or Meigs syndrome. Feces in the bowel cause abdominal distention and fecal masses in the pelvis sometimes resemble tumors. A fecal bolus can be differentiated from a tumor by its putty like consistency and its tendency to pit or dent on pressure. In doubtful cases a good cathartic eliminates the mass and allows a more satisfactory subsequent examination. Caseous distention of the abdomen may be from a functional cause such as air swallowing, or from an organic cause such as intestinal obstruction, tumor or inflammation. Often times the inflammation is of adnexal origin and involves the small bowel.

The large bowel is rarely if ever obstructed as the result of extrinsic pressure by tumors. A left sided mass associated with a large bowel obstruction is almost certainly therefore the result of either carcinoma or diverticulitis. If there is no large bowel obstruction, masses due to diverticulitis or carcinoma of the sigmoid or cecum, can mimic adnexal masses. If an operation is elected, a mistake of this kind can be embarrassing to the surgeon and dangerous to the patient. This is especially so if the surgeon is not too well grounded in gastrointestinal surgery. For this reason, some surgeons make it a rule to examine the colon radiologically in patients with left-sided masses. Certainly this in conjunction with a good gastrointestinal history will eliminate most mistakes of this kind. While ectopic kidney is a rare lesion, it will occur from time to time and for the sake of completeness it is well to keep the possibility in mind.

In practice, the elimination of an extragenital mass is largely dependent upon recognizing the possibility of an extragenital mass in diagnosis. Is it appendix, cecum, bladder, kidney or sigmoid? These are

routine questions which should never be overlooked. When these questions are answered in the negative one may safely assume the mass to be of genital origin.

Is the mass uterine? This is the next question and the answer depends in great part upon the vaginoabdominal examination. As soon as he inserts the finger the experienced examiner immediately begins to form impressions which gradually crystallize into definite opinions. Light palpation with one finger reveals a tremendous amount of information. From it, one can determine the characteristics and consistency of the cervix, its degree of dilatation and effacement and the presence of masses in the cervical canal. The finger gently sliding from the os up over the anterior surface of the cervix, may establish the presence of a continuity with the anteverted fundus or laterally the finger, reaching the fornix, comes upon an area richly rewarding to the gentle touch. In this area, the lower pole of many masses presents. Comparing the height of the vault yields much information. Detecting a relative diminution in the height of a vault frequently means trouble above. Posterior to the cervix is the area where light palpation is most valuable. As the finger passes along the posterior cervix to the cul-de-sac, it may encounter the buckshot nodularity of endometriosis, the induration of carcinoma or the lower pole of any of the pelvic masses. The most common of these is the retroverted uterus.

For testing the mobility of the uterus, two fingers are best. Most examiners use the first two fingers of the left hand in the vagina and the right hand on the abdomen for vaginoabdominal palpation. The initial position of the vaginal hand is midway between pronation and supination, the index finger points slightly to the patient's right, while the second finger points a little to the patient's left, thus forming a little V in which the cervix rests. From this position, the vaginal hand can elevate the uterus by exerting upward pressure on the cervix. Often one can see the impact of the uterus against the anterior abdominal wall. This is a great help in determining the placement of the abdominal hand, the most advantageous position being just at the upper margin of the point of impact. When the right hand is properly placed and presses down upon the uterus, the impulse is transmitted to the fingers of the vaginal hand. To bring the hands into the proper position for palpation of the uterus the left hand is supinated, the

tient having a perforated appendix with pelvic peritonitis. In this condition the fever may be high, the pulse rapid and the tenderness bilateral. The history is of the greatest value, especially so if at the onset the original localization was in the right lower quadrant or if there had been a previous attack of either salpingitis or appendicitis. In some cases, a differential diagnosis is impossible and it is better to open the abdomen than to allow possible appendicitis to progress. Before doing so, it is always helpful to re-examine the pelvis with the patient under anesthesia. With the patient asleep and tenderness and rigidity abolished it is possible to feel adnexal masses that heretofore were not palpable.

Other common diseases which must be considered in the differential diagnosis are urinary tract infection, twisted ovarian cysts and ectopic gestation. Of these the most difficult to differentiate is ectopic gestation. In this differentiation cul-de-sac aspiration and examination with the patient under anesthesia are often decisive.

There are two principles in the treatment of the patient having acute gonorrheal pelvic infection: first, treatment of the infectious process and, second, prevention of subsequent infections. Treatment of the primary infection is almost always successful with appropriate medication and adequate dosage. Penicillin, 300,000 units given intramuscularly daily for three to five days produces a dramatic effect. The oral administration of 200,000 units, three times a day for seven days, is also satisfactory. In resistant or difficult cases, the new broad spectrum antibiotics are useful. The sulfonamides are also effective, but less so than the antibiotics. Bed rest, hot douches and supportive treatment are helpful measures in severe cases.

It is now well established that a recurrence of symptoms following adequate treatment is caused by reinfection and not by an exacerbation of the original disease. Therefore, the prevention of subsequent attacks depends upon eliminating the source of the reinfection by either curing the infected consort or having the patient avoid him. In the case of the innocently infected wife the physician may have to exercise considerable tact and discretion in order to eliminate the erring husband's infection. The physician's obligation to the patient has not been fulfilled until this has been done.

In patients with neglected or untreated cases, a pelvic abscess may develop. This requires drainage, which is best accom-

plished through the cul-de-sac. Continuous drainage and obliteration of the abscess cavity depend upon the insertion of a rubber tube into the cavity. Generally the tube selected is of the type used for drainage of the common bile duct. This is far too small. A proper tube is as large as a finger. It is readily constructed from a chest tube. The tube remains in place until activity subsides and the cavity is obliterated. Generally this requires six or more weeks. Of course antibiotic therapy is continued throughout the febrile phase. The addition of cortisone hastens the absorption of the inflammatory exudate.

Chronic Gonorrheal Salpingitis. Repeated infections cause blunting and adhesions about the ends of the tubes with subsequent tubal obstruction. Infection persisting in the obstructed tube produces the well known "pus tube" or pyosalpinx. Not infrequently the fibrinated end of the tube becomes plastered against the ovary and the infection produces an abscess involving the ovarian stroma and the lumen of the tube. Unfortunately many patients return time after time with more severe and disabling infections until at last they become "pelvic cripples." Outstanding in these unfortunate women are one or more of the following complaints: sterility, dyspareunia, pelvic pain, menstrual disorders, pelvic mass, pelvic abscess.

In the course of subsequent attacks other organisms join the gonococcus in its destructive work. Owing to the factor of a mixed infection, the response to antibiotics is often not so immediate as in the initial attack. Further the infection is well walled off and for this reason not so amenable to treatment. In these difficult cases, the addition of cortisone has seemed to hasten the absorption of the inflammatory exudate in a manner which heretofore has not been seen. The dosage of cortisone should not exceed 1 gm. and the duration of treatment is almost four weeks. When cortisone is given under this plan the patient receives 50 mg. a day for two weeks and 25 mg. a day for the next twelve days.

Sterility is primarily a mechanical problem secondary to tubal occlusion. Surgical procedures for the reconstruction of the occluded fallopian tube are still in the developmental phase and are not recommended except in unusual circumstances. Ovarian failure may further complicate the sterility problem if the function of the ovary has been impaired by the inflammation.

Dyspareunia is always difficult to evaluate

publically. A mass the size of a four and one half months pregnancy however would be very unusual in these conditions.

Shape can be helpful but it also can lead one astray. Generally the fibroid uterus is asymmetrical and nodular. But fibroids can produce a symmetrical enlargement. Cysts are usually symmetrical but when multilocular they can be asymmetrical and even nodular. Shape therefore is of only relative, not specific, diagnostic significance.

Consistency is another characteristic of relative diagnostic significance. Fibroids usually are hard but, if edematous or undergoing cystic degeneration, they can feel semifluctuant. Cysts may be semifluctuant or hard and solid appearing. A pelvic abscess is not fluctuant until late. Usually the lower pole of an abscess is hard. Clotted blood in the cul-de-sac has a rather characteristic feeling. It is soft, cystic and may be semifluctuant. The margins are not well defined. Later as the clot organizes it becomes harder.

When a mass is fixed it is the result of inflammation, cancer or endometriosis (I-C-E). If a mass is freely movable it suggests pedunculation. Mobility and fixation are valuable in diagnosis when they can be clearly demonstrated. Pedunculated fibroids and many ovarian cysts are freely movable. Inflammatory masses are usually fixed. In retroversion, the uterus may be fixed or movable. If there is any doubt as to the nature of the condition, an attempt should be made to replace the uterus. The upward mobility of the normal anteverted uterus can be impaired by a previous inflammatory process. This is detected when the vaginal fingers attempt to elevate it in vaginoabdominal palpation.

Tenderness is of great diagnostic importance. However extreme care must be exercised in attributing the tenderness to a mass. Many masses which appear to be tender may be so on account of tenderness in the abdominal wall or as a result of a functional nervous condition. Real tenderness means inflammation resulting from an irritation due to bacterial causes, endometriosis or as the result of a degenerative process.

INFECTIONS

The surgical significance of pelvic infection is much less since the advent of chemotherapy and antibiotics. At present, these agents prevent or control most gonorrheal or puerperal infections, while in the past at

least one fourth of all gynecologic operations were for pelvic infections.

Acute Gonorrheal Infection. Often times the lower genital tract phase of gonorrheal infection is relatively asymptomatic. Elevation to the tubes occurs by way of the endometrium. The endometritis is usually transient and of no clinical significance. This is not so of salpingitis. At first there is purulent endosalpingitis, but it progresses rapidly to pansalpingitis and to pelvic peritonitis. The constitutional reaction is well marked with fever tending to be higher than in most intra-abdominal diseases. The temperature generally is above 101° F and frequently much higher. Leukocytosis is also well marked; often there are more than 15,000 white blood cells per cu. mm. of blood. Elevation of the sedimentation rate occurs early in the disease. The lower abdominal pain is severe and there is marked tenderness over the internal genitalia. Disturbance occurs early. Nausea and vomiting occur but are overshadowed by the violence of the other symptoms.

Pelvic examination reveals exquisite tenderness on the slightest manipulation of the cervix. Tenderness in the region of the adnexa is often unbearable. Early in the disease there may be a "fullness" in the adnexal regions, but it is difficult to feel circumscribed masses until somewhat later.

It is necessary to differentiate salpingitis from the other common conditions responsible for lower abdominal pain. Acute appendicitis by its very nature requires immediate operation. Salpingitis does not. Appendicitis is a gastrointestinal disease with nausea and vomiting as early symptoms. At first the pain is generalized. Later it localizes in the right lower quadrant. With salpingitis, the nausea and vomiting are not so prominent and the distribution of the tenderness is lower and generally bilateral. In salpingitis the patients look sick and have a well-marked febrile reaction. In appendicitis, the fever is not so high and neither is the leukocyte count. But the leukocytosis in appendicitis is greater in proportion to the fever than it is in salpingitis. In typical appendicitis, the pelvic examination usually reveals little or no pelvic tenderness. Movement of the uterus is free and painless. In salpingitis, digital examination reveals acute bilateral adnexal tenderness. Upward pressure on the cervix reveals extreme tenderness.

Differentiation is not so easy in the pa-

Perhaps the most characteristic feature of these cases is their slow resolution. There is no dramatic abatement of fever and infection as occurs in gonorrheal infections. The improvement is apparent from week to week rather than from day to day. Treatment must be aggressive and persistent until all signs of activity have disappeared.

Tuberculosis. At present genital tuberculosis has decreased in frequency to the extent that it has become one of the lesser problems in gynecology in this country.

The most common type of genital tuberculosis is the type encountered as an integral part of generalized tuberculous peritonitis. Tubercles are scattered over the surface of the genitalia, as well as the intestines, and may be associated with ascites and/or adhesions. This type of infection is not usually complicated by deep-seated genital foci and, consequently, is of little concern to the surgeon except in differential diagnosis.

Pelvic tuberculosis, exclusive of generalized peritonitis, is felt to be a local manifestation of a systemic disease. The primary focus in the lungs, bones, intestines or lymph nodes is thought to be the source of hematogenous spread to the pelvic organs. The fallopian tubes are involved in nearly every subject, while the endometrium is infected in about 70 per cent of the patients and the ovaries in from 30 to 40 per cent. The process usually starts in the tube where edema and tubercles develop and the tube may coalesce. The involvement of the endometrium and ovaries in a large percentage of subjects is due to extension from the tubal nidus. As the acute stage subsides healed tubercles develop with subsequent adhesions and, frequently masses. The lesions may break down and caseate producing marked systemic reactions, and it is this stage which is most easily recognized.

The diagnosis of genital tuberculosis is particularly difficult because of the variability and indefiniteness of the symptoms. The onset of symptoms is usually vague and generally the symptoms are those of a chronic pelvic infection. Frequently constitutional symptoms, such as malaise, anorexia, fever and weight loss, are present. The local symptoms may suggest gonorrhea, ovarian tumor or appendicitis; the resemblance varies with the stage of the disease. In the absence of a history of an acute pelvic infection, menstrual disturbances and sterility associated with pelvic pain and masses are strong argu-

ments in favor of the diagnosis of tuberculosis. The diagnosis of pelvic tuberculosis is frequently made by endometrial biopsy carried out during a routine sterility work up. If the diagnosis is suspected it may be confirmed by the use of Halbrecht's method of culturing menstrual blood.

It is obvious that frequently genital tuberculosis is not diagnosed correctly preoperatively. Often it is not diagnosed until the tubes are examined microscopically. Diagnostic observations which may help correct this deficiency are a history of chronic illness, palpation of masses in the pelvis, a low degree of fever, a low white blood cell count, no benefit from antibiotics other than streptomycin, improvement with streptomycin especially when combined with PAS, positive endometrial biopsy, positive smears, positive cultures of menstrual blood, peritoneoscopy, hysterosalpinography.

There remains considerable difference of opinion in regard to the accepted treatment of pelvic tuberculosis. The advent of the new antituberculous drugs streptomycin, para-aminosalicylic acid and isonicotinic acid hydrazine has, of course, modified the treatment of the disease and has made more cases amenable to surgery. It also has permitted the use of conservative surgery in selected patients and has led some observers to believe that medical treatment alone frequently is satisfactory. The variability of the type and extent of the lesions makes it difficult to prescribe a standard operative procedure. It is felt, however by a majority of gynecologists that the only successful definitive therapy in patients with serious cases is bilateral salpingo-oophorectomy and hysterectomy. When this procedure is not contraindicated, there is a better chance for cure. Optimal conditions for surgery are usually obtained by giving a course of streptomycin and PAS preoperatively and this medical treatment must be continued in the postoperative period for successful termination of the case. It is well to emphasize that drainage is contraindicated in these patients as this predisposes to fistula formation.

The final evaluation of various forms of surgery and medical treatment, or combinations of these forms of therapy must await the test of time.

OVARIAN TUMORS

The code word for pelvic masses is PREMIEO; the O designates ovarian masses. No other organ in the body develops such

Tender adherent adnexal masses or an adherent retroversion may produce this disturbing symptom. Surgical intervention may be necessary—the type and extent of the procedure vary with the needs of the patient. In general, the patients having the older or more severe cases will usually require hysterectomy.

Pelvic pain is a natural consequence of the lesions producing dyspareunia. Its evaluation proceeds along the lines discussed under pelvic pain. Surgery may be necessary in some subjects.

Menstrual disorders are frequent in these patients. Disturbed circulation or parenchymal ovarian damage produces the all too-familiar picture of ovarian failure. In many of the subjects clinical control may be attained by cyclic replacement of the deficient hormones. Eventually some of these patients will require surgery.

Pelvic masses, in addition to causing pain and dyspareunia, present a problem in differential diagnosis. If the differential diagnosis is uncertain exploration may be necessary.

Nowhere can the principles of the functional approach be better employed than in the treatment of the complications of chronic pelvic inflammation. Experience has demonstrated the danger of conserving a functionless uterus when surgery is necessary. Yet in some cases this danger must be accepted as a calculated risk. While most generally advisable the removal of the functionless uterus is not an invariable rule.

Puerperal and Postabortal Infections. By definition, puerperal and postabortal infections mean infection following delivery or abortion. Women who have gonorrhea can have a flare-up of this infection after labor or abortion. The pathologic state and the treatment are the same as for gonorrheal infection in the nonpuerperal state.

However infection, when it occurs, is usually with one or more organisms. Aerobic streptococci are present in 40 per cent of the subjects, *Staphylococcus pyogenes* var. *albus* in 25 per cent and hemolytic streptococci in 5 per cent.

Following labor or abortion, there is an open wound of the uterus at the placental site. In addition, there may be lacerations of the cervix, vaginal tract or perineum. The lochia provides a good culture medium and bacterial contamination almost invariably takes place. However the usual organisms are not very pathogenic and the body de-

fenses are adequate in all but the exceptional patients. In these, the defense mechanism is overcome. The bacteria then gain access to the uterine wall, rich in lymphatics and blood vessels. As a result, the infection spreads rapidly to the extraperitoneal tissue between the leaves of the broad ligament. This extraperitoneal cellulitis is the characteristic lesion of puerperal pelvic infection.

The diagnosis is usually not difficult. The patient always gives a history of recent delivery or abortion. The systemic reaction is usually marked with temperature elevation. There is generalized lower abdominal tenderness. Examination reveals a typical brawny induration of the pelvis.

Treatment consists of antibiotics, usually penicillin or one of the broad-spectrum antibiotics, oxytocics to insure contraction of the uterus and minimize bleeding, bed rest, fluid replacement and active search for complications.

Complications are frequent and consist of abscess formation, thrombophlebitis and peritonitis. The presence of one or more of these complications produces changes in the disease picture with the typical findings of these syndromes superimposed on the previously described symptoms and signs.

Abscess formation in the extraperitoneal spaces is not unusual but is often difficult to detect. Frequently these abscesses present in the inguinal region. The persistence of a septic course after adequate treatment and the appearance of fluctuant masses in the pelvis or abdomen calls for further treatment. When present, these abscesses must be drained. This is usually accomplished best through the cul-de-sac or through a lower abdominal extraperitoneal approach.

Septic thrombophlebitis is a common sequela of pelvic cellulitis and must be regarded with alarm. Most cases resolve following antibiotic therapy but when septic emboli occur they may be fatal. Collins and others advocate ligation of the vena cava in these subjects and, in an occasional patient, this procedure may be lifesaving.

Peritonitis may occur from perforation of the uterus, reflux through the tubes or by direct extension of the infection. When it occurs it produces paralytic ileus with abdominal distention and nausea and vomiting. The usual measures of intestinal intubation and decompression with fluid replacement and antibiotics will usually suffice. Here again, abscesses may form and must be drained when present.

both physical and mental is implicated. It is our belief that the function of one ovary is never as good as the function of two ovaries. The remaining ovary may not permanently compensate for the loss of the other ovary as for instance one kidney may do after surgical removal of the other kidney. As previously mentioned the basis of the functional approach to pelvic surgery is the conservation of useful function in terms of life health and the pelvic functions. This concept applies most aptly to ovarian surgery.

When an exploratory laparotomy is performed for a pelvic mass even if one is certain that an ovarian tumor exists, the exact nature of the tumor cannot be determined until the mass is visualized. Frozen sections as a rule are of little benefit. Because of this, microscopic diagnosis usually must be delayed until permanent sections are available. It is by a knowledge of the clinical and gross characteristics of ovarian tumors that the pelvic surgeon must make a tentative diagnosis. This diagnosis is applied to the patient in terms of function and the best solution possible for the patient is then offered. An example of two different individuals with the same type of tumor may best demonstrate this approach.

A twenty-three-year-old female gravida 0 had been married six months. This woman had been having menstrual irregularities since her marriage. The flow was excessive in amount and occurred at eighteen- to thirty-one-day intervals. She was examined because of this irregularity and a 9 cm. mass was found. At operation the mass was found to be a papillary serous cystadenoma of the left ovary. The right ovary, tubes and uterus appeared normal. Although a clinical characteristic of these tumors is bilateral occurrence with certain malignant potentialities it was felt that the functional needs of the patient outweighed the risks, and only a left oophorectomy was performed.

A forty-three year-old female gravida III saw her physician for the same complaint of irregular menstruation of the same type as that of the previous patient. A 9 cm. mass was felt in the left adnexa and at laparotomy a papillary serous cystadenoma with only internal papillary growths was found. Taking into consideration the age of the patient, the tendency to a bilateral occurrence, the malignant potential inherent in the tumor and the functional needs of the patient,

total hysterectomy and bilateral salpingo-oophorectomy were performed.

In the first patient a young woman, the pelvic duties of childbearing, ovarian function, menstruation and sexual function were at stake. With close observation and cyclic therapy it was not felt that the risk of recurrence, malignant potential and the present menstrual irregularities outweighed the positive value of preserving the childbearing, menstrual, sex and ovarian functions.

In the second patient the childbearing function had been fulfilled, menstruation was becoming a nuisance and the irregularities were apparently due to diminishing ovarian function. In this woman it was felt that the risks involved were not counterbalanced by the value of retaining the reproductive and menstrual function.

Our classification of ovarian tumors is very simple and is one which fits into the functional approach to pelvic surgery. It is a clinical classification based on the malignant characteristics of the various ovarian tumors. This malignant potential often is the determining factor in the functional approach.

The tumors are divided into three groups

- I. Ovarian tumors without malignant potential.
 - A. Nonproliferative tumors—follicular cyst, corpus luteum cyst, germinal inclusion cyst.
 - B. Endometrioid cyst.
 - C. Brenner tumor.
- II. Ovarian tumors with malignant potential.
 - A. Cystadenoma, serous, pseudomucinous.
 - B. Dysgerminoma.
 - C. Arrhenoblastoma.
 - D. Granulosa cell tumor.
 - E. Theca cell tumor.
 - F. Fibroma.
 - G. Dermoid cyst.
- III. Malignant ovarian tumors.
 - A. Carcinoma—primary solid carcinoma and cystic carcinoma.
 - B. Teratoma.
 - C. Sarcoma.
 - D. Metastatic carcinoma.

In order that these tumor masses can be classified at the operating table a working knowledge of the pathologic characteristics of the ovarian tumors is essential.

The symptoms of ovarian neoplasms are variable and depend on several factors, such as the type of tumor, its size and location. Frequently slow growing, benign tumors can become rather large neoplasms without producing any symptoms and occasionally are found on routine physical examination. Symptoms may occur earlier in intralig-

a variety of neoplastic growths as does the ovary. To understand the nature and significance of these new growths, one must look to the embryonal development of the ovary.

Beginning with an accumulation of cells on the anterior aspect of the wolffian body the ovary passes through several stages of development before assuming the characteristics of the female sex gland. It is important to understand that it is impossible at one stage of development to determine by histologic examination what the sex of the cell mass is going to be. This phase is spoken of by embryologists as the undifferentiated period of development. Of equal importance are the changes which occur during the stages of differentiation. At one particular stage, the primitive ovary shows many of the characteristics of the male testicle. These changes are only transient and are followed by changes showing the characteristics of the ovary. Several investigators have spoken of the development of the female gonad as bisexual in nature and consider the female ovary as an organ more advanced in development than the male gonad. These multiple developmental changes are the basis for the wide range of tumors that may occur in the ovary. In addition it is of importance that the mature ovary contains a variety of different types of cells which add to the complexity of tumor formation.

The classification of ovarian tumors has been a subject of much controversy, since Waldeyer in 1870 presented a classification based upon the histogenesis of the tumors. Many additional classifications of ovarian tumors have been presented in the literature by many authors. It is now realized that very little is known concerning the histogenesis of many of the ovarian neoplasms. The fallacy of the classifications is that the basis of knowledge is unstable.

Undoubtedly a universally acceptable classification is most important for clinical comparison and statistical reporting. One of the more applicable classifications of ovarian tumors is that presented by Kottmeier of the Radiumhemmet in Stockholm, Sweden. This classification is presented as an excellent histologic classification of ovarian tumors with clinical significance.

- A. Non-neoplastic tumors, follicle cyst, corpus luteum and regression cyst
- B. Endometriosis and endometriotic cyst
- C. Primary neoplastic tumors
 1. Neoplasms with endocrine significance:

1. Feminizing ovarian tumor, granulosa cell tumor, theca cell tumor
2. Virilizing ovarian tumors, androsthenoma, adrenal rest tumor, Leydig cell tumor
3. Ovarian tumor with inverse endocrine symptoms, folliculoma, lipiodome
4. Gynandroblastoma

II Neoplasms with no endocrine significance

- A. Germ cell tumor, dysgermoma, embryoma, ovarian tumors (sometimes producing gonadotropic hormone), dysgerminoma, mesonephroma (Schäfer), chorionepithelioma
- B. Ovarian tumors.
 1. Benign tumors, dermoid cyst, struma ovarii
 2. Malignant tumors, teratoblastoma, malignant degeneration of a dermoid cyst
- C. Stromatogenous tumors.
 1. Benign tumors, fibroma, myoma, sarcoma
 2. Malignant tumors, sarcoma
- D. Epithelial tumors
 1. Benign tumors, papillary tumors with no signs of marked proliferation, serous and pseudomucinous cystadenoma with no histologic signs of marked irregular proliferation of the epithelium, Brenner tumor
 2. Tumors with questionable malignancy also called seminomatous epithelial tumors, papillary tumors and cystadenoma with epithelial changes identical to those seen in invasive carcinoma, no proved invasion in the stroma, pseudoinvasive peritoneal
 3. Malignant tumors, carcinoma proved invasion in the stroma. The carcinoma can be divided into:
 - (a) Primary ovarian carcinoma
 - (b) Ovarian carcinoma developing from serous or pseudomucinous cystadenoma
 - (c) Cancer uteri et ovarii

D Metastatic neoplastic tumors

Although a good universal laboratory classification of ovarian neoplasm is desirable, a good clinical classification usable in relation to the functional approach is essential.

Because of the close interrelationship of all the female genital organs no one organ can be removed without having a definite effect upon the other organs and their functions. The ovary is the main site of estrogen and progesterone production, as well as the only site for the production of ova. Thus, in terms of pelvic function, reproduction and ovarian, menstrual and sexual functions are involved when ovarian surgery is undertaken. In addition to this the general health,

the cyst the more frequently the lining cells are flattened.

Many of these cysts are found accidentally on routine pelvic examination. Abnormal uterine bleeding resulting from ovarian dysfunction may be present in association with these cysts. Pain or lower abdominal discomfort may at times become a prominent feature.

A corpus luteum cyst is another non neoplastic cyst of the ovary which is a physiologic exaggeration of a normal structure. Instead of the corpus luteum undergoing its normal course of regression the cavity becomes distended with an amber fluid. Frequently the cystic enlargement is a result of excessive hemorrhage during the normal course of development. Occasionally the fluid is chocolate in appearance which points up the fact that all cysts with a thick chocolate material are not due to endometriosis.

The corpus luteum cyst is singular and rarely gets larger than 4 to 5 cm. in size. It is characteristically lined by a thinned-out layer of luteal cells. This type of cyst may produce hormones similar to the corpus luteum and therefore may be associated with amenorrhea or oligomenorrhea or it may disrupt the hormonal balance and produce irregular uterine bleeding.

ENDOMETRIAL CYST (ENDOMETRIOSIS)

Endometriosis is the growth of endometrial glands and stroma in an abnormal location. This condition divides itself into two categories: internal endometriosis, or adenomyosis of the uterus, and external endometriosis in which the ectopic endometrium is outside the limits of the uterus. The frequency and presence of endometriosis have seemingly increased in the past ten to fifteen years. The majority believe that increased incidence represents an increased knowledge of the disease as well as an increased ability and effort to recognize endometrial implants at the time of operation. For these reasons it is assumed that the increase in frequency of endometriosis is not real. Some surgeons have reported that 32 per cent of all abdominal gynecologic operations revealed endometriosis. The most common location of the implants of endometriosis are the ovaries (this may be bilateral), uterosacral ligaments, the cul-de-sac, broad ligament and the pelvic peritoneum.

It is generally accepted that internal endometriosis is due to a downgrowth of the

endometrium in the uterine musculature. The etiologic basis of external endometriosis was brought to medical attention in 1921 by Sampson's theory of tubal reflux. Sampson suggested that endometrium cast off in menstruation may regurgitate through the tube adhere and grow on the ovaries in the pelvis or in adjacent tissues. Meyer believes that endometriosis is due to a growth stimulus acting on cells derived from the coelomic epithelium. The endometrium and endosalpinx both represent a modified and differentiated coelomic epithelium. On the other hand the genital epithelium of the ovary represents the original coelomic epithelium. It is Meyer's theory that in ovarian endometriosis the genital epithelium of the ovary can differentiate cells which are capable of forming endometrial like tissue. The same possibility exists wherever coelomic epithelium or its vestiges occur including the pelvic peritoneal surfaces, the umbilicus and hernial sacs. Lastly there is Hallman's theory which states that the aberrant endometrium has its source in endometrial particles transported by the lymphatics to various locations of the pelvis. This theory can account for many of the lesions found in the lymph nodes of the pelvis, but it is doubtful if it can explain all of the various sites of endometriosis in the body. The presence of endometrial like tissue in lymph nodes has received much attention recently in connection with the study of the spread of adenocarcinoma of the corpus uteri. The tendency in the last few years has been toward acceptance of a combination of the Sampson and Meyer theory. The consensus however still favors Sampson's theory for the explanation of pelvic endometriosis. On the other hand many use the coelomic metaplasia theory to account for lesions not explainable by the regurgitation theory.

Endometriosis is a disease originating only during menstrual life. Usually it appears in women between the ages of thirty and fifty years. Sterility is one of the common symptoms associated with endometriosis. 30 to 40 per cent of the patients with endometriosis never become pregnant. Associated pathologic lesions are very frequent, these include myoma, endometrial hyperplasia and ovarian failure. Internal endometriosis or adenomyosis is found most frequently in women in their early forties and is not as commonly associated with sterility as is external endometriosis. Pain is the most common symptom of endometriosis but there are some

mentary tumors some malignant tumors or as a result of hemorrhage or torsion

Pain or discomfort is one of the earlier symptoms of ovarian neoplasm. Usually the pain is dull or "nagging" in nature, involving the lower abdomen and perhaps one side in particular. The pain may radiate down the thighs. Backache over the sacrum may be present, particularly if the cul-de-sac is involved. Unilateral backache just lateral to the sacrum, if present, is a good indication of the side of origin.

The effect of ovarian neoplasm on adjacent organs is again dependent on the characteristics of the neoplasms. Frequency, urgency and even urinary retention may be the result of pressure by the tumor growth on the bladder.

Often noted are the presence of vague gastrointestinal symptoms in relation to the ovarian tumors. Constipation, abdominal discomfort due to pressure and nausea and vomiting may all be present.

The more acute symptoms of ovarian tumors are due to the complications of the tumors. Torsion, rupture, necrosis, hemorrhage, infection and gangrene are the more frequent complications. The symptoms due to these complications may be short or long in duration. Fever is usually present. Pain is a dominant feature. In gangrene the patient is extremely toxic and the picture may resemble that of a severe intraperitoneal accident. In spontaneous rupture or hemorrhage, a shocklike picture may be seen. The treatment of patients in whom complications occur is surgical.

There are several features which may aid in the clinical and macroscopic diagnosis of ovarian neoplasms.

Clinical signs and symptoms of the patient must be considered. These findings may include uterine bleeding, pain, ascites, abdominal enlargement, evidence of hormonal changes and changes in sexual characteristics.

The gross appearance of the tumor such as color, size, degree of firmness and the presence or absence of a capsule is of much importance.

The location of the tumor or tumors is of significance. Whether the tumor is unilateral or bilateral is of importance when one considers either the clinical diagnosis or proper operative therapy. Other conditions in relation to the location of the tumor such as the involvement of adjacent structures, presence or absence of peritoneal implants, or in-

volvement of the mesenteric nodes, are of extreme importance.

The next aid in diagnosis is the gross section of the tumor. This involves such characteristics as whether the tumor is cystic or solid. If cystic, the characteristics of the fluid may be helpful. If solid, the color and consistency are of value in the diagnosis. Occasionally, the presence of necrosis, hemorrhage or cystic degeneration may add to the above findings.

The age of the patient is of great significance as certain tumors have a definite age preference which may be useful in diagnosis.

NONPROLIFERATIVE TUMORS

This group of tumors includes the persistent follicle cyst, corpus luteum cyst and the theca lutein cyst. They are classified as nonproliferative because there is no actual growth or proliferation of the cellular elements. The cysts may be said to represent nothing more than an abnormal physiologic accumulation of fluid within the ovary.

It is a normal phenomenon that only a few of the many primordial follicles develop to the stage of producing a mature ovum. The remaining follicles degenerate, their fluid is absorbed and is replaced by new and, in time, by the normal ovarian stroma. Atresia of a larger follicle is usually attended by the formation of a small cyst. This persistent follicle cyst is rarely solitary and seldom of more than 1 to 2 cm. in diameter. Generally the persistent follicles are asymptomatic unless the accumulation of fluid is excessive or the production of these small cysts is markedly increased. The enlargement of the cyst is not due to a cellular growth, but to an increased accumulation of fluid. These cysts are usually classified as multiple follicle cysts and the ovaries which contain them are known as polycystic ovaries. Not infrequently one of these multiple small follicle cysts accumulates more fluid than the other follicles. In size, it may reach a diameter of 8 cm. The main cavity of the cyst is unilocular, but there are other small follicle cysts in the walls or adjacent to the main cyst. These cysts are thin walled and usually translucent in appearance. The surface is smooth, both internally and externally. The fluid within the cyst is usually light amber in color.

The microscopic appearance depends somewhat on the size of the cyst. The smaller the cyst, the greater is the resemblance to the ovarian follicle. The larger

been associated clinically with postmenopausal bleeding, but there is no evidence that hormonal secretion could account for the bleeding. It appears, therefore, that there are no pathognomonic symptoms or signs of the Brenner tumor.

Grossly the appearance of the Brenner tumor is similar to that of fibromas, the mass is firm, smooth and varies in size from one which can be seen only through the microscope to a relatively large growth. Most of the tumors are small, averaging about 3 to 5 cm. in diameter. It is of interest that approximately 20 to 30 per cent are found in association with a pseudomucinous cystadenoma.

The cut surface of the Brenner tumor shows a characteristic uniform yellow tint. Necrosis, hemorrhage and cystic degeneration are rarely seen. The tumor does not have a definite capsule but has a pseudo capsule of compressed ovarian stroma.

The microscopic picture is characteristic: there are epithelial cell nests or columns in fibrous tissue matrix. The cell nests often show an area of central cystic degeneration which looks a little like an ovum in a follicle.

The Brenner tumor is generally regarded as a benign neoplasm, but an occasional malignant one has been reported.

CYSTADENOMAS

The cystadenomas are the largest group of proliferative tumors of the ovary. Approximately 75 to 80 per cent of all ovarian neoplasms are cystadenomas. The ratio of incidence of the cystic to the solid ovarian tumor is approximately 3:1.

There are two types of cystadenomas—the pseudomucinous and the serous. These tumors get their names from the character of the fluid in the cysts.

The pseudomucinous cystadenoma is so named because of the mucin-like contents of the cyst. Although the fluid appears and feels like mucin, it is not a proteid and does not give the characteristic reaction with acetic acid. These tumors may vary in size from the microscopic lesion to the most enormous of all tumors. Medical literature reports tumors of this type weighing as much as 300 to 350 pounds. Even today, fifty and sixty pound tumors are seen in patients from rural areas.

The pseudomucinous cystadenomas are more common in the forty to fifty year age group. The majority of the cysts are uni-

lateral in nature, less than 10 per cent are bilateral.

Grossly, the capsule is thickened and whitish blue in color. Areas of firmness can be felt scattered about the tumor surface. Brenner tumors and dermoid cysts may be found occasionally within the wall of the cyst.

On cutting of the tumor, the mucoid content is characteristic. The material is a thick jelly like substance with clear or greenish color. If internal hemorrhage has occurred a brownish color is seen. The cysts are always multilocular. Generally several locules have become one large cavity because of interlocular pressure. Papillary growths within the cyst cavity occur in only 5 to 10 per cent of these lesions. External papillary growths are relatively rare. These tumors rarely become malignant.

Microscopically the lining of the cyst is seen to be composed of a single layer of tall, columnar epithelium—the picket fence epithelium—resembling that of the endocervix.

The treatment, of course, is surgical. The extent and nature of the operation depend on the associated pathologic conditions and the functional needs of the individual. Since the malignant tendency of the tumor is low, conservatism is possible if it is necessary to preserve the reproductive function. In older patients, hysterectomy and salpingo-oophorectomy may be the operation of choice.

The tumor that Ephraim McDowell removed from Mrs. Jane Todd Crawford was of the pseudomucinous type. This operation marked the beginning of surgery of the abdomen. Fortunately for Mrs. Crawford, McDowell and surgical history, pseudomucinous tumors are usually not adherent and are well pedunculated.

The serous cystadenoma occurs as frequently as does the pseudomucinous cystadenoma. Serous cysts differ from pseudomucinous cysts in these ways: they rarely grow to a size larger than a man's head; more often than not they are bilateral; if a pedicle is present it is short; adhesions to surrounding structures are common; papillary growths, either internal or external, are frequent and the tendency to malignancy is great.

On incising the cyst, the serous, amber fluid is noted. When hemorrhage has occurred within the cyst, a more chocolate-colored fluid is seen. Internal papillary growths may sometimes occur in scattered areas.

subjects who have not had the slightest discomfort. The lesions vary in location; therefore the pain arising from them varies in location and nature. The pain is either unilateral or bilateral, usually it is bilateral but more marked on one side than on the other. Characteristically it is exaggerated premenstrually or menstrually and a residual soreness follows the period. In some instances, acute attacks of pain are entirely unrelated to menstruation. The pelvic distress may vary from a moderate bearing-down discomfort to a severe sharp knifelike attack. The invasion of the uterine musculature by endometrial tissue may be manifested by a visceral type of pain resembling dysmenorrhea or some change in the character of dysmenorrhea. Dysmenorrhea is a common symptom of both internal and external endometriosis. Frequently the dysmenorrhea is an acquired or secondary type. However there is nothing specifically characteristic about the dysmenorrhea associated with endometriosis. The duration of pain particularly in the more extensive cases is usually longer than the one-day cramps of primary dysmenorrhea. Menstrual irregularities are found in approximately one-third of the subjects. There may be either a decrease in the interval or an increase in the amount. The usual explanation for the menstrual disturbance is that it is secondary to ovarian failure resulting from the endometriosis. Other symptoms such as dyspareunia, backache and pain in the rectum or bladder may present. While all of the symptoms should suggest endometriosis, any or most may be present in association with other pelvic conditions. The physical findings may also vary in accordance to the extent and the nature of the disease. One or both adnexa may be fixed and adherent to the uterus. Frequently the uterus is in an adherent, retroflexed position and may contain one or more myomas. Myomas occur in more than half and nonproliferating cysts in more than a third of the women who have endometriosis.

The most characteristic physical finding is a "shotty" induration in the cul-de-sac and along the uterosacral ligaments. Usually there is, in addition, a lack of mobility of the uterus, enlarged and fixed adnexa and retroversion. Induration in the cul-de-sac and uterosacral ligaments is best detected by palpating the posterior vaginal fornix with the second finger while elevating the uterus with the first finger. Rectal palpation adds valuable information, especially concerning the uterosacral ligaments. The induration

feels like several buckshots imbedded in the area. At times, the lesion is so extensive that it invades the mucosa of the posterior fornix, producing scarring and small blue-black cysts.

Fortunately for the patient, endometriosis progresses slowly and rarely becomes malignant. Hence in women in whom the diagnosis is clear surgical treatment should be postponed until the severity of the symptoms forces operation. For reasons which are not clear, pregnancy seems to be the best treatment.

Large doses of testosterone produce pituitary depression with a decrease in estrogen production and improvement of the symptoms. Even small doses of androgens given for seven days before the onset of the menstrual period may produce a worthwhile diminution in symptoms. Large doses of stilbestrol are also advocated. The drug is given over a period of several months in a gradually increasing fashion. More recently progesterone and progesterone-like substances have been used. Amenorrhea is produced over a period of three to four months with clinical improvement noted in many of the cases. Its mechanism of action is probably the same as that of pregnancy. Irradiation will suppress estrogenic secretion and the lesions will regress. This form of treatment has the same disadvantages as does castration radiation for ovarian failure. We rarely use it.

In patients with disabling symptoms resistant to treatment, operation is necessary. No other pelvic condition tests our faith in the functional approach as does endometriosis. The problems are difficult. In the patients in whom childbearing is not a factor, hysterectomy may be the operation of choice. When childbearing is a factor conservative operations are in order. These combined with presacral neurectomy will often produce a satisfactory result. It is interesting that most of the pain in endometriosis is relieved by removing the uterus. This can allow the conservative treatment of involved ovaries if the preservation of ovarian function is considered necessary.

BRENNER TUMOR

Brenner tumors usually occur in the postmenopausal period. They produce no symptoms except for those arising from pressure. The majority of the neoplasms are discovered accidentally at routine physical examination or at operation for other causes. A few of the reported Brenner tumors have

gland can reproduce all of the signs and symptoms characteristic of a masculinizing tumor of the ovary. The Stein-Leventhal syndrome must be considered in patients in whom hirsutism and amenorrhea are the dominant features. Special laboratory procedures which include a glucose tolerance test, a ray study of the bones and determination of 17 ketosteroids and 11-oysteroids are helpful in arriving at the diagnosis. The response of the steroid to intravenous administration of ACTH is helpful in differentiating adrenal conditions. These procedures must be combined with a good physical examination. Examination with the patient under anesthesia and the use of the cul de scope aid in clarifying the diagnosis.

Grossly the tumor may vary from a very small size to a growth of 10 to 20 cm. in diameter. The small tumors are usually solid. As the tumor grows, cystic degeneration is common. The color varies from a grayish to a bluish or purplish hue. On cut section the tumor shows a definite lobulation not commonly observed in other ovarian tumors.

The histologic diagnosis frequently is made with ease because of the tumor's close resemblance to the male gonad. On the other hand, the diagnosis may be difficult because of the marked anaplastic nature of the cells. A cordlike arrangement of cells is helpful. The differentiation between arrhenoblastoma and sarcoma of the ovary may be extremely difficult.

Arrhenoblastomas should be considered as malignant. The true evaluation of the degree of malignancy has been confused because of the small number of reported cases and the early reporting of cases. Some of the tumors are highly malignant. In some patients recurrence and death have occurred as late as ten to twelve years after surgical removal of the tumor.

The prognosis and therapy depend on the findings at the time of operation. If the tumor is well encapsulated and can be removed without rupture the prognosis is good. If rupture of the capsule has occurred and metastatic spread is present, the prognosis is grave. The details of the treatment are similar in all respects to those of carcinoma of the ovary.

GRANULOSA CELL TUMOR

This is a functioning ovarian neoplasm, the origin of which is controversial. Robert Meyer suggested that the tumor arises from persistent granulosa cell rests remaining

from the early development of the primordial follicles. His concept was widely accepted until recently. There is now suggestive evidence that the tumor develops from cells of the early undifferentiated mesenchyme.

The tumor is most frequently reported in the prepuberty female child or the postmenopausal woman. Actually the majority of the neoplasms occur during the period of reproductivity. Only 5 to 10 per cent occur in children before puberty. 30 to 35 per cent of the tumors are seen in postmenopausal women.

The symptoms are due to increased secretion of estrogen. In the young child, the signs of precocious puberty are present. Menstruation may occur. The development of secondary sexual characteristics is seen. These are enlargement of the breast, the growth of pubic and axillary hair and pubertal changes of the vulva. In the postmenopausal woman the signs are again due to the stimulating effect of estrogen. Uterine bleeding is the one important sign. The return of secondary sexual characteristics does not occur. When the tumor develops in the mature menstruating female, menstrual irregularity is noticeable. Periods of amenorrhea lasting two months to two years are seen. This is usually followed by a menometrorrhagic type of uterine bleeding. Enlargement and softening of the uterus are felt. There is a sense of fullness in the breasts.

Grossly the tumor may vary in size from a few millimeters to an extremely large growth. A tumor of moderate size is the usual finding. The neoplasm is firm and encapsulated. The outer surface is smooth and lobulated. On section a yellow color is characteristic. Cystic degeneration is a common finding.

Microscopically the tumor is divided in three types—follicular, cylindrical and sarcomatous. The tumor type is dependent on the distribution of the granulosa cells. The follicular type is the most common variety. The cells are arranged in small clusters or rosettes, about clear spaces which often contain vacuoles similar to the Call-Exner bodies. Connective tissue gives the tumor a trabeculated appearance. Cellular areas are divided into lobules of varying sizes.

In the cylindrical type, the cells are arranged in columns. The connective tissue is more predominant and often vascular. The rosette-like arrangement of cells is lost.

others will fill the entire cyst cavity. Malignant changes are most often seen in large friable, necrotic cauliflower like papillary growths.

Microscopically the epithelium is variable in appearance. The lining cells may resemble tubal epithelium both ciliated and non ciliated, or simply consist of one or two layers of small, round, vesicular cells.

The treatment is always surgical. These are dangerous tumors and should be treated by hysterectomy and bilateral salpingo-oophorectomy in women of the older age group. In the younger woman, some consideration must be accorded to the functional needs. This must be balanced against the risk of retaining the opposite ovary. This is not too great if the tumor is not of the papillary variety. It is greater when there are internal papillae and very great when there are external papillae. The surgeon must examine the ovary to be retained with the greatest care.

DYSGERMINOMA

In most large gynecologic clinics one to two dysgerminomas will be seen each year. Approximately 450 of these tumors have been reported in the literature. The age distribution of the tumor varies; the ages of the patients may be from two to seventy-six years. More than 76 per cent of the lesions occur in patients between eleven and thirty years of age. The average age for the appearance of dysgerminoma is around twenty to twenty-one years.

There are no endocrine symptoms. This has led to the belief that the tumor originates from embryonal cells presenting from the undifferentiated stage of gonadal development. This tumor resembles the seminoma in the male. Underdeveloped genitalia and breasts are frequently associated. The lesion has been reported to occur frequently in the pseudohermaphrodite. This again, is suggestive as to the etiologic basis of the tumor.

Dysgerminomas grow rapidly. They are bilateral in approximately 15 per cent of the subjects. In size the tumor may vary from microscopic to one that is large enough to fill the entire abdomen. Under the microscope the tumor varies in appearance depending on the degree of cellularity. The cells are large and clear with centrally placed deep-staining nuclei. This tumor is spoken of as a large cell carcinoma. There is always a varying degree of fibrinous tissue with lymphocytic infiltration. The fibrinous

areas surround clusters of large, clear cells which give the growth a characteristic appearance. In the more cellular tumors, the characteristic fibrinous appearance is seen only in the edges of the lesion.

The prognosis of the dysgerminoma depends entirely on the findings at operation. All dysgerminomas are potentially malignant. In freely movable encapsulated tumor with no perforation of the capsule, the prognosis is favorable in approximately 90 per cent of the subjects. In bilateral tumor or tumors in which there is evidence of perforation or rupture the prognosis is poor.

Treatment depends upon the operative findings and the functional needs of the patient. Since most of these tumors are seen in younger individuals the functional needs are most important in selecting the type of therapy. If the tumor is encapsulated and movable unilateral oophorectomy is the treatment of choice in the young individual in whom the childbearing function is of significance. Postoperative radiation in small doses is advisable. This is directed at a 10-degree angle laterally to minimize the amount of radiation to the opposite ovary. The upper abdominal quadrants also receive small doses of x ray therapy as they are frequent areas of metastasis. On the other hand if the tumor shows evidence of capsular perforation or metastasis spread, the therapy should be the same as for ovarian carcinoma. Great benefit can be obtained by carefully directed deep x ray therapy. These tumors are exceedingly sensitive to x-ray.

ARRHENOBLASTOMA

The arrhenoblastoma is a relatively rare ovarian tumor. This neoplastic growth has received much attention because of the dramatic changes which it produces in the secondary sexual characteristics—defeminization and masculinization—of the patient.

The tumor is most frequently seen in the young, very rarely in postmenopausal individuals. The majority occur in women in the twenty to thirty year age group.

Amenorrhea and a loss of fat are the first noticeable signs. The breasts become flattened, the stature slowly acquires the male characteristics, the hair distribution becomes that of the male; a beard becomes prominent and shaving is frequently required, enlargement of the clitoris takes place and voice changes occur.

The differential diagnosis is difficult. These patients usually require very careful investigation. Disturbance of the adrenal

Calcification may be noted on cutting, imparting a gritty feeling to the knife.

Two per cent of these lesions become malignant. Soft areas are uncommon in fibromas, when they are found, sarcomatous changes must be suspected.

The therapy is always surgical. Fibromas and metastatic ovarian carcinomas sometimes appear quite similar grossly, therefore in all patients having fibromas, especially if these are bilateral, the abdomen should be explored with care.

DERMOID CYST

Dermoid cysts account for 10 to 15 per cent of all ovarian neoplasms. Much controversy exists concerning the origin of these cysts. There are two theories: (1) they arise from the imperfect development of blastomeres, (2) their source is an abnormal development of an unfertilized ovum. Of late the misplaced blastomere theory has gained much support.

At the operating table dermoid cysts are readily recognizable. They rarely grow larger than 12 to 15 cm in diameter; the capsule is thick and whitish blue in color. On palpation, firm, hardened areas can be felt in the capsule. Frequently the tumors are pedunculated and nonadherent to the surrounding structures. It is important to remember that in approximately one-quarter of the patients the tumor will be bilateral. The bilateral cysts are not necessarily of the same size. It is a good rule never to fail to examine the opposite ovary for a second dermoid cyst. In doing this, all cystic structures should be punctured. When found the small dermoid cysts are easily shelled out with preservation of normal ovarian tissue.

On incision of the capsule a yellow, thick, sebaceous material is found coiled through out the cavity are large masses of hair, teeth and bony structures may be present.

Microscopically, ectodermal elements are the predominant tissues. Stratified squamous epithelium, hair follicles and sebaceous glands may all be present. Other structures such as cartilage and stratified ciliated columnar epithelium can be found by a careful search.

The tendency of dermoids to become malignant is low. Less than 2 per cent of the tumors reveal malignant characteristics. While the dermoid is a teratoma, it differs from the malignant solid teratoma in that it is cystic, usually benign and shows microscopic predominance of ectodermal elements.

The treatment of dermoid cysts of the ovary depends on the age and the functional needs of the patient. In the younger patients every effort should be made to preserve normal ovarian tissue. Cystic masses 5 to 7 cm in diameter can frequently be shelled out leaving functioning ovarian tissue. This is most important in the young. A tumor in the opposite ovary must not be overlooked. In the older individuals who have satisfied their reproductive needs, hysterectomy with unilateral or bilateral salpingo-oophorectomy is probably the treatment of choice.

CARCINOMA OF THE OVARY

Carcinoma of the ovary comprises one of the larger groups of ovarian tumors and the ovary ranks second only to the uterus as a site of cancer in the female genital tract.

Within this group of tumors there are a number of special malignant types in which there is no agreement regarding classification. Clinically the group is subdivided into the primary solid adenocarcinomas and the cystic adenocarcinomas or the cystadenocarcinomas. The latter tumors are the more common in an approximate ratio of 2:1.

Clinically ovarian carcinoma is difficult to diagnose because in the early stages of the disease it produces no symptoms. Pain is one of the first symptoms to appear but when this occurs, metastatic spread is likely. Abdominal enlargement, due either to ascites or the tumor, is noted in more than 50 per cent of the subjects. In Meigs' series 30 per cent demonstrated vaginal spotting. Postmenopausal spotting was a feature in 10 per cent of the patients. It is of interest that sterility has been noted in as many as 40 per cent of the women who develop carcinoma of the ovary.

Age is an important consideration in this group of tumors. Shands and Clarke found that the percentage of ovarian tumors which were malignant among patients less than forty years of age was only 2.7. In patients between forty and fifty years of age, it was 14.3. In those over fifty years of age, it was 69. The condition can be summed up in the following manner: a primary ovarian carcinoma is a common tumor growth; it is practically never observed in children and is rare among women younger than forty years of age; once the menopause has been reached, the likelihood of an ovarian neoplasm being malignant is greater than 50 per cent.

Primary Solid Adenocarcinoma. Occasionally the determination of whether a solid tumor is benign or malignant must be made

The sarcomatous variety is very cellular. Large diffuse areas of closely packed cells are characteristic. The diagnosis is frequently difficult.

The therapy is surgical removal. Unilateral oophorectomy is the procedure of choice in the younger individual with a clinically benign tumor. In the postmenopausal woman, bilateral salpingo-oophorectomy and hysterectomy is a better approach. In the older patient, the presence of an associated adenocarcinoma of the uterus should not be overlooked. Histologic examination of the endometrium is essential in the preoperative work up. Tumors which are clinically malignant should be treated as carcinoma of the ovary.

The prognosis of the clinically benign tumor is good. Unfortunately, 20 to 25 per cent of the patients will be found to have a clinically malignant lesion at operation. Recently the development of late recurrence has been noted fifteen to twenty years after the removal of the primary tumor. The material from the Radiumhemmet shows a poorer prognosis in the sarcomatous variety of the granulosa cell tumor.

THECAL CELL TUMOR

The reported incidence of thecal cell tumor has increased in the past few years. More than 150 of such tumors have now been reported in the literature. This growth occurs most frequently in women over fifty years of age. However, 30 to 40 per cent are in women between thirty-five and fifty years of age. It is very rare that a thecal cell tumor is reported in an individual under thirty-five years of age.

The symptoms of thecal cell tumor are due to the fact that the tumor secretes estrogen. As a result postmenopausal bleeding and menstrual irregularity are common symptoms. Disturbances of menstruation, however, are not invariable as thecal cell tumors have been found in postmenopausal women who showed no symptoms.

Grossly the tumor may vary from a microscopic size to 15 to 20 cm. in diameter. The small tumors have no capsule but are sharply demarcated by color and consistency from the surrounding ovarian stroma. The large tumors have a pseudocapsule formed by compression from the adjacent stroma. In shape, the tumors are usually ovoid or spherical, in consistency firm and somewhat elastic, depending on the degree of cellularity. Cystic degeneration is occasionally present and focal areas of calcification occur

In cut sections, the tumor is seen to have an irregular yellowish appearance with some grayish white mottled areas. In some areas it may resemble a fibroma.

Microscopically the appearance varies from that of a highly cellular structure containing small clumps of spindle cells with well vacuolated cytoplasm and clusters of ovarian lutein-like cells to a more fibrous form in which the predominant cell is an elongated spindle cell. The use of special stains to demonstrate a reticulum and the presence of fatty materials may be helpful. Collagenous strands of tissue are prominent in the fibrous type tumor. This can be shown with Masson's trichrome stain. The tendency to malignant degeneration is slight, only three or four malignant theca cell tumors have been reported. It is the general opinion that the tumors are almost always benign, but Greenhill calls attention to the possibility of late recurrences which are not reported in the literature. He states that malignant change occurs in about 25 per cent of these growths.

The therapy consists of removal of the tumor. As a general rule, these tumors occur in the older individual and bilateral salpingo-oophorectomy and hysterectomy is probably the therapy of choice.

FIBROMA

Fibromas of the ovary are usually benign and may develop any time after the onset of puberty. The majority are found clinically in women in the later years of reproductive life. Only 1 to 2 per cent of ovarian neoplasms are fibromas.

The growth rarely produces symptoms until it becomes large enough to fill the true pelvis. Then the complaints are usually vague and result from pressure on the other pelvic organs. Other than presence of a mass and a sense of heaviness, urinary and gastrointestinal symptoms are the most common. Ascites is present in almost 40 per cent of the patients whose tumor measures more than 6 cm. in diameter. In 2 to 3 per cent of the patients with ascites, hydrothorax is also present. This is the so-called Meigs syndrome.

At operation the ovarian fibroma is found to be solid, encapsulated, hard and gray white. In more than 90 per cent of the cases the tumor is unilateral.

The cut surface is white with small areas showing a yellow tinge. Occasionally a small hemorrhagic area is noted. The surface is trabeculated with a whorl arrangement.

the process of seed metastasis. Cells from an external papillary growth form implants throughout the peritoneal cavity. This group of tumors, therefore, should be classified separately from those tumors which are both clinically and histologically malignant.

Grossly the cyst is thin walled and multiloculated. Its contents are serous, varying from yellow to brown in color. A chocolate color results from an intracystic hemorrhage.

The presence and location of the papillary growths vary. The internal proliferative areas may involve only a small area of the cyst wall, but the usual appearance is that of closely matted together papillary growths that almost fill the cyst cavity. The papillae are cauliflower like and friable and the same type may appear on the external surface.

These tumors are usually fixed to surrounding structures and this makes a clean surgical removal difficult. Metastatic implants are most frequently seen in the cul-de-sac. However the process may be so extensive that all the abdominal viscera are involved.

Microscopically the epithelial cells appear in multiple layers. The cells are anaplastic with large, dark, irregular nuclei, characteristic of carcinoma cells. In some tumors, areas with benign epithelium are seen in contrast to other areas with malignant changes. Glandular structures are common. When stromal invasion has occurred cell masses resembling solid adenoma may be present.

The prognosis of patients with serous cystadenocarcinoma which is malignant clinically but benign histologically is not too bad. The tumor should be treated, however as any malignant lesion of the ovary. Patients with tumors which are histologically malignant have a five-year survival rate of 18 to 20 per cent. In patients in whom wide spread metastatic implants are present, the prognosis is graver. It is very heartening, however to find an occasional patient having serous cystadenocarcinoma with wide spread metastasis that responds dramatically to therapy.

Pseudomucinous cystadenocarcinoma. The frequency of this tumor is much less than that of the serous variety; the incidence of malignancy is also much less.

Malignant pseudomucinous cysts are usually unilateral and relatively large. They may fill the entire abdominal cavity. External papillae are rare. On section, the tumor is seen to be composed of numerous irregular shaped cysts with solid areas of varying size.

These areas contain the malignant portion of the tumor.

Microscopically histologic sections of benign tall cylindrical cells of the picket fence type are usually present but the solid areas have a more adenomatous appearance. The glands are lined by multiple layers of anaplastic cells. Nuclear changes and mitoses are present and solid cellular masses invade the fibrous stroma.

The prognosis of the patient having pseudomucinous cystadenocarcinoma is dependent on the clinical findings at the time of operation. Many of these tumors with early malignant changes can be removed surgically with good results. In patients in whom stromal invasion has occurred or in whom obvious metastasis is present the prognosis is poor.

SOLID TERATOMA

Solid teratoma of the ovary is a very rare tumor. It may occur at any age but is more common in the young person, the majority of patients being under twenty years of age. The tumor is rare in the postmenopausal woman.

Teratomas are almost always unilateral. They grow rapidly and wide metastasis is common.

Grossly they are firm, solid tumors with a tendency for early capsular perforation with involvement of adjacent organs. On section their appearance is variable. Portions of any tissue or organ in the body may be reproduced, though usually in the rudimentary form. Even a partially developed fetus may be seen.

The therapy is surgical removal followed by postoperative x ray therapy.

The prognosis is poor. More than 90 per cent of the subjects will die within one year after the onset of therapy.

SARCOMA OF THE OVARY

Sarcoma of the ovary is very rare. No age groups are exempt. However there appears to be an increased incidence in the younger individual. In some series, 30 to 40 per cent of the ovarian tumors in children are sarcomatous.

Grossly the tumor is solid with a nodular surface. The color varies from gray to yellow. Cystic degeneration is common and on section gives the tumor a Swiss-cheese appearance. The consistency varies with the degree of cellularity. The more cellular tumors are soft and brainlike.

Microscopically the picture is variable.

from the specimen obtained at operation. If the tumor growth has broken through the capsule and formed peritoneal or visceral implants the diagnosis is easy. The diagnosis is more difficult when the tumor is encapsulated. The majority of the encapsulated ovarian malignant growths are less than 5 cm. in diameter. In some series the tumors are bilateral in 80 to 90 per cent of the subjects.

In general, the tumor surface is irregular with areas of nodularity. On palpation, areas of softness are felt throughout the tumor. The mass may vary from round to kidney shaped. On cross section, the color is usually grayish yellow or grayish pink. The consistency is variable, but the majority have soft, friable and brainlike tissue.

In the advanced cases the tumor growth invades the adjacent structures, and its normal anatomic relationship is completely lost. The growth may fill the entire pelvis and incorporate the small and large bowel. One sees tumor implants on the peritoneum and all of the abdominal organs. The omentum is frequently adherent to the pelvic mass and large neoplastic growths are found throughout the omentum.

The treatment of carcinoma of the ovary is a combination of radiation and surgery. Preoperative x ray offers much to younger patients with a sound diagnosis and to older patients in whom the ovarian function need not be conserved. Cell blocks and Papanicolaou smears of ascitic fluids and fluid aspirated from the cul-de-sac are helpful in the suspicious case. Preoperative therapy is given through two pelvic portals— anterior and posterior. A dosage of 800 r to 1200 r is given through each portal. Surgery follows eight to fourteen days after the completion of the radiation therapy. The structures are edematous, the cleavage planes are better and a more complete removal of the tumor is possible.

When at operation a malignant tumor of the ovary is found with evidence of pelvic metastasis, bilateral salpingo-oophorectomy and complete removal of the omentum is the procedure of choice. The uterus is not removed. It is preserved for the use of intra uterine radium two to three weeks postoperatively. With this procedure the amount of radiation to the cul-de-sac can be greatly increased. The dosage varies with the length of the uterine cavity.

Deep x ray therapy is begun immediately after the operation. The therapy is given to

the lower abdomen and pelvis in small fractionated doses for three to six months. Therapy is also given to the upper abdominal quadrants in patients in whom definite implants are found in this area. Hormonal therapy with 20 to 30 mg. of testosterone given daily seems to help.

In patients with ascites, preoperative x ray therapy is given to large anterior and posterior fields. This is followed by fractionated doses of deep therapy directed at the tumor growth. Only after adequate shrinkage and mobility of the tumor have occurred should surgery be performed. It should not be rushed.

In patients having inoperable tumors, palliative therapy helps. These patients are given both intrauterine radium and fractionated doses of deep x ray therapy. In subjects with ascites, injection of radioactive gold intraperitoneally may control the ascitic fluid formation for as long as four to six months. The dose is 125 to 150 mc, and this can be repeated at intervals. Generally the use of palliative therapy can add one to two years to a patient's life.

In those patients in whom the malignant growth is discovered at operation and who show no evidence of peritoneal spread, the operation should be bilateral salpingo-oophorectomy and total hysterectomy. Deep x ray therapy follows in the postoperative period.

Primary Cystic Carcinoma. Much controversy exists concerning the origin of the cystic carcinomas. Undoubtedly many of the cystic malignant growths arise from a benign cystadenoma, some of the cystic carcinomas are primary growths. The tumors from which cystic carcinomas arise are the cystadenomas, either pseudomucinous or serous, and the dermoids. The treatment of the cystadenocarcinoma is similar in all respects to the treatment of the primary solid carcinoma.

Serous cystadenocarcinoma. In addition to being cystic, these tumors are bilateral. Most are characterized by papillary proliferations. These are both internal and external papillary types.

The pathologic diagnosis does not at all times agree with the clinical behavior of the tumor. For example, one may find bilateral cystic tumors with external papillations and widespread implants throughout the abdomen at the time of operation, the tumor is clinically malignant, but, on microscopic examination, the pathologist reports a benign tumor. Such a situation results from

these lesions to subside and recur in a fashion which may seem never ending. It is difficult to eradicate the staphylococci they like the warmth and moisture of the vulva. But their presence is not the sole factor. Here as in all infections there is a general as well as a local factor. As long as the body defenses are in good working order the bacteria give no trouble, but when resistance becomes low infection occurs. Generally one looks for chronic infection, malnutrition, anemia and diabetes as well as debilitating conditions. Locally trauma from scratching, pruritus, intertrigo or discharge may be found.

Furunculosis. Pain and itching attract the attention of the patient to the parts where she finds a red, tender and slightly elevated nodule. This can either regress or proceed onward to suppuration. In the latter case it will become more brown, larger and very tender. In time, central necrosis occurs and the apex becomes yellow, indicating that the boil is ripe. Following rupture or incision, the boil drains until the core or central necrotic mass separates. The condition is usually minor but can be very painful, troublesome and annoying.

Folliculitis. In follicular vulvitis the infection occurs within a hair follicle and forms a lesion which may be similar to a boil. At other times the lesions are superficial and usually multiple. They have a tendency to occur in groups. The lesion is a pustule with a hair projecting from its center. Around the pustule there is a zone of redness.

Hidradenitis of the vulva. Vulvar hidradenitis is similar to axillary hidradenitis but is rarer. The infection occurs in the large coiled apocrine glands. The lesion is somewhat similar to a boil but is situated in a deeper structure. It is slower in coming to a head and has a greater tendency to burrow. As a rule, the infection seems to be of a lower grade, taking longer to suppurate and reach the surface. The chronicity of the condition is disturbing to the patient.

Treatment of staphylococcal infections of the vulva. The principles of treatment of staphylococcal infections of the vulva are definite. First, the condition of the individual lesion is attended to: incision is made when suppuration is present. Next, the vulva is decontaminated by the daily use of a detergent with a skin antiseptic. This is followed with an appropriate antibiotic ointment. Lastly the general resistance is built up and

generally antibiotics are used only in severe cases.

Herpes Vulvae. In many respects herpes vulvae resembles the common fever blisters occurring on the lips. A sensation of heat and tension precedes the development of blistery lesions. These form on a red indurated base. The lesions are painful and spread easily. In patients with simple cases the application of antibiotic ointments with cortisone is effective. In patients with recurrent or intractable cases, weekly inoculation with smallpox vaccine builds up resistance and helps to overcome the infection.

X-ray Burns of the Vulva. Unfortunately one of the more frequent types of acute inflammation of the vulva is the result of x-ray therapy given to patients with pruritus vulvae. The skin of the vulva is very sensitive and can tolerate only a small amount of deep x-ray therapy and in these particular subjects the results have been quite unsatisfactory. The pruritus usually persists and the end result is a reddened, swollen, acutely infected vulva.

Bartholinitis. This is an infection of the Bartholin's glands which are located at 4 and 8 o'clock in relation to the vaginal outlet. A frequent cause of the infection is the gonococcus. Streptococcus or Staphylococcus may also be the etiologic agent. Even *Trichomonas* has been found in many chronically infected Bartholin's glands.

In the acute stage, the gland is swollen, tender, hot and painful. On careful examination the duct of the gland may be seen to exude a small amount of pus with pressure on the swollen gland. Occasionally the infection will subside gradually if the duct remains open and acts as a drain for the gland. The resolution is seldom complete and the usual result is a chronic infection of the Bartholin's gland. The other alternative of the acute stage is abscess formation. The abscess will either drain spontaneously or it must be incised. In spontaneous drainage the usual result is inadequate drainage and a prolonged course of infection and chronic inflammation. If the abscess is incised adequately the course is much shorter and much more gratifying.

In patients with chronic bartholinitis, in duration may be present with a slight enlargement of the gland. The areas may be asymptomatic for years. On the other hand, acute exacerbations may occur with abscess formation or cyst formation due to an occlusion of one of the ducts. The size of the

The cells may be spindle like or of the round cell variety.

The treatment is surgical. The tumor is very malignant and the prognosis is poor.

METASTATIC OVARIAN CARCINOMA

The ovaries are well known sites for involvement by metastatic implants. Two abdominal areas furnish the majority of the secondary ovarian malignant processes.

First, carcinoma arising from the pelvic viscera may involve the ovary. The primary lesion may be the corpus uteri, fallopian tubes or the pelvic portion of the large bowel. Lymphatic metastasis or tubal regurgitation is the method of spread particularly in uterine carcinomas.

The other source is the viscera of the upper abdomen. Carcinomas of the gastrointestinal tract, gallbladder and pancreas are the usual primary lesions. The secondary metastatic tumors in the ovary are known as Krukenberg tumors.

Grossly, these tumor growths are solid and usually about the size of a man's fist. They are rarely larger than 6 or 7 cm in diameter. The tumors are almost always bilateral. They have a tendency to retain the general shape of the ovary and not infrequently are lobulated. The external surface is relatively smooth and adhesions to surrounding structures are rare. There is usually a well-developed capsule.

On section, the appearance is variable. Some areas are firm, others are spongy. Hemorrhage or cystic degeneration may be seen.

On microscopic examination the stroma is seen to be fibrinous with a myomatous appearance in areas due to edema and degeneration. Scattered throughout the stroma are nests or strands of round swollen cells. The cells are vacuolated. The nuclei appear as long, crescent-shaped bodies situated against the cell membrane. These epithelial cells have undergone mucoid change and give the cells the characteristic signet ring appearance.

Clinically the symptoms referable to the primary neoplasm of the upper abdomen are overshadowed by those of the ovarian neoplasm in 30 to 40 per cent of the patients. Ascites may be present in approximately one-third of the subjects. The important point for the surgeon to keep in mind is that bilateral solid, lobulated ovarian tumors may be metastatic and a thorough investigation of the upper abdomen is most essential.

THE VULVA

Skin covers the specialized tissues which make up the vulva. Of course, this skin is somewhat different from that on other portions of the body but it is skin nevertheless and general skin diseases do affect it. The vulva has an environment all its own and this modifies the diseases and makes it difficult to diagnose them. To complicate the situation a little more, one must recognize another group of diseases which largely limit themselves to the vulva and have little or no general manifestations.

Women are very conscious of their vulva and attach an unusual significance to its diseases. Sometimes even the actual disease stimulus is cerebral in nature and the vulva symptoms are but distant manifestations of deep mental unrest. The vulva, then, is an area in which dermatology, gynecology and psychiatry overlap.

Intertrigo. Intertrigo by definition, is a chafe or a chafed patch of skin. It is caused by the rubbing together of two skin surfaces. In fat women the inner side of the thigh rubs the outer side of the labia majora. When the skin is thin, as in the young and the old, less rubbing produces more of a chafe. Moisture from excessive sweating, discharge or urinary incontinence macerates the skin. To make matters worse, itching occurs and results in scratching, infections with the ordinary bacteria or with fungi follow. In diagnosis the first consideration is to determine the presence or absence of a fungus infection. When fungi are not present, treatment depends on simple measures to prevent chafing, dry the moisture and overcome infection.

Staphylococcal Infections on the Vulva. Staphylococcal infections of the vulva cause furunculosis, folliculitis and hidradenitis. These three conditions are often described as separate, clear-cut diseases, which they really are not. While one type of infection overshadows the others, all of the lesions may not be of the same type. With furunculosis the infection takes place in a sweat gland, with folliculitis, in a hair follicle, and with hidradenitis, in an apocrine gland. Each of these lesions has a different appearance because it occurs in a different skin structure. One can readily see when a lesion suppurates and ruptures that it will contaminate the skin of the area. This can cause lesions of the original type or of the other types depending on the skin structure infected. This accounts for the tendency of

structive in nature. The therapy of syphilitic lesions of the vulva has been revolutionized since the advent of penicillin and its various forms of administration.

Tuberculous Vulvitis. This is a rare condition. As a general rule tuberculosis in the female genital system is usually secondary to tuberculosis of the fallopian tubes. Tuberculosis of the vulva can be primary, particularly in patients whose husbands have tuberculous epididymitis. The lesions are usually irregular small punched-out ulcers that are hard to heal by the usual methods of therapy. Making the diagnosis is frequently difficult. Many times it is impossible to differentiate the lesions even by biopsy from those of syphilitic chancre. The use of smears and tumor biopsy tissue stained for the acid fast bacillus is often diagnostic. Guinea pig inoculations are an old but important means of diagnosis and can be helpful when other means fail.

Cancer of the Vulva. An ulcerative lesion of the vulva, particularly in the elderly woman, must be considered as cancer until proved otherwise. The average age of patients with carcinoma of the vulva is fifty-five to sixty years. The carcinoma is squamous cell in type. Under the microscope, it has a very characteristic appearance; the cells are very highly differentiated with frequent pearl formation. Usually the lesion develops as a small nodule which later ulcerates or becomes papillomatous. Unfortunately, the physician rarely sees the patient when the lesion is small. In one recent study the average diameter of the lesion was 5 cm. when the patient first consulted a physician. The lesion can become extensive and involve the urethra and the opposite labia forming a kissing type of ulceration. The disease spreads by direct extension and by way of the lymphatics. Lymphatic extension is thought to be by the process of embolism. The superficial femoral and inguinal nodes are frequently involved early in the course of the disease. Lymphatic extension can occur to the nodes on the same side as the lesion or to the opposite side; this results from the rich anastomosis which exists between the lymphatic channels of each side of the vulva. The deep femoral and deep inguinal nodes and the nodes upward along the iliac vessels are involved at a later stage of the disease. It is important to remember that the presence of palpable nodes is not a certain indication of carcinomatous involvement, in many pa-

tients the nodes are palpable because of inflammatory changes. On the other hand the lack of palpable nodes does not exclude node involvement.

It is the consensus that surgery is the preferred form of therapy in carcinoma of the vulva, the operation has progressed from simple vulvectomy to a far more radical excision of the vulva and the adjacent lymph nodes. In this country Taussig was the pioneer in advocating an extensive en bloc dissection of the vulva and lymph nodes. The operation calls for radical removal of the vulva and large wedges of skin extending from the vulvar incisions toward each inguinal area plus a bilateral node dissection including all of the superficial and deep nodes, and an extraperitoneal dissection of the nodes up to the bifurcation of the aorta. In many patients the skin cannot be primarily completely approximated. However this is of less significance than in other areas as rapid granulation and epithelization of the open wounds result in an acceptable scar. Infection of the wound postoperatively is present in most patients. This is combated by the use of antibiotics and drainage. The accumulation of serous fluid and lymph beneath the skin flap is treated by aspiration or by the insertion of catheters or polyethylene tubes in the dependent areas and the use of constant suction. Thrombophlebitis and phlebothrombosis must be constantly guarded against in the patients. The use of leg exercise of elastic support and ambulation is good prophylaxis.

The employment of radiation is extremely limited except in patients in whom urethral involvement is present. Radon seeds are used by some physicians in subjects having local recurrences. In the experience of many the use of electrocoagulation has been more successful in the treatment of local recurrences than has any other form of therapy. The Radiumhemmet in Sweden uses electrocoagulation of the vulva instead of surgical vulvectomy as a primary form of therapy.

Kraurosis and Leukoplakia. Kraurosis is a shrinkage of the tissues of the vulva. It is seen in elderly women in patients following radiation menopause and in patients who have had radiation therapy for carcinoma of the cervix. The condition consists of a loss of subcutaneous fat; the labia become flattened, the skin is thin and is usually reddened. There is a narrowing of the introitus and, as a result, dyspareunia is a common

cyst depends on the site of the duct blockage. The cysts may vary from 1 to 5 cm. in diameter. The treatment consists of excision or marsupialization of the cyst. In recent years because of the extreme vascularity of the tissue and the painfulness of the condition, these cysts have been marsupialized by making a longitudinal incision across their entire diameter and suturing the linings to the skin surface. They are packed open for forty-eight to seventy-two hours and thereafter the patient is instructed to keep the opening patent by inserting her finger into the incision each day. The end result is a small dimple at the site of the previous cyst. Hospitalization of these patients is usually for a two- to three-day period. The tenderness is markedly less and the end results are as good as the complete removal of the cystic structure.

Gonorrhea. Gonorrheal urethritis and gonorrheal infections of the Skene's ducts still occur, however, the frequency in both private practice and clinic practice has decreased markedly with the appearance of chemotherapy. The urethritis does not play a prominent role in the over-all picture of infection in the female. The female urethra is short and in most patients there is a tendency for spontaneous resolution of the infection of the urethra. Trigonitis, however, may be persistent. On the other hand, the drainage of the Skene's ducts is inadequate and persistent infections are common. At one time it was necessary to destroy these ducts by the use of the cautery. However, with the advent of bactericidal drugs the results are quite different.

Chancroid. Ducrey's bacillus causes this ulcerative lesion; it is sometimes called a dirty chancre or a soft chancre. The lesions, usually multiple and very ragged in appearance, have a purulent ulcerative surface. They are soft and not indurated as in syphilis; edema frequently is present if they are on the labia majora. A characteristic feature of the disease is secondary inguinal lymphadenitis. Frequently the lymph glands suppurate and form inguinal abscesses. These are known as buboes.

The diagnosis can be made by smears of the lesion with the demonstration of a gram-negative, short, coccus-like rod. The microorganisms frequently appear in pairs or chains. Cultures are used and are most reliable. The intradermal skin test, developed by Greenblatt, is useful to confirm the diagnosis. It is diagnostic three to six weeks after the initial infection.

In the treatment of Ducrey infection, sulfadiazine gives very satisfactory results. The dosage is 1 gm. every four hours. Several of the wider range antibiotics also can be helpful. It is better to aspirate the buboes than to incise them.

Granuloma Inguinale. This is a chronic skin infection that is usually very distressing to the patient. Developing as a small papule and followed by ulceration, it spreads over the entire genital area. This condition is limited almost exclusively to members of the Negro race. The etiologic agent is the Donovan body. Diagnosis depends on demonstration of these bodies. The smear is best stained by either Wright's or Giemsa's method. Tissue biopsy is important in the diagnosis of this lesion. Good surface smears are difficult to obtain because of the large amount of cellular debris that is present. For this reason the bodies are more easily seen in smears from tissue or in stained sections. In treatment, good results follow the use of Chloromycetin and Terramycin.

Lymphogranuloma Inguinale. This condition is caused by a virus. The lesion usually develops as a superficial ulcer followed by multiple ulceration and inguinal adenitis. The spread of the infection is by the lymphatics. Diagnostically the Frei test is important. In the treatment of lymphogranuloma inguinale sulfadiazine has been an old stand by. Terramycin, Chloromycetin and other wide-range antibiotics are more frequently used at the present time.

Syphilis. As in the male the primary lesion of syphilis is the chancre. In the female, it is most often seen on the labia. It is firm, hard and nodular in character. It is usually single, however, sometimes there may be a kissing type of lesion. The chancre is not as commonly seen in the female as in the male, because of its relative painlessness and the inaccessibility of the female vulva to personal inspection. The diagnosis is made by the darkfield examination. Reactions to serologic tests are usually positive within two to three weeks after a primary lesion occurs.

The condyloma latum, the flat, moist papule of secondary syphilis, is important. It appears slightly elevated, but with a flat surface that looks grayish and necrotic. The lesions are multiple and may involve only a small area of the vulva or, on the other hand, they may involve the entire vulva and perineal region. The tertiary lesions of the vulva have been seen very rarely in the past ten years. When present, they consist of extensive gummatous ulcerati-
on are de-

- Martzkoff, A. H.: Epidermoid Carcinoma of Cervix Uteri, Histologic Study to Determine Resemblance between Biopsy Specimens and Latent Tumor Obtained by Radical Panhysterectomy. *Am. J. Obst. & Gynec.* 16, 578, 1928.
- Mills, J. V.: Cancer of Ovary. *Surg. Gynec. & Obst.* 144, 1910.
- Mills, J. V.: Endometriosis—Its Significance. *Ann. Surg.* 114, 960, 1911.
- Mills, J. V.: *Surgical Treatment of Carcinoma of the Cervix*. New York, Grune & Stratton, 1931.
- Mulge, J. V., and Cass, J. W.: Fibroma of Ovary with Ascites and Hydrothorax, with Report of 7 Cases. *Am. J. Obst. & Gynec.* 32, 219, 1937.
- Novak, E., and Novak, E. R.: *Gynecologic and Obstetric Pathology*, 11th ed. Philadelphia, W. B. Saunders Company 1938.
- Novak, E., and Yul, E.: Relation of Endometrial Hyperplasia to Adenocarcinoma of Uterus. *Am. J. Obst. & Gynec.* 32, 674, 1938.
- Papantolaiou, G. V., and Traut, H. F.: *Diagnosis of Uterine Cancer by the Vaginal Smear*. New York, The Commonwealth Fund, 1943.
- Tchana, H., and Amurck, J.: *Operative Gynecology*. Philadelphia, J. B. Lippincott Company 1934.
- Reynolds, S. R. M.: *Physiology of the Uterus*. New York, Paul B. Hoeber 1919.
- Sampson, J. A.: Peritoneal Endometriosis Due to Menstrual Dissemination of Endometrial Tissue into Peritoneal Cavity. *Am. J. Obst. & Gynec.* 11: 422, 1927.
- Tausig, F.: *Diseases of Vulva*. New York, D. Appleton-Century Company Inc., 1921.
- Te Linder, R. W.: *Operative Gynecology*, 2nd ed. Philadelphia, J. B. Lippincott Company 1933.
- Truelsen, F.: *Cancer of the Uterine Cervix*. London, H. K. Lewis & Company Ltd., 1919.
- Trussell, R. E.: *Trichomonas Vaginalis and Trichomoniasis*. Springfield, Ill., Charles C. Thomas Publisher 1917.
- Way, S. A.: *Malignant Disease of the Female Genital Tract*. Philadelphia, The Blakiston Company 1931.
- White, J. C., Smithwick, R. H., and Simone, F. A.: *The Autonomic Nervous System*, 3rd ed. New York, The Macmillan Company 1932.

symptom. The pasty thin skin frequently develops infection. The result is a secondary condition known as chronic atrophic vulvitis. At times this condition may become acute with redness, tenderness and edema of the vulva. Leukoplakia like changes may develop.

There are two types of leukoplakia—atrophy and hypertrophy. Hypertrophic leukoplakia is characterized clinically by elevated whitish, parchment like areas with a tendency to fissure formation and infection. Pruritus, dysuria and vulvar irritations are prominent symptoms of this condition. Microscopically there is an increased keratinization of the surface of the squamous epithelium with an increase of cellularity of the basal layers and with pronounced invagination of the rete pegs into the underlying tissue. Mitotic figures are common and hyperactivity of the cells is of frequent occurrence. In the atrophic form there is a thinning of mucous membrane and a loss of collagen in the underlying stroma.

Of great importance is the relationship of leukoplakia to carcinoma of the vulva. It has been stated by some that one half of all cases of carcinoma of the vulva are preceded by a leukoplakic vulvitis. The therapy of leukoplakia varies widely. The usual patient will relate a large number of different types of therapy which have been tried in her particular case. They include use of various salves, ointments, dyes and x-ray. In general, these therapies have not been too satisfactory. In some of the patients with an atrophic type of the disease the use of estrogen creams has been of benefit. Recently the advent of cortisone ointments has improved the results in some of these patients. On the other hand, the usual course consists of multiple forms of therapy followed in time by vulvectomy. In most subjects vulvectomy is unnecessarily delayed and frequently carcinomatous changes are present before it is performed. The patient's fear of mutilation has much to do with this delay. Fortunately mutilation is not nearly as great as expected and the cosmetic result is generally well accepted. The relief of the itching and irritation is so great that the patients readily accept their postoperative states and are well pleased with their operations.

READING REFERENCES

- Adair, F. L., and Davis, M. E. Chronic Atrophic Dermatitis of Vulva. *Surg. Gynec. & Obst.* 61: 433, 1935.
- Annual Report of the Results of Radiotherapy in Carcinoma of the Uterine Cervix, Stockholm, Sweden, 1949, vol. 8.
- Beck, A. C., and Rosenthal, A. H.: *Obstetrical Practice*. 7th ed. Baltimore, Williams & Wilkins Company 1953.
- Bernatine, J. B. and Rakoff, A. E.: *Vaginal Infections, Infestations and Discharges*. New York, Blakiston Company 1953.
- Berven, E. G.: Carcinoma of Vulva, Treatment of Cancer of Vulva, Symposium. *Brit. J. Radiol.* 2: 498, 1949.
- Bonney, V.: *Textbook of Gynecologic Surgery*. New York, Paul B. Hoeber 1948.
- Brown, A. B., Gilbert, R. A., and Te Linde, R. W.: *Pelvic Tuberculosis*. *Obst. & Gynec.* 2: 476, 1953.
- Burch, J. C. and Lavelle, H. T.: *Hysterectomy*. Springfield, Ill., Charles C. Thomas, 1955.
- Burch, J. C., and Phelps, D.: *General Concepts of Etiology of Functional Menstrual Disorders*. *South. M. J.* 35: 150, 1942.
- Burch, J. C., and Phelps, D.: *Endometrial Biopsy*. *J. Clin. Endocrinol.* 3: 475, 1943.
- Burch, L. E., and Sestchuk, J.: *Ectopic Gestation, Diagnostic Value of Cul-de-Sac Aspiration*. *Am. J. Obst. & Gynec.* 50: 785, 1945.
- Concepcion, J. A.: *Gynecologic Cancer*. 2nd ed. Baltimore, Williams & Wilkins Company 1950.
- Everett, H. S.: *The Treatment of Vulvovaginitis in Children*. In *Progress in Gynecology* edited by J. V. Meigs and S. H. Sturgis. New York, Gross & Stratton, 1950.
- Ewing, J.: *Neoplastic Diseases*. Philadelphia, W. B. Saunders Company 1940.
- Gest, S.: *Ovarian Tumors*. New York, Paul B. Hoeber 1942.
- Greenblatt, R. B.: *Office Endocrinology*. 4th ed. Springfield, Ill., Charles C. Thomas, 1953.
- Greenblatt, R. B., Diem, R. B., Prod, E. R., and Torpin, R.: *Experimental and Clinical Gynecology*. *Inguinale*. *J.A.M.A.* 123: 1109, 1939.
- Greenhill, J. P.: *Obstetrics*, 11th ed. Philadelphia, W. B. Saunders Company 1935.
- Hamblen, E. C.: *Endocrinology of Women*. Springfield, Ill., Charles C. Thomas, 1948.
- Hertig, A. T. and Sommer, S. C.: *Genesis of Endometrial Carcinoma; Study of Prior Biopsies*. *Cancer* 2: 964, 1949.
- Hoseltine, H. C., and Beckett, E. S.: *Specific Treatment of Vaginal Mycosis*. *Am. J. Obst. & Gynec.* 58: 533, 1949.
- Heyman, J.: *Radiotherapeutic Treatment of Cancer Corporis Uteri*. *Brit. J. Radiol.* 20: 85, 1947.
- Jacoby, A. and Bobler, D. L.: *Clinical Evaluation of Sulfonamides in Cervical and Vaginal Therapy*. *Am. J. Obst. & Gynec.* 63: 1848, 1952.
- Johnson, W. O.: *Study of 245 Cases of Ruptured Ectopic Pregnancy*. *Am. J. Obst. & Gynec.* 64: 1102, 1952.
- Jones, C. P. and others: *Mycotic Vulvovaginitis and Vaginal Fungi*. Report of 280 Patients. *Am. J. Obst. & Gynec.* 54: 738, 1947.
- Jones, W. N., Howe, E. H., and French, J. H.: *The Role of Antibiotics in Management of Incomplete Abortions*. *Am. J. Obst. & Gynec.* 67: 823, 1954.
- Karnaky, K. J.: *Use of Stilbestrol for Endometriosis*. Preliminary Report. *South. M. J.* 41: 1109, 1948.
- Kotliester, H. L.: *Carcinoma of the Female Genitalia*. Baltimore, Williams & Wilkins Company 1953.

An *incomplete fracture* is one in which the continuity of the bone is only partially divided. There are several varieties. A *fissure fracture* is one in which there is a crack partially through the bone. This may be either transverse or longitudinal. A *depressed fracture* may involve only the outer table of the skull not passing completely through the bone. A bullet wound may create a *puncture fracture* without a break across the bone. A *greenstick fracture* is one which has occurred from marked bending, so that the bone is splintered on the convex side, but remains intact on the concave side like a splintered branch of a green tree.

A *complete fracture* is one in which the entire continuity of the bone is divided. The fracture line if single may be transverse, oblique, spiral or even longitudinal and often there is more than one line of fracture. A *comminuted fracture* is one in which there are two or more lines of fracture which divide the bone into more than two fragments. At a joint, this may be a combination of a transverse and a longitudinal fracture producing a T fracture or a combination of two oblique fractures producing a V fracture. An *impacted fracture* is one in which, with or without lateral displacement the inner cortical bone is telescoped into the softer cancellous bone. This occurs particularly at the neck of the humerus and of the femur. A *compression fracture* is one in which two opposite surfaces of the bone are driven closer together than normal. This usually is noted in cancellous bone with considerable fine comminution, often resulting in a definite diminution in the area of bone shown in the x ray picture as in fractures of the vertebrae. A *sprain fracture* is a sprain in which a small fragment of one of the bones has been pulled off usually at the site of a ligamentous attachment. A *spontaneous fracture* is one which occurs without apparent competent exciting cause. Strictly speaking, it is not spontaneous as the break is necessarily caused by trauma, even though this is slight. It is usually found in patients with bone atrophy osteogenesis imperfecta or general conditions affecting bone. A *pathologic fracture* is one which occurs without apparent competent exciting cause at the site of local disease of the bone.

Displacements of the fragments may be lateral, angular longitudinal and rotary, or a combination of these types. The displacements interfere with the proper axis of an extremity. Posterior angulation produces what is known as *dishing*. Longitudinal dis-

placement may produce shortening of an extremity called *overriding*, or it may result in separation of the fragments as seen in fractures of the patella or olecranon or in overpull in the use of traction.

A fracture is described as being located either in the shaft or in the extremities. At the ends of the bones there is a further designation according to the anatomic part broken e.g. surgical neck, tuberosity, condyle, head or articular fracture or epiphyseal separation. An articular fracture is one in which the fracture line runs across the attachment of the joint capsule. An intra-articular fracture lies entirely within the joint. An epiphyseal separation is a fracture which passes wholly or in part through the epiphyseal line. It is often unrecognized but is of great importance because bony union of the epiphysis may result with arrest of growth. A *subperiosteal fracture* is a complete fracture without displacement in which it is believed that the periosteum has not been torn. Multiple fractures may be at different levels in the same bone or in different bones. The term *complicated fracture* is sometimes used when recovery will be complicated because of damage to artery, nerve or joint.

Etiology. Fractures, particularly of the clavicle, elbow and forearm, are common in children between the ages of two and four years on account of unsteady gait and frequent falls. Between the ages of four and six years, many greenstick fractures occur because the bones are soft. Between six and twelve years, if a fracture occurs near a joint, there is likely to be epiphyseal separation. The incidence of fractures increases from six years on, reaching its maximum between the ages of thirty and forty years; that is, it coincides with the age of greatest activity. On account of bone atrophy fractures of the neck of the humerus and of the neck of the femur are particularly common in old age.

The incidence is about the same in boys and girls up to four or five years of age, after which fractures occur more commonly in males up to forty-five years old. This is undoubtedly a result of the more active life and more hazardous trades of males. Some authors state that fractures are from seven to ten times more frequent among males than among females during this period. After forty-five years, the number is nearly equal in the sexes, with a slight preponderance among females in advanced age.

Bone atrophy is a predisposing cause of

THE BONES AND JOINTS

Considerations in the Treatment of Closed and Open Fractures

By ROBERT H. KENNEDY M D

ROBERT HAYWARD KENNEDY has been active for many years in educating the medical profession and the laity to the many facets in the surgery of trauma in which they have a common responsibility. He is well qualified because of his wide experience. Educated at Amherst College and Columbia University, he has spent his professional life in New York City. Dr. Kennedy was formerly Professor of Clinical Surgery at New York University Post-Graduate Medical School.

A fracture is the breaking of a bone or cartilage usually resulting from external violence. Injury to the soft parts overlying a fracture is an almost invariable part of the injury and always requires consideration.

Varieties. A *closed fracture* is one in which there is no communication between the bone at the site of fracture and the outside air. Usually this means that there is no overlying wound of the skin or mucous membrane. An *open fracture* is one in which there is a communication between the site of the fracture and the outside air through

an opening in the skin or mucous membrane. This may have been caused either from within or from without. It is possible for an overlying wound not to communicate with the fracture, this then being a closed fracture, although it is preferable for purposes of treatment to consider it as open. If an open operation is performed on a closed fracture an open fracture is produced. If the unbroken skin and soft parts slough away in a closed fracture an open fracture may be produced.

Fractures may be incomplete or complete.

ment, but if it is lived up to it is obvious that the splinting will rarely be done by a physician. Splinting must be considered as only a means of aiding transportation not a part of the definite treatment of the fracture. The physician must be prepared to instruct the public on the most effective manner of transporting a patient with a fracture to the physician's office or to a hospital. In general those trained by the Red Cross and Boy Scouts know more about first aid than most physicians, because little responsibility has been accepted by the physician until the injured person arrives at the office or hospital.

To quote from *An Outline of the Treatment of Fractures* issued by the American College of Surgeons

The injuries resulting from fractures are not limited to those occurring at the time of the accident. Unwise attempts to use the injured extremity may cause or increase displacement of fragments, increase the lacerations of soft parts, and perhaps lead to penetration of the skin by the ends of the bone. Similar additional trauma is often due to the awkward efforts of the bystander. A man is struck by an automobile thus breaking his leg. Except for the broken bone without displacement, the original injury may be merely a slight periosteal tear and a mild contusion of the soft parts, but he is helped to his feet and the leg gives way and the fragments slide by each other thus stripping off the periosteum and tearing the muscles. He falls to the ground only to be picked up and carried to the sidewalk with the leg dangling. Lacerated blood vessels are torn and the end of the bone cooks through the fascia, perhaps the skin and even the trouser. He is laid at rest with a coat beneath his head and surrounded by people anxious to help. Someone sees that his leg is crooked and straightens it out. The exposed end of the bone re-enters the wound with a bit of trouser and the dirt of the street. He is lifted up and carried to a car or ambulance. This time someone carries the injured leg with better intentions than coordination and the ends of the bone are charmed around in their bed of lacerated tissues and the contaminating organisms are well disseminated throughout the area. During his ride and in the transfer to the accident ward or the doctor's office unless he has been carefully splinted, there is more jolting and more damage. Would that his troubles were over but too often the sad story continues. Lack of sufficient protection as he is lifted to and from the x-ray table and as he is being anesthetized results in still more injury.

Compare this exaggerated picture with a similarly injured man who is allowed to remain where he is until a proper splint can be applied, or at least until he can have someone pull hard on his foot as he is being lifted and carried, whose examination is thorough but gently carried out, and whose treatment is instituted with but little additional injury. The difference in these two cases as regards period of disability and amount of permanent functional disturbance is great.

Principles of first aid (1) If suspicious

that a fracture is present, render care as for a fracture (2) combat any existing shock (3) avoid every unnecessary manipulation, (4) protect any existing wound by the best means available (5) splint effectively before transporting the patient wherever found and (6) transport carefully.

The injured person should first be examined rapidly in order to attempt to determine whether he is seriously hurt. The clothing usually does not need to be removed. In examining a patient try to keep all parts thoroughly covered except the part which is actually being examined at the moment. One should depend on pain, loss of function or deformity for the emergency diagnosis of fracture and should rarely if ever try to obtain crepitus or false point of motion. If the patient thinks that he felt a bone snap for emergency purposes one should be satisfied with his history and not attempt to prove the diagnosis on the street. If suspicious that a fracture is present always treat the patient as if the diagnosis of fracture had been confirmed.

The saving of life comes first and the saving of limb second. If the patient is in shock, this demands treatment before anything is done for the fracture. There is no use in splinting a limb carefully while neglecting to treat shock from which the patient may be dying. On the other hand, traction on a extremity may be the most effective means of combating shock. If morphine is available and the injured person is in pain, its administration is frequently the first and best treatment for shock. It allays pain and thereby quiets the patient's restlessness. The body heat must be maintained by sufficient extra covering.

If a wound is present which possibly connects with a fracture it should not be washed. A sterile compress should be placed over the wound preferably without the previous application of any antiseptic. Bleeding should be stopped. It is rare that this cannot be done with a firm compression bandage. Tourniquets should not be used until compression has proved of no avail. Death has been caused by an improperly applied tourniquet which allowed some arterial blood to enter the part and only shut off the venous return. It is common to see all bleeding stop as soon as a tourniquet is removed. When a tourniquet has to be applied it should not be removed until definitive shock therapy has been instituted.

Do not attempt to replace the fragments in an open fracture which has projecting

fracture in old age, from prolonged lack of use as in patients with paralysis or ankylosed joints and in those with general paresis, tabes and syringomyelia. Osteogenesis imperfecta, or idiopathic fragilitas ossium is an inherited tendency to spontaneous fracture. Generalized bone conditions such as rickets and osteomalacia and localized bone disease such as sarcoma, metastatic carcinoma, destruction of bone in acute osteomyelitis and bone cysts are favorable to the occurrence of fracture.

There are three exciting causes of fractures—direct violence, indirect violence and muscular action.

A fracture by *direct violence* is one in which the bone is broken immediately beneath the point of impact. Injury to the soft parts always accompanies the fracture which is quite frequently open. The line of fracture usually is transverse or slightly oblique and is often comminuted. The common example at present is found in a patient whose leg has been run over by the wheel of an automobile.

A fracture by *indirect violence* is one in which compression or bending is exerted at a distance so that a bone gives way at its weakest point. A typical example of the compression action of indirect violence is seen in a fall from a height in which the person lands on his heels. According to the position of the person landing, he may sustain comminuted fracture of the os calcis, fracture of the lower extremity of the tibia often with fissures running longitudinally, fracture of the shaft of both bones of the leg, compression fracture of one or more vertebrae or fracture of the base of the skull. Similarly a fall on the extended palm may result in a fracture of the lower extremity of the radius, the head of the radius, the lower extremity of the humerus, the surgical neck of the humerus or the clavicle. The site of the fracture varies both with the exact position of the skeleton at the moment of impact and with the age of the patient. The bending action of indirect violence is well seen in fractures sustained in turning the ankle in which the weak point of the fibula immediately proximal to the external malleolus is the frequent site of fracture. If at the same time that the bending action occurs there is a certain amount of rotation of the body on the foot, the fracture is often spiral rather than simply oblique. Fractures caused by indirect violence are less likely to be comminuted. If a fracture is open, it is usually opened from the inside by a fragment

of bone which pierces the skin. The injuries of the soft parts are those caused by the sharp ends of the fragmented bone. Fractures by indirect violence occur much more commonly than those by direct violence.

Fractures by *muscular action* are less common, the most frequent examples being fractures of the olecranon and patella. A sudden contraction of the triceps or the quadriceps usually because of an unexpected movement, fractures these bones transversely. More rarely a long bone is fractured by muscular action, e.g., the shaft of the humerus when the person is throwing a baseball or the shaft of the femur when a patient with tabes merely rolls over in bed. The mechanism of fracture may be due in an individual patient to a combination of any two or all three of the direct causes.

The comparative frequency of fractures in various bones is difficult to determine. Many series of such statistics have been published, including thousands of cases, but they are usually in entire disagreement as to the order of frequency. This is easy to understand. The usual site of the fractures seen in hospital wards is different from that seen in the dispensaries. A hospital in a residential district admits patients with fractures which are usually in other locations than those of patients from an industrial or congested district. A hospital with an ambulance service may have more lower extremity and skull fractures while a hospital without an ambulance may have more upper extremity fractures because the patients are ambulatory. All of these factors obviously account for the statistical variations.

There are probably more fractures of the upper than of the lower extremity. Fractures of the phalanges and ribs are probably most numerous. Following these without attempting to place them in order of frequency the common fractures are of the lower extremity of the radius, the region of the ankle, the metacarpals, the shafts of the bones of the leg, the skull, the clavicle and the humerus.

First Aid and Transportation. Except on hospital ambulances first aid after fractures is more likely to be rendered by laymen than physicians. In many instances this is the critical part of the whole treatment. It is necessary to know the principles of first aid for use while serving as ambulance surgeon, but it is of even greater importance to be able to guide and instruct the general public. "Splint em where they lie" is properly the first principle of fracture treatment.

the spine sometimes unrecognized than from any other injury. If the injured person complains of pain in his back it may be that his back is broken. If he complains of pain in his neck, he may have a broken neck. Do not lift an injured person or his head until he has told you whether he can move his legs or fingers. If he cannot move his legs, he has a broken back. If he cannot move his fingers, his neck may be broken. In either instance the spinal cord is injured. If the patient's body is bent while he is being carried in the former instance or if his head is lifted so as to give him a drink in the latter instance the injured spinal cord is inevitably ground between parts of the broken spine thereby destroying any useful remnant of the cord which may have escaped injury. Do not allow the injured person to sit up. If a fracture of the spine may be present and the victim is unconscious, handle him as though his neck were broken.

For the patient with possible fracture of the dorsal or of the lumbar region the underlying principle of hyperextension should be observed in moving and transferring him from the site of accident. He should be carried face downward on a rigid support. Wherever found, the patient should be slowly and evenly rolled over so that he rests on his abdomen on this rigid support. One of his forearms should be placed under his head, which is turned to the side as in the Schäfer resuscitation method. Whenever the patient must be moved at all before the rigid support is obtained, he may be rolled onto his abdomen on a sheet or blanket. This sheet may then be lifted by two persons, one at each end and the patient carried prone in this improvised hammock.

In case of possible fracture of the cervical vertebrae, gently slide the patient sidewise onto a rigid support so that he rests face upward with the arms at the side. Never tilt the head forward. No pillow should be placed under the head or neck. Pillows or sandbags may be placed each side of the head to prevent rotation.

Pelvis. The injured person should be transported on a rigid support lying on the back. If the injury seems severe, the thighs as well as the legs should be tied together. The position of the thighs and legs should be that naturally assumed. If enough assistance and the necessary material are at hand, a body swathe may be placed about the pelvic girdle. This should be applied snugly but not tightly.

Lower extremities. The only effective and

advisable method for transporting a patient with a fracture of the long bones of the lower extremity is by some form of traction fixation. This requires the use of a splint of the Thomas type so that pull is exerted from fixed points above and below the traction remaining the same whatever the position of the limb or splint. Overriding is characteristic of these fractures and there can be no generally effective immobilization without traction. In addition, traction relieves pain and shock and prevents further damage during transportation. The traction fixation splint should be applied if there is any possibility of fracture between the hip joint and the foot no matter whether the injuries are closed or open.

The full ring Thomas splint can be used. More generally applicable and advisable splints of the Thomas type are the Keller-Blake hinged half ring splints. These should be applied wherever the patient is found without changing his position.

There are certain standards that are necessary in the application of this general method. It does not make any difference what particular procedure is used so long as one appreciates what must be accomplished. In the use of traction there are six requirements. There must be some adequate form of hitch and it is necessary to protect the part beneath the hitch so that it will not be injured. The traction hitch should be applied above the ankle or wrist. There must be some means of increasing traction by twisting so that the desired pull is obtained. The extremity being in traction must be supported. One must not depend merely on traction for the entire support of the limb. Not only must the extremity be supported from below but lateral movement must be prevented. The whole splint must be suspended so that the heel will not, even for a moment, rest on the ground, the floor of the ambulance or elsewhere.

Take for example a patient with a possible fracture of the shaft of the femur. The patient is covered with blankets. Morphine is administered, if available, under medical supervision. The foot with or without the shoe on, is grasped firmly by an assistant with both hands placed laterally. Gentle, steady firm traction is made in the axis of the extremity. Manual traction is maintained until splint traction is established. While traction is being made, the whole limb is slowly lifted several inches from the ground. The half ring splint is slipped from the outer side beneath the extremity. The side bars of



Figure 1 Sling and swathe application for emergency care of a fracture of the humerus. A, Triangular sling laid against the body B Lower half of sling brought up in front of injured arm and tied behind neck. C, Broad swaths encircling body, injured arm and sling.

bone. If the bone disappears beneath the skin in the course of the application of traction, this should not cause anxiety. The patient must be operated on in any case to clean up the wound. It is important that word shall accompany the patient that the bone has been exposed, in order to guide the treatment by the next physician.

If materials are not available to splint the fracture properly the patient should not be lifted into the first automobile and rushed to a doctor's office or hospital. It is far better to cover the patient adequately and let him lie on the ground where he was found until he can be splinted and moved in an ambulance. If it is necessary to change his position at all before splinting, in the instance of an extremity fracture, pull should be exerted on the injured part while he is being moved.

Special Indications in Individual Fractures. *Lower jaw* Try to bring the pa-

tient's lower teeth against the upper teeth gently. Apply the center of a narrow bandage under the chin. Pass one end over the top of the head and cross the two ends above one ear, tying them tightly at the base of the skull, the crossing being on the side injured. Do not use a four tailed bandage.

Clavicle scapula humerus and elbow Use a sling to support the upper extremity with the elbow flexed and a body swathe to diminish movement at the shoulder (Fig. 1).

Forearm wrist and hand. Padded splints of wood or heavy cardboard will suffice for transportation.

Spine If the mechanism of injury is such that a fracture of the spine may have been produced, transport the patient as though a fracture had occurred, even though no objective signs are discovered in a cursory examination. Death or permanent disability results more commonly from improper transportation of the patient.

a fracture of

bones. From 10 to 30 cc. of a 2 per cent solution is sufficient. The skin at the site of the needle puncture must be prepared in the same way as for any operating room procedure. The needle is inserted at a point close to the fracture site; the plunger is withdrawn slightly until blood appears in the syringe; the Novocain is then injected. Relaxation is usually obtained within from two to five minutes. Unless the injection is into the hematoma as proved by blood in the syringe it is rarely successful. It is a simple procedure. No extra anesthetist is required. There is no struggle and consequently no danger of further displacement of the fragments either while the anesthesia is being induced or during recovery. Furthermore, the patient can cooperate in the application of splints for example by sitting up for the application of a shoulder spica. Twenty-four hours after a fracture is sustained, the blood is frequently coagulated; diffusion of the anesthetic does not occur and no relaxation results. During the first twenty-four hours the anesthetic is quite uniformly successful. The only other contraindication is the occasional damage to a large vessel or nerve by the needle. Its use is indicated in all simple fractures of an extremity. If more than one bone is broken injection will be required at each fracture site. The relaxation usually lasts from one-half to three-quarters of an hour and gives sufficient time for the physician to reduce the fracture, apply splints, take a roentgenogram and if the position is not satisfactory attempt reduction again without further injection of the anesthetic. Meantime the patient is awake and cooperative. Local anesthesia can rarely be used in children. It should not be used in patients with open fractures.

Two other forms of regional anesthesia are used, nerve block and field block. Nerve block, or plexus block, requires considerable skill and practice. Brachial plexus block is excellent in cases of fracture of the humerus. Field block does not demand special skill, but does require a considerable amount of anesthetic. Both of these forms may be employed for open fractures.

General anesthesia has been used in patients with fractures for a great many years. Ether is preferred for a long procedure. Nitrous oxide, cyclopropane or ethylene with oxygen, is suitable for short procedures. Ether gives more complete relaxation. All require the presence of a skilled anesthetist. When the fluoroscope is used in

connection with reduction there is too much danger of ignition of the ether vapor or of cyclopropane or ethylene to permit employment of such anesthetic agents. Gas-oxygen is less dangerous from this point of view, but its administration is difficult in a dark room.

Spinal anesthesia is excellent in cases of fractures of the lower extremities. From 50 to 100 mg. of Novocain injected beneath the dura in the fourth lumbar interspace will usually give complete relaxation for an hour.

Intravenous anesthetics are used considerably, particularly Pentothal sodium. It is best given in small doses repeated if necessary with skilled observation of the patient. Complete relaxation is liable to be accompanied by dangerous respiratory depression.

In the late treatment of fractures, an anesthetic is sometimes advised when it is thought necessary to manipulate joints and break up adhesions. This is a dangerous procedure and should never be resorted to until every other method of stretching has failed.

Open (Compound) Fractures. The basic principles in the management of a patient with an open fracture are as follows: treatment of shock and hemorrhage, prophylaxis and treatment of wound infection, accurate reduction and complete immobilization. In actual practice these three are associated at the same time and disaster may be the result of neglect of any one of them. If a tourniquet must be used, it should be applied as close to the wound proximally as possible since gangrene may result. Intermittent release of the tourniquet may result in profound shock.

Treatment of shock and hemorrhage. Shock and hemorrhage are frequently grave and attention must be devoted to them first.

Morphine is always indicated for pain except in head injuries but only in the amount necessary to control pain. In the instance of multiple injuries including the brain, it may still be given if accompanied by caffeine.

Body temperature must be maintained in some way but this must be done with judgment and the patient not kept bathed in perspiration, with loss of body fluids.

In most instances a pressure dressing is all that is necessary to control bleeding. The tourniquet has been grossly misused. An ineffective tourniquet has often caused serious loss of venous blood, which ceases as soon as the tourniquet is removed.

The major function of traction fixation as

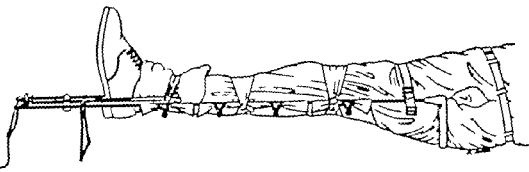


Figure 2 Lower extremity in a Keller-Blake hinged half ring splint with traction fixation applied in transportation.

the splint are placed in the median horizontal plane of the thigh so that the ring rests against the tuber ischii. The strap is buckled over the anterior surface of the thigh. The lower end of the splint rests against the body of the assistant who is still applying traction. The ankle region is protected by cotton wadding or the shoe. A traction hitch is applied—an army traction strap or muslin bandage about the ankle or adhesive plaster to the leg. The traction hitch is fastened to the end of the splint and traction is increased by a Spanish windlass action with a tongue depressor and adhesive plaster or a long nail. The manual traction is then released. The extremity is supported in the splint by means of muslin hammocks and clips applied before the splint is put on or by a wide cotton bandage wrapped from below upward about the extremity and traction bars after traction is applied. Lateral movement is prevented by turns of muslin bandage tied above and below the knee in the former instance (Fig. 2). The end of the splint is suspended to the stretcher bar or to the roof of the ambulance. A foot piece is advisable if transportation is to be prolonged.

In hospital practice a traction splint should be applied immediately in the emergency room, if it has not been done before. The patient is transported to the x-ray room and to the ward with traction on. The removal of the patient's clothes is supervised so that if traction must be loosened, it is maintained manually until it can be reapplied. The traction splint is kept on until the patient is in the operating room and under the anesthetic.

If a Thomas splint is not available for a fracture of the lower extremity, it must be appreciated that unless manual traction is exerted during transportation there is always danger of increasing the extent of the injury. Formerly long, side wooden splints were applied from the axilla to beyond the

foot for fractures of the hip and thigh, but these are difficult to apply and probably do more harm than good. It is preferable simply to bind the thighs and legs together using the uninjured leg as a splint.

For a fracture of the patella, leg or ankle a pillow which is drawn firmly around the part in its proper relation is fairly satisfactory. A padded posterior wooden splint will suffice for patellar fractures and two lateral wooden splints extending from midthigh to foot may be used for leg and ankle fractures. For all fractures of the foot, any splint of firm material which keeps the heel and toes from touching any object will suffice.

It is important to remember that the type of first aid and transportation is a large factor in the ultimate recovery. It has been stated that in World War I the mortality of gunshot wounds of the femur was reduced from 80 to 20 per cent by the routine use of the Thomas splint. Physicians need to be cooperative in urging the public to carry out these methods of transportation and not insisting, as do some, that a fracture should not be touched until the patient reaches the doctor's office or the hospital.

Anesthesia. Whenever any change in position or manipulation of fragments is indicated, an anesthetic should be used, this insures relaxation of muscles and allows the surgeon to be free to do whatever he finds necessary. Because one believes that reduction will be easy, this does not justify failure to use an anesthetic. Even if it is possible to obtain a perfect reduction without its aid, more damage to soft parts is liable to occur as a result of the resistance of unrelaxed muscles. The patient's statement that he can stand the pain is not of importance, as the anesthetic is not given for this reason, but to relax the muscles and allow as perfect replacement of fragments as possible.

Local anesthesia is the best, induced by an injection of Novocain directly into the hematoma around the ends of the fractured

bones. From 10 to 30 cc of a 2 per cent solution is sufficient. The skin at the site of the needle puncture must be prepared in the same way as for any operating room procedure. The needle is inserted at a point close to the fracture site; the plunger is withdrawn slightly until blood appears in the syringe; the Novocain is then injected. Relaxation is usually obtained within from two to five minutes. Unless the injection is into the hematoma, as proved by blood in the syringe, it is rarely successful. It is a simple procedure. No extra anesthetist is required. There is no struggle and consequently no danger of further displacement of the fragments either while the anesthesia is being induced or during recovery. Furthermore the patient can cooperate in the application of splints for example by sitting up for the application of a shoulder sprca. Twenty four hours after a fracture is sustained the blood is frequently coagulated; diffusion of the anesthetic does not occur and no relaxation results. During the first twenty-four hours, the anesthetic is quite uniformly successful. The only other contraindication is the occasional damage to a large vessel or nerve by the needle. Its use is indicated in all simple fractures of an extremity. If more than one bone is broken injection will be required at each fracture site. The relaxation usually lasts from one half to three-quarters of an hour and gives sufficient time for the physician to reduce the fracture, apply splints, take a roentgenogram and, if the position is not satisfactory, attempt reduction again without further injection of the anesthetic. Meantime the patient is awake and cooperative. Local anesthesia can rarely be used in children. It should not be used in patients with open fractures.

Two other forms of regional anesthesia are used, nerve block and field block. Nerve block, or plexus block, requires considerable skill and practice. Brachial plexus block is excellent in cases of fracture of the humerus. Field block does not demand special skill, but does require a considerable amount of anesthetic. Both of these forms may be employed for open fractures.

General anesthesia has been used in patients with fractures for a great many years. Ether is preferred for a long procedure. Nitrous oxide, cyclopropane or ethylene with oxygen, is suitable for short procedures. Ether gives more complete relaxation. All require the presence of a skilled anesthetist. When the fluoroscope is used in

connection with reduction there is too much danger of ignition of the ether vapor or of cyclopropane or ethylene to permit employment of such anesthetic agents. Gas-oxygen is less dangerous from this point of view, but its administration is difficult in a dark room.

Spinal anesthesia is excellent in cases of fractures of the lower extremities. From 50 to 100 mg. of Novocain injected beneath the dura in the fourth lumbar interspace will usually give complete relaxation for an hour.

Intravenous anesthetics are used considerably, particularly Pentothal sodium. It is best given in small doses, repeated if necessary, with skilled observation of the patient. Complete relaxation is liable to be accompanied by dangerous respiratory depression.

In the late treatment of fractures an anesthetic is sometimes advised when it is thought necessary to manipulate joints and break up adhesions. This is a dangerous procedure and should never be resorted to until every other method of stretching has failed.

Open (Compound) Fractures. The basic principles in the management of a patient with an open fracture are as follows: treatment of shock and hemorrhage, prophylaxis and treatment of wound infection, accurate reduction and complete immobilization. In actual practice these three are associated at the same time and disaster may be the result of neglect of any one of them. If a tourniquet must be used, it should be applied as close to the wound proximally as possible since gangrene may result. Intermittent release of the tourniquet may result in profound shock.

Treatment of shock and hemorrhage. Shock and hemorrhage are frequently grave and attention must be devoted to them first.

Morphine is always indicated for pain except in head injuries, but only in the amount necessary to control pain. In the instance of multiple injuries, including the brain, it may still be given if accompanied by caffeine.

Body temperature must be maintained in some way, but this must be done with judgment and the patient not kept bathed in perspiration, with loss of body fluids.

In most instances a pressure dressing is all that is necessary to control bleeding. The tourniquet has been grossly misused. An ineffective tourniquet has often caused serious loss of venous blood, which ceases as soon as the tourniquet is removed.

The major function of traction fixation as

a first aid measure before the patient is transported is to prevent or relieve shock and hemorrhage. Traction on a broken extremity relieves pain by putting muscles at rest and it diminishes extravasation by causing relaxed fascia again to exert pressure on tissues within it. Traction also limits further injury due to the churning about of fragments thereby preventing increased shock and increased hemorrhage.

Fluid replacement is necessary at the earliest possible moment. Loss of plasma from the blood not lowered systolic pressure is the early sign of shock. Whether whole blood or plasma expanders are required has now been fairly well standardized by accurate laboratory tests.

Multiple injuries are frequent in patients with open fractures. One must endeavor to determine immediately and without added trauma or exposure whether injuries to the brain, spine, chest, abdominal viscera and kidneys are present, are a probable source of shock and hemorrhage require postponement of any further treatment of the open fracture or require first consideration when the patient's condition will allow any further procedure.

Prophylaxis and treatment of wound infection. This should commence at the scene of accident and should be continued concurrently with the treatment of shock and hemorrhage.

The wound should be covered, if possible with a sterile dressing. The use of anti-septics and irrigations is contraindicated.

Traction fixation before moving the injured person from the site of accident is important in the prevention of infection. Whether the fracture is opened from within or from without, the tissues are contaminated. Not to put the muscles at rest, and therefore to allow the fragments to move about, increases the area of contamination.

The wounds of open fractures are of three types and with each of them traction fixation is to be employed.

In a type 1 wound, if the open fracture is one in which there is merely a slight wound and if there is no known soiled bone inside the wound, the traction splint will not only protect the underlying tissues but will keep the bone from coming out through the opening and getting dirty or spreading dirt around inside.

In a type 2 wound, if the open fracture is one which has been produced by a blow from some sharp object which penetrated the extremity and if the sharp object was

dirty causing contamination of the wound, the application of traction will keep the contaminating organisms from being spread around underneath the skin.

In a type 3 wound, if the fracture is such that the end of the bone comes out through the skin and becomes dirty a more difficult decision must be made. One fears the result of carrying soiled bone back into the soft parts as may happen under traction. The type of fracture is not common and is an injury extremely serious as regards both life and limb under any circumstances. The patient must be operated upon at the first possible moment. In order to transport the patient without danger of added trauma from the fragment of bone which remains inside the wound or without added contamination of the soft parts, the use of traction is proper.

At first, wounds are contaminated, not infected, and this contamination is in large part removable. With laboratory control of the requirements of the circulatory system, shock does not necessitate waiting beyond the optimum time before excision, shock or hemorrhage can be treated and the wound debrided all at the same time. Without chemotherapy we set an arbitrary limit of six hours from the time of accident within which excision may be done with expectation of success. But the danger of infection is greatly lessened if it can be done in one or two hours instead of six hours. In some instances, when the injured person does not reach the doctor until between six and twelve hours have passed, excision may still be tried, although with much less hope of success for contamination will usually no longer be limited to the surface. After twelve hours, excision is contraindicated. Operative intervention then must be limited to enlarging the wound, laying open deep pockets and removing gross foreign bodies. With the use of antibiotics from the time of injury to the time of operation, the period during which only contamination may exist and infection may not have set in has been found prolonged in many instances to seventy-two hours or more.

Unless the injured person is known to be immunized by toxoid, tetanus antitoxin should be given immediately to all in whom there is possible clothing or soil contamination. Repeating it is to be considered if there is any further operative intervention after the first week. If the injured person is known to have been immunized by toxoid more than three

0.5 cc. of tetanus toxoid should be injected if available. Otherwise tetanus antitoxin should be administered.

Gas gangrene serum is useless as a prophylactic. It should be used in therapeutic doses on the least suspicion of the presence of gas bacillus infection in the wound.

If it is believed that the excision has been well made, that circulatory disturbance or contamination has not been too severe and that the general condition of the patient is good, primary closure of the wound without a drain is the procedure of choice. This was not true until the antibiotic era. The antibiotics must never be substituted for adequate surgery. Closure may require relaxing incisions or primary split skin grafts. Partial closure with a drain is not good surgery in these patients. When conditions make it advisable that the wound be left open it is often proper to do a delayed closure after seven to ten days if the wound is then dressed for the first time and appears clean. This delayed closure does not include undercutting of the edges of the wound.

All pockets should be kept open. If their sides are allowed to fall together secretions may pocket off making ideal media for growth of bacteria. Two to four layers of dry gauze act as an effective drain. It must reach every part of the depth of the wound, preventing dead spaces. The wound must never be packed tightly and it should not be dressed frequently.

Without complete immobilization muscular action may change the site of contamination or infection at any moment. An unpadded plaster encasement with or without internal fixation or two-pin traction, comes nearest to furnishing complete immobilization of the soft parts in order to prevent or delay infection. This does not include a window in the plaster at the site of the wound, which destroys even pressure and allows edema and stasis of fluid, with increased danger of infection.

If emergency traction excision serums and drugs, drainage of the wound and immobilization have not been adequate or successful and infection is established the principles are the same as in the care of any soft part infection early and of osteomyelitis later with this difference—in spite of treatment of the infection, one must bend every effort to preserve reduction and immobilization of the fracture. The two problems cannot be separated.

In the prophylaxis and treatment of wound infection, whether early or late, the

virulence and extent of the contamination and the severity of the soft part damage must be considered as well as the elapsed time. More contamination will usually take place in a fracture which is opened from the outside than in one that is opened from the inside but both unquestionably demand excision. Major blood vessel damage may be such that the part distal to it cannot survive. The muscle damage may be such that it is evident immediately or at the time of excision that a useful limb cannot possibly be obtained. With these factors in mind primary amputation should sometimes be done to save life or prevent prolonged morbidity with a useless extremity as a final result.

Accurate reduction and complete immobilization. It is necessary to save the patient from death due to shock and hemorrhage and from death or loss of limb from infection. It is necessary also to aim for proper union of the fracture at the same time.

Application of traction fixation at the site of accident makes reduction simpler in the operating room. Traction should be continued under all circumstances until the patient is on the operating table and in some form even then.

Unless hemorrhage or the time factor makes it impossible x-ray examination should be carried out before excision.

In many instances, adequate excision will result in visualization of the line of fracture and perfect reduction can be obtained by sight rather than by palpation. Its accuracy is important as regards both infection and union. Reduction should not be delayed until infection is controlled.

Complete immobilization is necessary if excision is to be effective in preventing infection. If the fragments in an oblique fracture are slipping slightly each day the likelihood that infection will become established is increased. Unquestionably immediate rigid internal fixation of the fragments is the best answer. A dry gauze drain, without closure of the wound, can be used in the presence of metal. Internal fixation must be rigid. This can be accomplished by screws, plate and screws, or in highly selected cases the intramedullary nail.

There is always the possibility that infection may be spread through the medulla from one end of the bone to the other. Then the results of an intramedullary nail are tragic. Possibly in experienced hands in lightly contaminated, well-excised wounds, particularly where multiple injuries make the general management of the patient sim-

a first aid measure before the patient is transported is to prevent or relieve shock and hemorrhage. Traction on a broken extremity relieves pain by putting muscles at rest and it diminishes extravasation by causing relaxed fascia again to exert pressure on tissues within it. Traction also limits further injury due to the churning about of fragments, thereby preventing increased shock and increased hemorrhage.

Fluid replacement is necessary at the earliest possible moment. Loss of plasma from the blood, not lowered systolic pressure is the early sign of shock. Whether whole blood or plasma expanders are required has now been fairly well standardized by accurate laboratory tests.

Multiple injuries are frequent in patients with open fractures. One must endeavor to determine immediately and without added trauma or exposure whether injuries to the brain, spine, chest, abdominal viscera and kidneys are present, are a probable source of shock and hemorrhage, require postponement of any further treatment of the open fracture or require first consideration when the patient's condition will allow any further procedure.

Prophylaxis and treatment of wound infection. This should commence at the scene of accident and should be continued concurrently with the treatment of shock and hemorrhage.

The wound should be covered, if possible with a sterile dressing. The use of antiseptics and irrigations is contraindicated.

Traction fixation before moving the injured person from the site of accident is important in the prevention of infection. Whether the fracture is opened from within or from without, the tissues are contaminated. Not to put the muscles at rest, and therefore to allow the fragments to move about, increases the area of contamination.

The wounds of open fractures are of three types and with each of them traction fixation is to be employed.

In a type 1 wound, if the open fracture is one in which there is merely a slight wound and if there is no known soiled bone inside the wound, the traction splint will not only protect the underlying tissues but will keep the bone from coming out through the opening and getting dirty or spreading dirt around inside.

In a type 2 wound, if the open fracture is one which has been produced by a blow from some sharp object which penetrated the extremity and if the sharp object was

dirty, causing contamination of the wound, the application of traction will keep the contaminating organisms from being spread around underneath the skin.

In a type 3 wound, if the fracture is such that the end of the bone comes out through the skin and becomes dirty, a more difficult decision must be made. One fears the result of carving soiled bone back into the soft parts as may happen under traction. This type of fracture is not common and is an injury extremely serious as regards both life and limb under any circumstances. The patient must be operated upon at the first possible moment. In order to transport the patient without danger of added trauma from the fragment of bone which remains inside the wound or without added contamination of the soft parts, the use of traction is proper.

At first, wounds are contaminated, not infected, and this contamination is in large part removable. With laboratory control of the requirements of the circulatory system, shock does not necessitate waiting beyond the optimum time before excision, shock or hemorrhage can be treated and the wound debrided all at the same time. Without chemotherapy, we set an arbitrary limit of six hours from the time of accident within which excision may be done with expectation of success. But the danger of infection is greatly lessened if it can be done in one or two hours instead of six hours. In some instances, when the injured person does not reach the doctor until between six and twelve hours have passed, excision may still be tried, although with much less hope of success, for contamination will usually no longer be limited to the surface. After twelve hours excision is contraindicated. Operative intervention then must be limited to enlarging the wound, laying open deep pockets and removing gross foreign bodies. With the use of antibiotics from the time of injury to the time of operation, the period during which only contamination may exist and infection may not have set in has been found prolonged in many instances to seventy-two hours or more.

Unless the injured person is known to be immunized by toxoid, tetanus antitoxin should be given immediately to all in whom there is possible clothing or soil contamination. Repeating it is to be considered if there is any further operative intervention after the first week. If the injured person is known to have been immunized by toxoid more than three months previous to injury

Everyone knows that ecchymosis is one of the cardinal signs of fracture. But not every one realizes that its location, the time of its appearance and its extent may be valuable indications pointing to important pathologic occurrences and vital therapeutic needs for the best results.

LOCAL CHANCES FOLLOWING FRACTURE

The consensus today, based on both clinical and experimental evidence, supports the view that the repair process following fracture is a local phenomenon. The general state of the patient's health, his age, with the exception of infancy and childhood, the existence of acute or chronic general disease (syphilis, arteriosclerosis, nephritis, tuberculosis, arthritides, cardiac disease) and the existence of generalized metabolic disturbances (osteomalacia, rachitis, scurvy, diabetes) have no proved effect on the rate or degree of healing of a fracture. Of the various endocrine functions, not one has been shown to have any appreciable effect on the actual primary healing process, in the presence either of excessive or of diminished activity. It is true that individual researches may indicate to the worker some effect, but for each contribution of this type one may find, from apparently just as reliable a source, a contradictory conclusion. Despite suggestive reports from certain reliable authors of a correlation between the serum calcium and phosphate values and their interrelationship with fracture healing, extensive checks have failed to support this point of view. Moreover, a patient with a negative calcium balance and with marked generalized bone decalcification has, after sustaining a fracture, shown a normal or increased rate of fracture healing, later decalcifying the callus to the same degree as the rest of the skeleton. On the basis of clinical and experimental findings, major attention should be devoted to the local problems involved in the pathology and healing of fractures.

Consider what actually happens. At the time of injury the bone is broken and, coincidentally, there is a variable degree of damage to the surrounding soft parts by laceration or contusion.

The blood free in the tissues and the dead tissue resulting from the injury constitute irritants. In fact, even with a moderate pathologic condition, the patient may show within a few hours a rise in temperature, an increase in the leukocyte count and a raised sedimentation rate. There is, in effect, an

islet inflammation; the parts surrounding the fracture are rapidly infiltrated by hemorrage, edema and inflammatory exudate. Here then is the first clinically applicable knowledge of the pathologic picture. At the time of injury and for a variable period of from ten to thirty or forty minutes after injury the part may be said to be in a state of local shock. It is analgesic after the initial momentary sharp pain of the fracture, and its neuromuscular apparatus is practically paralyzed. A patient seen in this period will state that the arm or leg is "numb." It will be markedly flaccid and relaxed. It is in this stage that a bystander by merely pulling on the part can readily and accurately reduce a dislocation or even a badly displaced fracture with practically no pain to the patient. It happens practically every day on some field of sport or athletic activity and is accepted as a matter of course even by the layman. But only within the last few years has there been an appreciation of its application to the emergency use of traction through the application of Thomas type of arm and leg splints with windlass traction, which is now the approved standard of the American College of Surgeons as represented by its Fracture Committee. The use of immediate emergency traction on simple fractures if universally adopted, would constitute the biggest single advance in the treatment of fractures in the present generation.

Following the passing of the short period of local shock, sensation returns to the part and muscle spasm and pain follow. With muscle spasm there is the risk of increased deformity and additional interference with the vascular and lymphatic circulation. Within a few hours, hemorrhagic and inflammatory infiltration has pooled about the site of the fracture, forming a fusiform swelling and exerting lateral pressure on the surrounding soft tissues. The shortened and spastic muscles change from elastic, extensible structures to lardaceous semisolid masses which can be stretched only with great difficulty, even with damage. These muscles now constitute a mechanical bar to reduction. Had the fracture been reduced and held before this happened, the hardened and less distended tissues would act instead as splinting for the reduced fracture. This is the basis for the general principle that a fracture should be reduced and reduction retained as soon after injury as possible. Since this process is rapidly progressive and is complete within from six to eight hours

pler by its use, a real gamble may be taken by inserting an intramedullary nail. How ever its use in open fractures should be rare. It should never be used in children.

Any metal may be removed if infection sets in or when bony healing has progressed sufficiently. Although ideal, it is questionable whether this method should be used in patients with open fractures by surgeons inexperienced in the rigid internal fixation of closed fractures. Of course, external immobilization is required in addition at the completion of the operation.

Next in order of effectiveness of immobilization is the unpadded plaster encasement. It presents grave dangers and offers less security but is probably safest for the surgeon who treats only an occasional fracture patient.

Skeletal traction, with a distal pin only. Russell traction in patients with fracture of

the femur adhesive plaster traction padded plaster of paris encasement and splints of any material do not measure up completely to the requirements for immobilization. Conditions arise in which any one of them may have to be the method of choice in the individual patient. If there is a question of adequate circulation of the part, of the thoroughness of the excision or of the virulence of the contaminant a closed plaster encasement should not be used for several days at least, but some form of traction and suspension should be used instead which allows observation of the wound and the surrounding tissues.

READING REFERENCES

- An Outline of the Treatment of Fractures. Chicago, American College of Surgeons, 1954.
American Red Cross First Aid Textbook, 4th ed. Garden City N. Y., Doubleday and Co., 1947.

Pathology and Repair of Fractures

By PAUL C. COLONNA, M.D.

PAUL CRESHAW COLONNA, born in Norfolk, Virginia, educated in Virginia and Maryland, came north for his training in orthopedic surgery. He spent five years as Professor of Orthopedic Surgery at the University of Oklahoma and then went to Philadelphia where he was the Professor of Orthopedic Surgery at the University of Pennsylvania School of Medicine for sixteen years. An interest in anthropology has correlated well with his chosen surgical specialty.

The intelligent care of fractures demands, as a prerequisite, a general knowledge of the pathologic condition accompanying the injury, the factors involved in the process of repair and the correlation of these with the disturbed physiology which one hopes to restore to normal. Without such knowledge a fracture bids fair to remain an x-ray picture throughout its course. Such a point of view can never lead to really adequate treatment. An understanding of pathology and repair, the biochemical changes and minute cellu-

lar activities brought into play are matters of great academic interest. These processes are reflected in the signs, symptoms and disturbed physiology which indicate the basic principles of treatment. A fracture must not be regarded only as a solution of bony continuity but equally and sometimes more important is the accompanying trauma to the surrounding soft parts with minor or serious injury to the vital structures such as the skin covering, circulation or nerve tissues.

becomes established as adult fibrous connective tissue then no bone formation occurs. If this is so, and if our ideas on the deposition of calcium as a result of phosphatase activity within a limited pH range approaching the alkaline side are correct it would seem that if the acid pH phase is prolonged by reason of the amount of tissue death or by reason of a circulatory status which makes impossible the carrying away of the products of tissue death the healing tissue may differentiate before any calcium deposition can be effected with the result that the healing remains fibrous. The deposition of calcium in the lower pH ranges may be so slow that so-called delayed union occurs.

In this understanding of the process lie possible explanations for many of the apparent vagaries connected with fracture healing. It is to be kept in mind that, when local destruction of intrinsic bone circulation has occurred as a result of fracture then the most important factor in the maintenance of an efficient repair level is the available accessory circulation of the soft parts—blood vascular and lymphatic. On this factor hinges to a large extent how much growth of healing granulation tissue can occur and how soon deposition of calcium in this tissue can take place.

The study of these phenomena has led to an appreciation of the fact that attention to the circulatory efficiency of the part, particularly in its more minute phases rather than in the major vessels, is a big factor in promoting normal, or rather average, healing. This has a definite bearing on the present day tendency toward pin fixation and open operations, thereby permitting early active mobilization of the part early intensive physical therapy and elevation, since those three adjuvants, in that order are the most effective means of improving the local circulatory status.

The pathologic condition of the fracture line is a matter of considerable importance in an evaluation of the suitability of various methods of treatment in a given case.

GUIDES FOR FRACTURE TREATMENT

Some guides for fracture treatment based on the principles of wound healing are as follows:

1. *First aid* in fracture cases should aim at as extensive and complete immobilization of the part as can be achieved and should include fixed traction, if possible applied as soon after injury and with as little trauma as feasible.

2. *Shock and injury* should be treated coincidentally. Except when the patient is moribund the treatment of shock should be carried out simultaneously while the fracture is being given early definite treatment.

3. *Reduction* should be accomplished before the muscles and soft parts have lost their elasticity by reason of infiltration of exudate and hemorrhage. This is just as true for traction suspension open reduction and fixation, pin fixation or any other method as it is for manual manipulation and plaster fixation. It means that reduction by open or closed methods should be complete within eight to twelve hours or sooner if possible.

4. That method of treatment which allows the most *active function* of the extremity during bone healing is if it can be adequately carried out, the best method from the dual standpoint of successful bone healing and of short convalescence time. Whether or not the theoretically best method is practically applicable in the individual case depends upon the analysis of the patient and his lesion, upon the qualifications of the doctor and upon the organization and equipment available.

5. *Physical therapy* should be regarded as an adjunct to, or an inadequate substitute for active function and both patient and doctor should thoroughly understand this.

6. *Postreduction immobilization* should aim at as rigid fixation of the fragments as possible.

7. Fractures heal primarily by the *formation of callus* about the fracture site and circulatory restoration in the soft parts is a vital factor in the process.

8. In *compound fractures* the treatment of the wound along with adequate reduction and fixation of the fracture should be accomplished just as soon, in terms of minutes after injury as it can be done. Early wound treatment with delay in reduction and fixation is a mistake.

The pathologic condition of the soft tissues has a definite bearing on the methods of reduction and should constitute the basis of any intelligent effort at reduction.

Lastly it is essential to recognize that, for each region of the body important soft part structures are liable to damage. Examination of the patient includes elimination of the possibility of injury to them. The soft-part lesion, if present, may constitute an urgent reason for altering the method of treatment ordinarily used for the fracture in question.

For instance, in a lesion involving the middle third of the humerus, the radial nerve is

it becomes obvious that one should think of reduction in terms of minutes after injury not in terms of hours or days. An hour spent in waiting around an accident ward may mean the difference between ease and difficulty of reduction. This is of major importance in any fracture of the shaft of a long bone but of less practical significance in fractures such as Colles fracture at the wrist and uncomplicated malleolar fracture at the ankle, where muscular resistance is not so potent a factor. It is, however, a matter of considerable import in patients who require skin or skeletal traction. When a patient has waited overnight to have pin or wire or adhesive traction applied, what might have been an easily accomplished reduction by the stretching of elastic and extensible muscle bellies with a minimum of trauma may turn out to be an indifferent reduction accomplished with difficulty through the necessity of dealing with infiltrated, inelastic muscle masses. The blame for failure is placed on the shortcomings of traction and suspension instead of on ignorance of or lack of consideration for the pathologic change going on during the overnight wait. If emergency fixed traction had been applied in the interim, much of the difficulty would have been obviated. When a wait is necessary this type of traction is the best means of minimizing the potential damage due to delay.

Following this pathologic stage there is clotting, i.e., laying down of a fibrin network in the hemorrhage and exudate in the tissues. This network joins the bone ends and adjacent soft parts in a web of interlacing fibrils along which tissue repair is to take place. The remaining fluid portions of blood and exudate seep to the surface slowly along the tissue planes if the soft parts about the fracture are but little lacerated, or appear rapidly beneath the surface if there has been extensive laceration of the soft parts. The ensuing discoloration of skin due to the presence of the blood pigment in the fluid portion of the blood is known as ecchymosis. It is obvious that an early rapid appearance of ecchymosis at the fracture site indicates a large amount of tissue damage being proportionately greater the deeper the involved bone lies from the skin. *Ipso facto* it indicates the need for methods of treatment which will allow maximum early attention to the pathologic condition of the soft parts.

On the other hand, ecchymosis which appears late, particularly at a point distant from the fracture, indicates a minimal

amount of soft part damage with a more favorable prognosis for the early return of function. If the elbow joint is involved, for example as in fractures of the radial head or lower humerus and in dislocations very early ecchymosis on the front of the elbow sometimes within an hour from the time of injury indicates extensive laceration of the joint capsule and brachialis anticus and a threat of ossification about the front of the elbow. Special treatment is urgently needed under such circumstances.

As a result of the death of tissue, hemorrhage and progressive circulatory stagnation of lymph and blood following rapidly after fracture the *pH* of the local tissue fluid becomes acid. With this acidity there occurs over the next week or ten days a decalcification of the fracture site.

Coincident with this chemical change the healing process proceeds, as with any other wound, by the growth of new fibroblastic cells along the fibrin network, from all available connective tissue sources—the marrow cavity with its so-called endosteum, the periosteum and the fascial planes and muscular stroma, if they are opened into by the fracture.

This healing process is what one would expect in any wound. At some stage during the process calcium is deposited in the healing tissue to surround the cells. The tissue then is called *callus* instead of granulation tissue. Whether or not osteoid tissue (early callus) contains calcium or merely a proteinaceous substance in which calcium is deposited whether or not specific bone-forming cells are involved in the process, either by direct growth and migration of "osteoblasts" from adjacent bone and periosteum and endosteum or by "metaplasia" of connective tissue cells in reply to the "physiologic demand," and whether or not ferment activity (phosphatase) is an integral part of the story are at present matters of academic interest. The practical clinical application of this phase of the story lies in the fact that the calcium deposited in the newly formed connective tissue is for the major part derived from local decalcification at the fracture site and that its deposition occurs coincidentally with the alteration of the *pH* of the tissue fluids locally toward the alkaline side.

Moreover there is a great deal of evidence to support the viewpoint that this deposition of calcium into the healing tissue to form bone can occur only while the tissue is still undifferentiated. Once the healing tissue

becomes established as adult fibrous connective tissue then no bone formation occurs. If this is so, and if our ideas on the deposition of calcium as a result of phosphatase activity within a limited pH range approaching the alkaline side are correct it would seem that if the acid pH phase is prolonged by reason of the amount of tissue death or by reason of a circulatory status which makes impossible the carrying away of the products of tissue death, the healing tissue may differentiate before any calcium deposition can be effected with the result that the healing remains fibrous. The deposition of calcium in the lower pH ranges may be so slow that so-called delayed union occurs.

In this understanding of the process the possible explanations for many of the apparent vagaries connected with fracture healing. It is to be kept in mind that when local destruction of intrinsic bone circulation has occurred as a result of fracture, then the most important factor in the maintenance of an efficient repair level is the available accessory circulation of the soft parts—blood, vascular and lymphatic. On this factor hinges to a large extent how much growth of healing granulation tissue can occur and how soon deposition of calcium in this tissue can take place.

The study of these phenomena has led to an appreciation of the fact that attention to the circulatory efficiency of the part particularly in its more minute phases rather than in the major vessels, is a big factor in promoting normal or rather average healing. This has a definite bearing on the present-day tendency toward pin fixation and open operations, thereby permitting early active mobilization of the part, early intensive physical therapy and elevation since those three adjvants in that order are the most effective means of improving the local circulatory status.

The pathologic condition of the fracture line is a matter of considerable importance in an evaluation of the suitability of various methods of treatment in a given case.

GUIDES FOR FRACTURE TREATMENT

Some guides for fracture treatment based on the principles of wound healing are as follows:

1. *First aid* in fracture cases should aim at as extensive and complete immobilization of the part as can be achieved and should include fixed traction, if possible, applied as soon after injury and with as little trauma as feasible.

2. *Shock and injury* should be treated coincidentally. Except when the patient is moribund, the treatment of shock should be carried out simultaneously while the fracture is being given early definite treatment.

3. *Reduction* should be accomplished before the muscles and soft parts have lost their elasticity by reason of infiltration of exudate and hemorrhage. This is just as true for traction suspension, open reduction and fixation, pin fixation or any other method as it is for manual manipulation and plaster fixation. It means that reduction by open or closed methods should be complete within eight to twelve hours, or sooner if possible.

4. That method of treatment which allows the most active function of the extremity during bone healing is if it can be adequately carried out the best method from the dual standpoint of successful bone healing and of short convalescence time. Whether or not the theoretically best method is practically applicable in the individual case depends upon the analysis of the patient and his lesion upon the qualifications of the doctor and upon the organization and equipment available.

5. *Physical therapy* should be regarded as an adjunct or an inadequate substitute for active function and both patient and doctor should thoroughly understand this.

6. *Postreduction immobilization* should aim at as rigid fixation of the fragments as possible.

7. Fractures heal primarily by the formation of callus about the fracture site and circulatory restoration in the soft parts is a vital factor in the process.

8. In *compound fractures* the treatment of the wound along with adequate reduction and fixation of the fracture should be accomplished just as soon in terms of minutes after injury as it can be done. Early wound treatment with delay in reduction and fixation is a mistake.

The pathologic condition of the soft tissues has a definite bearing on the methods of reduction and should constitute the basis of any intelligent effort at reduction.

Lastly, it is essential to recognize that for each region of the body important soft part structures are liable to damage. Examination of the patient includes elimination of the possibility of injury to them. The soft part lesion if present may constitute an urgent reason for altering the method of treatment ordinarily used for the fracture in question.

For instance, in a lesion involving the middle third of the humerus, the radial nerve is

frequently injured. If this is not noted at the time of the original injury it may not be discovered until six or eight weeks later when immobilization is ended. Similarly the median nerve at the wrist may be involved in badly displaced Colles fractures and in lunate dislocations. The peroneal nerve is often involved in lesions about the fibular neck or by direct violence in this region, though the fracture is of the tibia. Lesions about the internal humeral condyle may involve the ulnar nerve and those about the external condyle may involve radial or superficial radial nerves. Dislocations of the shoulder or surgical neck fractures frequently involve the axillary nerve. Supracondylar fractures of the humerus with displacement are the commonest cause of Volkmann's ischemic contracture. The local tension about the front of the elbow and the state of the radial pulse before reduction, after reduction and in the immobilized position must be carefully noted. One frequently sees patients in whom prompt and adequate provision for the correction of the circulatory embarrassment has aborted a Volkmann's contracture the very early signs of which were already present when the patient was first seen. Unfortunately in many subjects, a check on the radial pulse is not considered as part of the examination in handling a lesion of the elbow. Knowledge of this complication of supracondylar fractures requires immediate concern for the radial pulse.

There are numerous examples of impending disaster lurking in lesions of the soft parts complicating a fracture. Each region of the body has its soft part trap, so to speak, and it behooves the surgeon to know these so they can always be looked for anticipated and, if possible, avoided.

If the laceration extends through the skin, allowing communication between the fracture and the outside air the fracture assumes a specific significance as a compound fracture.

DELAYED UNION AND NONUNION OF FRACTURES

On the basis of experimental procedures, clinical observation and roentgen ray studies the present-day conception of the problem of delayed union and nonunion may be epitomized in the following brief statements.

1 All fractures heal as do wounds elsewhere unless there is a mechanical chemical anatomic bar to healing. This healing, in common with that of wounds of the soft parts, takes place through the medium of

new connective tissue known as *granulation tissue*. When referring to delayed union and nonunion, one in reality refers to a delay or failure in the process of calcium deposition in the healing connective tissue, which is commonly designated as new bone formation. The anatomic bar to healing lies in the absence of a sufficient accessory circulation to compensate for that destroyed by trauma and to remove adequately and rapidly the products of tissue death. Some regions of the body even under apparently optimum conditions are characterized by slow average healing time and by the frequent occurrence of delayed union and nonunion in adults. The neck of the femur (subcapital or midcervical) the junction of the lower and middle thirds of the tibia, the scaphoid of the carpus (proximal half) and the base of the fifth metatarsal are characteristic regions.

In patients who have fractures in these regions and simultaneous fractures in regions not so characterized, there is no perversion of healing in the latter all other factors except location being equal.

2 Variations in the character and amount of the healing process in bone following a fracture are dependent on factors limited to the region of the body involved, quite independent of the subject involved. Slow union and nonunion in the adult are not dependent on the age per se of the patient, on his general state of health, on the presence of a chronic general disease such as syphilitic or cardiovascular or renal disease, on general wasting due to other causes, on general metabolic disturbances affecting either the general calcium and phosphorus metabolism (osteomalacia) or other phases of metabolism (diabetes) or on acute infectious diseases. Nonunion, except for mechanical reasons, is practically never seen in children.

3 The four factors involved in the mechanism of the healing of fractures and capable of being clinically influenced are the local pathologic condition the growth of granulation tissue, an available local source of calcium for the ossification of the healing tissue and a proper biochemical status of the local tissue fluids throughout the healing process.

Local Pathologic Condition. The items of importance under this factor are (a) The amount of tissue necrosis. When massive, it destroys the available sources for the growth of granulation tissue and under all circumstances creates a pit to the acid side in the hematoma and tissue fir the

the fracture. This pit is of importance in the production of the local source of calcium and is the essential early stage of the biochemistry of the tissue fluid. An acid pit interferes with calcium deposition.

b. The growth of granulation tissue is influenced by the hematoma or blood clot soon at the site of the fracture: first by the fibrin and fluid content locally and second by the establishment of a local source of calcium, holding the freed calcium in situ.

c. The significance of the circulatory status in the different stages of fracture healing lies in its influence on the growth of granulation tissue and on the establishment and utilization of a local source of calcium through its effect on the pit of the tissue fluids.

d. Chemical influences which have an effect on the growth of granulation tissue are the local use of chemotherapeutic agents or disinfectants. The use of radiation diathermy and too early mobilization are physical influences.

e. The mechanical element is the impenetrability of living or dead material impenetrable to the growth of granulation tissue between the fragments.

Growth of Granulation Tissue. The items of importance under this factor are as follows:

a. All other things being equal the amount of callus produced is directly proportional to the amount of granulation tissue produced.

b. The destruction of available sources for the growth of granulation tissue.

c. The anatomic characteristics of the part regarding sources for the growth of granulation tissue and accessory circulation.

d. The amount of fluid persisting for a long period at the site of the fracture.

e. Chemical and physical influences and the mechanical element which limits the growth of granulation tissue between the fragments.

Available Local Source of Calcium. The items of importance are the amount of bone necrosis, the density and degree of fragmentation of the necrotic bone, the influence of fibrin and collagen in fixing the calcium locally through chemical affinity and the biochemical status of the local tissue fluids.

Proper Biochemical Status of the Local Tissue Fluids throughout the Healing Process. This factor involves the necessity in the local tissue fluid of a pit on the acid side in the early days following the fracture in connection with the establishment of a local

source of available calcium and a reversion of the pit toward the alkaline side before the calcium made available can be to any extent deposited in the new tissue to form bone. The constituents normally involved in this phase of the process are the amount of tissue necrosis and the blood and lymph circulatory status of the part.

Prevention of Delayed Union and Nonunion. What then can be done to prevent delayed union and nonunion? The objects of concern are:

1. Early and accurate replacement of displaced fragments with assurance at the time of reduction that there is no interposition of tissue.

2. The restoration to normal of the lymph and vascular circulation as rapidly as possible through elevation of the part, early physiotherapy and the use of functional muscular activity whenever possible.

3. The meticulous care of wounds in cases of compound fracture. Gentleness and conservation of sound tissue are not incompatible with thorough and efficient debridement. It pays to be conservative on the score of considering a debrided wound safe to sew up tightly. Chemical disinfection of a wound is not desirable. No method of wound treatment which creates tension in the tissues can be considered sound.

Where facilities for operative work on bone are of high standard as regards equipment, technique and personnel, many feel that in an open fracture rigid fixation of the fragments by plate or screw at the time of the debridement with the wound left open is justified through minimizing the chances for delayed union or nonunion by eliminating the foothold for infection engendered by constant movement of the fragments. Such a procedure is never justified unless the facilities for carrying it out are as specified.

4. Where the facilities for operative work on bone are of the highest standard as regards equipment, technique and personnel, it is today the belief of many that operative reduction with internal fixation sufficiently rigid to permit postoperative active mobilization in balanced suspension, is the ideal method for fractures of the long bones in adults. It is certainly very doubtful whether operative reduction and internal fixation are justifiable except on the basis of absolute necessity unless rigid fixation cannot otherwise be secured. The early active use of the musculature as an aid to the minute circulation is one of the chief recom-

mentations for the use of the method. Any necessity for postoperative immobilization nullifies this important advantage. Moreover it is felt that if open reduction is indicated, it should be done within the first few hours after injury in order that the blood, dead tissue and inflammatory exudate can escape through the operative wound or can be removed. This minimizes the burden placed on the local circulation in removing products of tissue death in order that the pit of the tissue fluids can revert toward normal as rapidly as possible thereby facilitating the early deposition of calcium in the undifferentiated healing tissue.

When the fracture lies in a region characterized by slow or indifferent healing, the rigid internal fixation can be augmented by the use of osteoperiosteal or other forms of grafts as prophylaxis against nonunion.

It is to be strongly stressed that the use of operative reduction as a method of choice can be conscientiously adopted only when exacting conditions as to organization equipment, personnel and technique can be met and when the mechanical principles involved in securing rigid fixation capable of standing functional strain are thoroughly understood.

TREATMENT

There are many reasons to believe that a bone graft does not grow but acts as a scaffolding and as a source of calcium for the calcification of the healing granulation tissue and is one of the sources for the enzymes concerned in the calcium deposition. The removal of fibrous tissue and sclerotic bone aids circulatory increase and granulation tissue growth and invasion. The graft rarely presents sufficient fixation to permit early active movement, especially since osteoperiosteal or cancellous grafts offer the most effective osteogenetic qualities—a fact which has been demonstrated both clinically and experimentally. If possible, there should be additional rigid fixation, obtainable from plate screw or other rigid media when feasible such as intra medullary nailing or massive bone graft, for this permits earlier activity. The postoperative treatment should follow the same prin-

ciples as those laid down for the prevention of delayed union and nonunion.

In addition to the prophylactic measures which may be employed against delayed union and nonunion, the following rather conservative measures are available

1 Needling of the fracture site This consists of passing a sharp heavy needle repeatedly about and through the fracture site producing a certain amount of tissue stimulation and hemorrhage about and between the bone ends and setting up a variable degree of inflammatory reaction. According to one school of thought, this may stimulate osteoblasts to produce bone according to another it recreates the original pathologic conditions of the fracture and is followed by ossification in accordance with the conditions which obtain in fracture repair. At any rate, this procedure is followed by union in a certain number of cases. There are no positive criteria whereby one may gauge what fractures are apt to respond to this treatment.

2 The injection of whole blood between and about the bone ends The same objection applies here as in the case of needling without injection. In addition to whole blood, various other mildly irritating substances have been employed with similar results. The use of calcium emulsions for injection has been attempted, but has not given very promising results. In instances of late nonunion, this method is not applicable. Although the successful use of this procedure has been reported, it does not seem on the whole to be any more valuable than plain needling.

3 The use of Bier's hyperemia intermittently over a considerable time The effect here is again on a circulatory stimulation basis. This method is not in general use today for many practical reasons, yet it is physiologically sound in producing an increase in the blood supply locally.

4 Cotton's method of tapping the greater trochanter with a mallet. This technique has been employed in stimulating a delayed union in fracture of the neck of the femur.

5 Mommensen's method of subminimal stimulation This is of particular use in stimulating and hastening delayed union of the

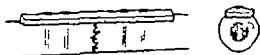


Figure 3. Massive onlay graft.

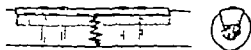


Figure 4. Inlay graft.

tibia. In this method a suspended mallet is attached to a brace which is arranged to strike the heel of the foot by the patient controlling the mechanism by hand.

6. The initiation of cautious weight bearing in patients with fracture of the lower extremity either with a so-called Bohler walking iron or walking plaster.

7. The drilling or boring of numerous holes in the bone cortex after an open exposure of the fracture site. This creates channels through the sclerotic bone for revascularization of the fragments and may be very helpful.

Perhaps the most successful of these methods is based on the following three principles: removal of the avascular scar tissue between the separated bone ends, the bringing of healthy bone into close contact, locally bringing to the part additional bone elements which may act as a scaffolding along which healthy granulation can invade itself.

In addition to these more or less conservative methods of stimulating bone healing in delayed and nonunion of fractures, one must consider the great value which the different types of bone grafting offer. One may select the homogenous bone graft when there is an area or a large cavity to be filled in, as this type of graft allows using the bone bank. This method has its vigorous adherents because of its availability and because of the ease of storing large quantities of bone. But it has its great limitations, one of these being that, statistically, bone union can be more successfully obtained with the autogenous type of graft than with the homogenous. However the use of homogenous bone provides a valuable method of bone grafting and should be available in all reconstructive work on the skeletal system.

The autogenous graft is available within limited amounts but has been used most successfully by a great number of surgeons for many years. The tibial graft introduced by Albee many years ago opens up a wide field for the application of bone grafting. Cortical bone is available from any of the long bones that are superficially placed, espe-

cially the rib and fibula. The osteogenetic power of the rib and the tibia makes them favorite sites of election but any and all of these cortical bone grafting methods can be improved by additional use of cancellous bone packed in and about the site of the delayed union or ununited fracture. The crest of the ilium is a favorite site for obtaining cancellous bone.

All of the grafting methods should follow the general rules of fracture treatment, that is, fresh bone surfaces apposed and rigid immobilization for a sufficient time to allow bone union.

The massive onlay graft (Fig. 3) has much to recommend it. It was primarily popularized by Thomister and Henderson.

The inlay type graft (Fig. 1) represents a common method of treatment.

The osteoperiosteal type of graft does not give the rigid immobilization from the graft but brings many of the osteogenetic elements in close contact at the site of delayed union or nonunion.

The sliding type of graft is often utilized because of the ease of application and because it does not require a separate incision. It does not, however, produce statistically as high a percentage of bone union (Fig. 5).

Another method is the dual graft popularized by Lloyd (Fig. 6). This consists of two grafts placed on either side of the fracture site and held by through-and-through screw fixation giving rigidity. Frequently it is augmented by cancellous bone packed in and about the site of fracture. It has the particular advantage of bringing a great mass of bone, both cortical and cancellous to the site and has produced a most satisfactory percentage of successes, particularly when used with many difficult types of nonunion such as in a fractured bone of a congenital pseudarthrosis patient.

The intramedullary type (Fig. 7) is not as popular today as formerly and should be used only as a last resort. The medullary cavity is blocked by inserting a large graft into the marrow cavity.



Figure 5. Sliding graft.

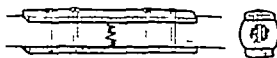


Figure 6. Dual graft.

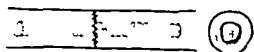


Figure 7. Intramedullary graft.

READING REFERENCES

- Albee, F. H. *Bone Graft Surgery* Philadelphia, W B Saunders Company 1915
- Blount, W P. *Fractures in Children*. Baltimore, Williams & Wilkins Company 1935.
- Bonfiglio, M. Repair of Bone-Transplant Fractures. *J Bone & Joint Surg.* 40A:446, 1958
- Boyd, H B. The Problem of Difficult and Unusual Non-unions. *J Bone & Joint Surg.* 25:535 1943.
- Copp D H and Greenberg, D M. Studies on Bone Fracture Healing, Effect of Vitamins A and D *J Nutrition* 29:261 1945
- Croftin, A. Les formations cartilagineuses dans le cal des fractures experimentales. *Presse med.* 54:74 1946
- Dragstedt, C. A., and Kearns, J E., Jr. Experimental Study of Bone Repair: Effect of Thyroparathyroidectomy and of Administration of Parathormone. *Arch. Surg.* 24:963, 1932
- Fitts, W T. Jr. Healing of Fractures. *S Clin. North America* 26:1470 1946.
- Friedenberg, Z. B.: Recent Advances in Bone Physiology. *Internat. Abstr. Surg.* 98:313 1954
- Hass, S. L. Stimulation of Bone Growth. *Am J Surg* 65:12, 1958.
- Haldeman, K. O. Role of Periosteum in Healing of Fractures, Experimental Study. *Arch. Surg.* 24:440, 1932.
- Ham, A. W. and Harris, W R. Repair and Transplantation of Bone. In Bourne, G H. *Biochemistry and Physiology of Bone*. New York, Academic Press, 1956.
- Hellstrand, A. Study of New Bone Formation Provoked by Subperiosteal Injections of Blood Plasma, Extract of Bone Marrow etc. Investigation by Experiments on Animals. *Acta chir. scandinav.* 95:31 1947
- Henderson, M. S. The Massive Bone Graft in United Fractures. *J.A.M.A.* 107:1104, 1938
- Hernndon, C. H., and Chase, S. W. Fate of Massive Autogenous and Homogenous Bone Grafts, Including Articular Surfaces. *Surg. Gynec. & Obst.* 98:275, 1954
- Jaffee, H. L. Structure of Bone, with Particular Reference to Its Fibrillar Nature and Relation of Function to Internal Architecture. *Arch. Surg.* 19:24, 1929
- Jay H. D.: Plasma Phosphatase. II. The Enzyme in Disease, Particularly in Bone Disease. *J Biol. Chem.* 89:249 1930.
- Jay H. D.: Phosphatase in Growth and Disease of Bone. *Physiol. Rev.* 12:384, 1932.
- Leriche, R., and Polkard, A. De quelques notions physiologiques utiles à connaître pour la compréhension de la pathologie osseuse. *Presse med.* 30:1222, 1923.
- Marshall, A., and Byron, R. L., Jr. Method for Study of Healing of Bone. *J Bone & Joint Surg.* 27:95 1945.
- McKeown, R. M., Lindsay M. K. Harvey S C., and Howes, E. L.: Breaking Strength of Healing Fractured Fibulae of Rats, Observations on Standard Diet. *Arch. Surg.* 24:458, 1933.
- McLean, F. C., and Urist, M. R.: *Bone*. Chicago, University of Chicago Press, 1955.
- Murray C. R.: Repair of Fractures. *Minnesota Med.* 13:157 1930
- Murray C. R. Delayed and Non-union in Fractures in Adult. *Ann. Surg.* 63:961 1931
- Murray C. R. Proper Place of Physical Therapy in Treatment of Fractures. *J.A.M.A.* 97:235, 1931
- Murray C. R. The Modern Conception of Bone Formation and Its Relation to Surgery. *J Dent. Res.* 11:837 1931
- Murray C. R.: Exact Role of Physical Therapy in Treatment of Fractures. *Surg. Gynec. & Obst.* 56:479 1933.
- Murray C. R. Healing of Fractures; Its Influence on Choice of Methods of Treatment. *Arch. Surg.* 29:446 1934
- Murray C. R. Timing of Fracture-Healing Process; Its Influence on Choice and Application of Treatment Methods. *J Bone & Joint Surg.* 23:598, 1941.
- Murray C. R. Surgical Principles Opposed to "Rule of Thumb" in Treatment of Compound Fractures. *Ann. Surg.* 118:305 1943.
- Murray C. R., and Murray M. R. The Mechanism of Deposition of Calcium to Form Extracortical Bone in the Adult Animal—A Preliminary Study. *Anat. Rec.* 47:222, 1930.
- Phemister D B. The Fate of Transplanted Bone and Regenerative Power of Its Various Constituents. *Surg. Gynec. & Obst.* 19:303, 1914
- Phemister D B. Treatment of Un-united Fractures by Onlay Bone Grafts without Screws or Tie Fixation. *J Bone & Joint Surg.* 29:946, 1917
- Phemister D B. Recognition of Dead Bone Based on Pathological and X-ray Studies. *Ann. Surg.* 72:466, 1920
- Phemister D B. Repair of Bone in Presence of Aseptic Necrosis Resulting from Fractures, Transplantations and Vascular Obstruction. *J Bone & Joint Surg.* 12:769 1930.
- Phemister D B. Aseptische Knochennekrose bei Frakturen, Transplantationen und Gefässverhinderungen. *Zschr. f. orthop. Chir.* 53:181 1931
- Ravdin, I. S., and Morrison, M. E. Ossification After Fracture, Experimental Study. *Arch. Surg.* 17:813, 1928.
- Schram, W R., and Foadick, L. S. Studies in Bone Healing. *J Oral Surg.* 1:191 1943.
- Sieard, A., and Moaly R. Frozen Homogenous Bone Grafts. *Presse med.* 62:863, 1954.
- Speed, J S., and Knight, R. A. *Campbell's Operative Orthopaedics*, 3rd ed. St. Louis, C. V Mosby 1956
- Spemann, H. Embryonic Development and Induction. New Haven, Yale University Press, 1938.
- Swenson, O. Biochemical Changes in Fracture Hematomas. *J Bone & Joint Surg.* 29:288 293 1946.
- Swenson, O. and Claff C. L. Changes in Hydrogen Ion Concentration of Healing Fractures. *Proc. Soc. Exper. Biol. & Med.* 61:181, 1946.

Fractures and Dislocations

Fractures and Dislocations of the Upper Extremity and Spine

By SAM W. BANKS, M.D.

SAM WALLACE BANKS, of North Carolina by birth, was educated at the University of Richmond and the University of Chicago. Trained in anatomy, his early interest in investigation centered about the bacteriology and immunology of chronic staphylococcal otitis media. He has made many contributions to the literature of orthopedic surgery and is Associate Professor of Orthopedic Surgery at Northwestern University.

THE HAND

Fractures of the Distal Phalanx. These fractures usually are the result of direct crushing violence and are associated with contusion of the soft tissues and hemorrhage beneath the nail. They are frequently of the comminuted variety. There is ordinarily little displacement of fracture segments and in the absence of an open wound the fingers should be immobilized on a curved metal or plaster splint for three or four weeks. Open fractures must be promptly debrided, explored and the bone fragments reduced. Small fragments of bone may be removed if necessary, but large pieces should be aligned for maximum length and shape of the phalanx. Primary arthrodesis of the distal interphalangeal joint is justified if the articular surface is irreparably damaged. An intramedullary pin traversing the distal and middle phalanges facilitates fixation and early fusion. The wound should be closed either by suturing the skin margins or by using a skin graft. Every effort should be made to salvage as much of the finger as possible. Secondary infection is not uncommon and requires vigorous treatment.

Fractures of the Middle Phalanx. Displacements due to injuries of this phalanx depend on the action of the tendon of the flexor digitorum sublimis as related to the fracture site (Fig. 8). If the site is proximal

to the tendon attachment, it will result in flexion angulation of the distal fragment and phalanx with the proximal fragment in extension. If the site is distal to the insertion of the tendon, it will produce flexion of the proximal fragment and dorsal displacement or angulation of the distal fragment. It is important to recognize these two types of displacement if failure in correcting the deformity is to be avoided.

Reduction is accomplished by re-aligning the distal fragment on the proximal one with the patient under local anesthesia. The digit is immobilized either by applying a skin-tight plaster splint with the interphalangeal joints in flexion or preferably by use of a forearm cast which includes the finger. The latter procedure is especially suitable when the fracture is unstable and the deformity is likely to recur. Skeletal traction obtained by a transversely placed pin in the distal phalanx and attached by rubber bands to a wire projecting from a forearm cast is necessary in some oblique and comminuted fractures. Union can be expected to take place in four weeks.

Fractures of the distal end and comminuted fractures elsewhere in the bone may require continuous traction and cast (Fig. 9).

Fractures of the Proximal Phalanx. The proximal phalanx is fractured almost twice as often as the middle or distal phalanx.

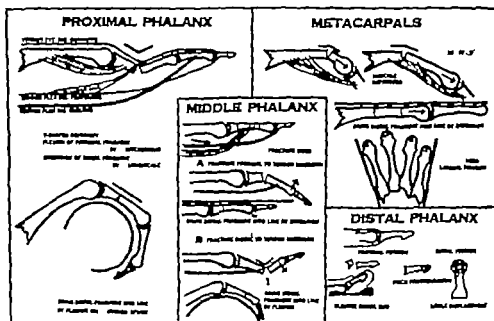


Figure 8. Drawings showing deformities of fractures of the phalanges and metacarpal bones. Note the methods for correcting the alignment. The interphalangeal and metacarpal-phalangeal joints should be immobilized in some degree of flexion whenever possible.

The fracture is usually transverse through the middle third of the bone but may occur in any portion and may frequently be comminuted or open. It either results from a force directed upward from the distal end of the finger or it may be produced by a blow over the bone. The deformity is usually one of forward angulation due to contraction of the lumbricals and interosseous muscles, which normally flex the proximal phalanx and extend the distal interphalangeal joints (Fig. 10).

Treatment consists of a manual reduction of the fracture under local anesthesia. Alignment is brought about by flexing the extended distal fragment on the proximal one. The finger is then immobilized in a forearm cast which includes the involved digit with the metacarpal-phalangeal and interphalangeal joints in a moderate degree of flexion (Fig. 11). Comminuted but stable fractures may be treated in the same way. Unstable oblique, spiral or comminuted fractures require continuous rubber-band traction from the ends of a pin which has been inserted transversely through the distal phalanx. The finger is supported on a curved splint which forms part of the forearm cast; it must not be kept in extension on a banjo splint, for this will lead to contractures of the collateral ligaments of the metacarpal-phalangeal joints and permanent crippling of the hand. Proper alignment is important because resulting rotary or angular deformities with overlapping digits may require osteotomy to

restore satisfactory function of the hand. Union is usually effected in four to six weeks, although it takes much longer to mobilize the finger joints and restore full function of the hand. Paraffin baths are perhaps the best and cheapest form of physical therapy for painful, swollen and stiff joints of the fingers. Forceful manipulation delays recovery and must be avoided. The patient should be encouraged to move the digits actively during the period of cast treatment and especially after immobilization is discontinued.

Avulsion of the Extensor Tendon of the Fingers. A common injury which is frequently mistreated is that of an avulsion fracture of the proximal and dorsal portions of the distal phalanx resulting from a blow on the end of the finger while the distal interphalangeal joint is actively extended. The result is the typical drop or mallet finger seen frequently in baseball players. This condition can also result from a tear of the extensor tendon of a finger at the point of its attachment to the base of the distal phalanx. It is usually a subcutaneous injury without an external wound. Roentgenograms may show a separated small fragment of bone. Pain and swelling may be minimal, but the flexed position, the loss of active extension of the distal phalanx and the local tenderness are diagnostic.

Treatment is the same whether a fragment of bone is torn off by the tendon or the tendon is ruptured at its insertion. In both cases the finger is immobilized for six weeks with

the distal interphalangeal joint in maximum extension and with the proximal joint flexed 45 degrees in order to relax the flexor profundus tendon. A molded plaster cast which extends from the tip of the finger to the metacarpal-phalangeal joint is highly satisfactory although a metal splint will serve just as well. Intraosseous fixation is recommended by some surgeons but should be reserved for exceptional cases. Open reduction and tendon suture are rarely necessary in acute cases but may be necessary to restore extension of the distal phalanx in instances of neglect.

Dislocation of the Metacarpal Phalangeal Joint of the Thumb. Posterior dislocation of the proximal phalanx on the first metacarpal bone may result from a fall on the hand or a blow on the hyperextended thumb. The anterior capsule is torn and in many instances the proximal end of the metacarpal bone is protruded through the defect and may interfere with the reduction. The patient complains of pain, deformity and loss of function of the metacarpal-phalangeal joint. The proximal end of the phalanx can be palpated prominently posterior to the distal end of the metacarpal bone. Roentgenograms should confirm the finding.

The dislocation can be reduced in most instances by downward traction on the thumb and countertraction on the forearm. The reduction may be facilitated by hyper-

extension of the thumb in order to approximate the end of the proximal phalanx to the metacarpal head after which the thumb is realigned. In other cases the head must



Figure 10 Fracture of the proximal phalanx. The characteristic volar angulation is seen.

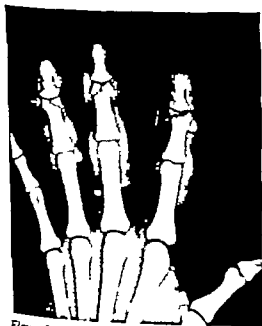


Figure 9 Fractures of the middle phalanges of the long and ring fingers. Note the steplike deformities of the articular surfaces of the middle phalanges of the long and ring fingers. They can usually be reduced by side-to-side compression of the fragments or by traction through the distal phalanx.



Figure 11 Forearm cast which includes one finger with the digit in flexion at all joints. This is a satisfactory means of immobilizing fractures of a metacarpal bone or the proximal and middle phalanges.

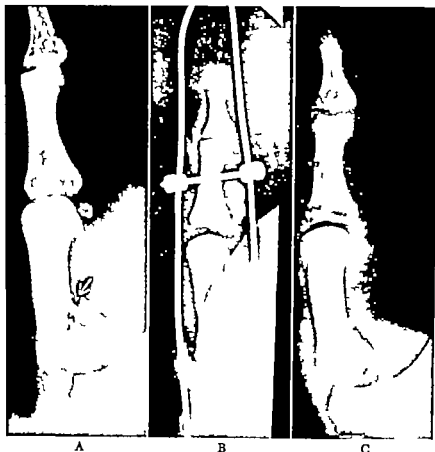


Figure 12. Unstable fracture of the metacarpal bone of the thumb treated by skeletal traction. A, Lateral roentgenogram before treatment. B, Reduction by skeletal traction using threaded wire connected by rubber bands to a metal outrigger. C, Anatomic result after six weeks of traction.

be pushed backward through a rent in the anterior capsule to achieve reduction. Occasionally the flexor pollicis longus tendon becomes caught around the metacarpal bone, necessitating open reduction. After open or closed reduction, the thumb is immobilized in a forearm cast which should extend as far as the distal phalanx of the thumb. The cast may be removed after three to four weeks, when active motion can be resumed.

Fractures of the Metacarpal Bone of the Thumb. Fractures of the shaft of the metacarpal bone of the thumb are a common occurrence because of the exposed position of this organ. The fracture takes place in any portion of the bone and may assume a transverse, spiral or oblique direction. Comminution is frequently encountered. There also may be marked angulation or overriding at the site of the fracture.

The diagnosis is based on the history of the injury with resulting pain, swelling, deformity and loss of function of the thumb. Roentgenograms will reveal the exact nature of the fracture.

Simple transverse fractures with overriding

may be reduced by traction and counter traction or by angulation to engage the ends of the fragments and effect their realignment distally and proximally. Immobilization is obtained by a forearm cast which extends to and encircles the thumb as far as the distal phalanx. Severely comminuted and unstable fractures are treated by skeletal traction similar to that used for the Bennett fracture (Fig. 12). Union occurs in four to six weeks. The final result is usually satisfactory.

Bennett's Fracture Dislocation of the Thumb. There are two fractures which occur at the base of the first metacarpal which are common and characteristic. One is a fracture about $\frac{1}{4}$ inch from the base (Fig. 12) which may be transverse or comminuted and the other is the so-called Bennett's fracture. The latter is distinct in both diagnosis and treatment from the other types of fractures and is, moreover, a potentially disabling injury of the hand which therefore requires special attention.

Bennett's fracture and resulting dislocation involve the proximal articular surface of the metacarpal of the thumb and the ad-

parent bone. The fracture usually results from a force which drives the metacarpal bone up against the multangulum majus and shears off a triangular fragment on the volar side of its base. Consequently the normal stability of the metacarpomultangulum majus joint is lost and the main fragment and the two phalanges dislocate proximally and posteriorly. The small anterior fragment usually maintains its normal relationship to the multangulum majus.

Characteristic signs of this fracture-dislocation are painful motion, local tenderness and marked swelling at the base of the first metacarpal. Roentgenograms are necessary for complete diagnosis.

Reduction is obtained by downward traction on the thumb so that the main fragment of the metacarpal bone approximates the small fractured piece left in situ. Six weeks are required for solid union to occur. It is necessary to maintain continuous skeletal traction during this time. This can be achieved by drilling a wire transversely through the proximal phalanx and attaching it, by means of rubber bands, to a small outrigger which projects from the forearm cast (Fig. 13). End-to-end approximation can be aided by a pressure dressing over the dorsal aspect of the proximal end of the metacarpal. Roentgenograms should be made two days after traction is commenced to determine whether reduction has been effected and at weekly intervals for three more weeks to make sure that alignment is maintained. Union, as confirmed radiologically, should take place within six weeks.

If the fracture is impacted with downward displacement of the small fragments—which fortunately is rare—and both the small and the major fragments are pulled distally by the traction, satisfactory reduction cannot be obtained without recourse to surgery (Fig. 14). The reduction must be accurate

in all cases for incongruous joint surfaces lead to early traumatic arthritis of the metacarpal-carpal joint and marked disability of the thumb.

Fractures of the Metacarpal Bones Exclusive of the Thumb. Fractures of the metacarpal bones are produced indirectly by violence directed upward through the knuckles or directly by a blow to the dorsum of the hand. The most common fracture is a transverse one with impaction and dorsal angulation. Reduction is accomplished by extending the distal fragment into alignment with the proximal portion of the bone. A forearm cast is applied which includes the hand and the finger corresponding to the injured metacarpal.

Transverse fractures of the shaft of the bone with overriding are reduced by manipulation using the angulation method. The fragments are manually angulated dorsally so as to engage the bone ends after which reduction is completed by extending the distal fragment. Open reduction is occasionally indicated because not all of these fractures can be adequately reduced by manipulation. Skeletal traction through the corresponding finger cannot usually mobilize the distal fragment sufficiently to effect a reduction.

Oblique or spiral fractures of the metacarpal bone with slight overriding can be treated by continuous traction by means of a threaded wire drilled through the middle phalanx with the interphalangeal and metacarpal phalangeal joints flexed on a curved wire or metal splint which is incorporated in a forearm cast. The patient is encouraged to move the distal phalanx to prevent fixation of the tendons by adhesions. Traction may be discontinued at the end of four weeks in the presence of adequate early callus although six weeks or more may be required for complete healing. Open reduc-

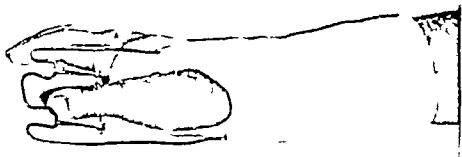


Figure 13. Type of cast and skeletal traction used for fractures and fracture-dislocations of the metacarpal bone. The web of the thumb must be well padded to prevent pressure sore.



Figure 14. Bennett fracture treated by open reduction after failure of skeletal traction. A, Lateral view showing persistent displacement of small fragment. B, Pin fixation following open reduction. C Final result after removal of pins.

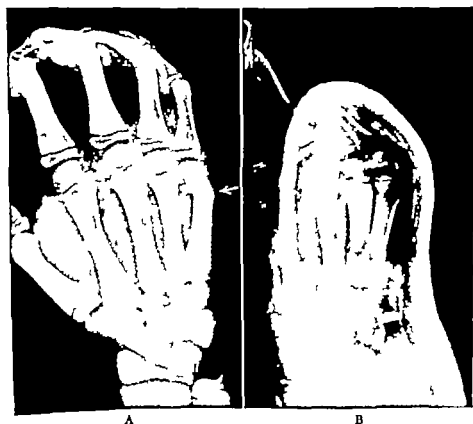


Figure 15. Impacted fracture of the neck of the fifth metacarpal bone. A, Before reduction, B, after reduction and application of cast.

tion and internal fixation by screws designed for this purpose are justified in some cases.

Fractures through the neck of the metacarpal bones involve most often the fifth metacarpal (Fig. 15). They result most frequently from a direct blow to the head of the metacarpal bone, as happens especially in fist fights. The distal fragment is forced volarward and usually impacts into the end of the proximal fragment. Reduction is effected, with the patient under local or general anesthesia, by disimpacting the distal fragment and restoring bone alignment—a procedure which often requires considerable force. It should be emphasized that this deformity cannot be corrected by traction on the extended finger, since the collateral ligaments of the metacarpal phalangeal joints are relaxed in this position and the traction force is not transmitted to the metacarpal bone. Some surgeons recommend that reduction be effected by applying upward pressure on the distal end of the proximal phalanx after putting the metacarpal phalangeal and proximal interphalangeal joints in 90 degrees of flexion. In other cases the impaction is best corrected by pressure applied to the volar aspect of the distal fragment by the physician's index finger, while a counterforce is made over the site of fracture with the thumb (Fig. 16). The digit is immobilized in a flexed position following restoration of alignment of the metacarpal bone. A band of plaster is placed

about the wrist. A splint of plaster is made to extend distally from it over the metacarpal bone around the finger and back to the volar aspect of the wrist. The plaster is molded so as to exert upward pressure on the distal fragment of the metacarpal bone. A pad over the flexed proximal interphalangeal joint avoids pressure necrosis. Roentgenograms are made at the end of ten days because the tendency for the deformity to recur is greatest at that time. Union is usually sufficiently advanced at the end of four weeks so that the cast may be discarded and function resumed. Malunited fractures with the metacarpal head projecting prominently in the palm may require surgical correction.

THE WRIST JOINT

Fractures of the Navicular Bone. The navicular bone is the most commonly injured bone in the wrist. Trauma to this bone is especially frequent in young persons who engage in sports and similar physical activities. Fractures result from a fall on the outstretched hand, the force of which compresses the navicular bone between the multangulum majus distally and the end of the radius proximally. The same type of fall may produce a Colles' fracture in older individuals and a fracture of the distal portion of the shafts of the radius and ulna in young children. Navicular bone fractures offer an excellent prognosis for union and restitution of normal wrist function, provided the injury is recognized early and proper treatment is instituted.

Fractures are of three varieties, based on anatomic location. They are respectively fractures through the distal portion or tubercle through the waist or midportion, or through the proximal third of the bone. Fractures through the waist or midportion are more common than those of the other two varieties (Fig. 17). Nonunion is the rule in fractures of the body if they are not diagnosed accurately and treated properly.

The pain may be so slight as not to induce the patient to seek medical care. The swelling is most marked in the radial half of the wrist joint and restricts motion. Tenderness is elicited over the navicular and especially the anatomic snuff box, which itself might become obliterated by the swelling. The diagnosis should be suspected when these findings are present and confirmed by adequate roentgenograms, including anteroposterior, lateral and oblique views of the wrist. In some cases the fracture line may



Figure 16. Alternate method of reducing impacted fracture of neck of fifth metacarpal bone.

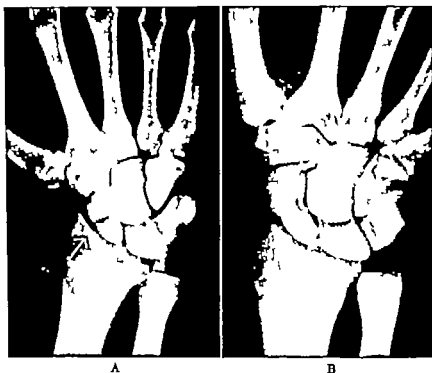


Figure 17 Anteroposterior views of fracture of navicular bone. A, Before treatment; B the final result.

be so faint that it cannot be detected on the x ray film. In these patients, the diagnosis is usually a sprain of the wrist and treatment is accordingly misdirected.

It is safest, therefore to treat all so-called sprained wrists as though they were fractures of the navicular bone and apply a forearm cast which encases the hand up to the distal flexion crease and extends to the thumb as far as the distal phalanx. The wrist is placed in dorsiflexion and the thumb in neutral position. The cast will relieve symptoms and also immobilize a fracture when present (Fig. 18). If after three weeks of immobilization, the x ray picture again shows no fracture line, the cast is discarded and the wrist is permitted to resume function. If on the other hand, a fracture is demonstrated, a similar snug fitting plaster cast is applied and immobilization is continued until union is demonstrated on the roentgenograms. The cast may be changed at intervals of six weeks, at which times roentgenographic checks should be made to determine the degree of healing.

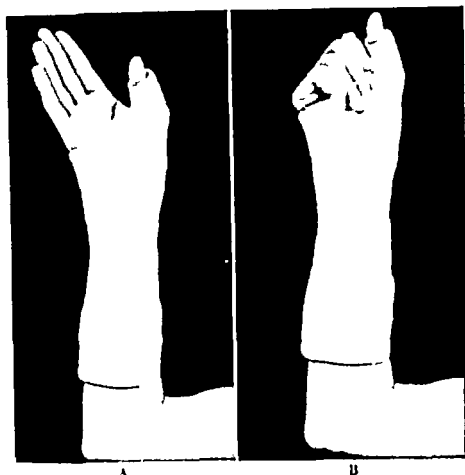
Fractures through the tubercle, or the extra-articular portion of the navicular bone, retain an adequate blood supply and union occurs in about six weeks. Fractures through the body or waist, of the bone usually require three to six months to unite. Practically all fresh fractures of the navicular bone will heal if uninterrupted immobilization of the wrist is continued long enough,

which may be up to twelve months. Loss of blood supply to the proximal fragment at the time of injury may prevent union and a satisfactory result in some cases (Fig. 19).

Nonunion may be due to one or more of the following factors: failure to recognize an acute fracture or to institute immediate cast immobilization and continue it until union is shown on the roentgenograms, an insufficient blood supply with resulting necrosis of the proximal fragment.

Nonunion with two viable fragments is compatible with normal functioning of the wrist. If pain is present, union may be achieved by means of an intramedullary bone graft obtained from the styloid process of the radius. The styloidectomy will at the same time relieve the navicular bone of pressure which, in some patients, may cause pain at the extremes of flexion and extension.

Nonunion, which is accompanied by aseptic necrosis of the proximal fragment, offers a less favorable prognosis. The clinical diagnosis of the necrosis can be made only with the aid of roentgenograms. Immobilization of the fracture in a cast causes the viable distal fragment and the surrounding living bones to undergo atrophy of disuse, while the dead proximal navicular fragment will retain its original density. After eight to ten weeks of treatment in a cast, x ray evidence will show the necrotic proximal fragment to have a relatively high and unchanged density and the living distal frag-



A

B

Figure 18. Photographs showing plaster cast used in the treatment of fractures of the navicular bone. The plaster is molded well in the palm at 1 permit full flexion of the first ray.

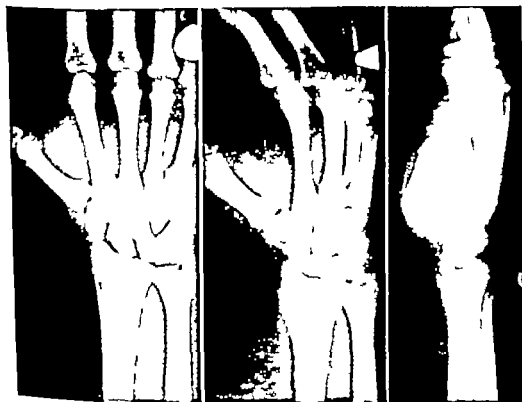


Figure 19. Aseptic necrosis of navicular bone following fracture and union. Note unchanged density of proximal half compared to atrophic distal portion and other carpal bones, due to osteoporosis from cast treatment of original fracture.

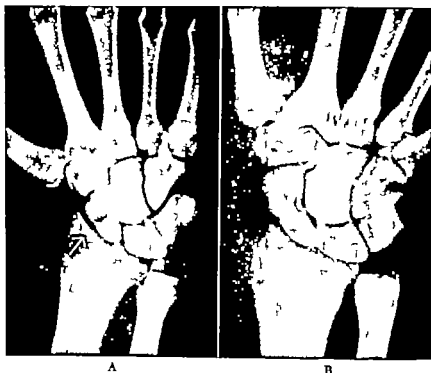


Figure 17 Anteroposterior views of fracture of navicular bone. A, Before treatment; B, the final result.

be so faint that it cannot be detected on the x-ray film. In these patients the diagnosis is usually a sprain of the wrist and treatment is accordingly misdirected.

It is safest, therefore to treat all so-called sprained wrists as though they were fractures of the navicular bone and apply a forearm cast which encases the hand up to the distal flexion crease and extends to the thumb as far as the distal phalanx. The wrist is placed in dorsiflexion and the thumb in neutral position. The cast will relieve symptoms and also immobilize a fracture when present (Fig. 18). If after three weeks of immobilization, the x-ray picture again shows no fracture line, the cast is discarded and the wrist is permitted to resume function. If, on the other hand, a fracture is demonstrated, a similar snug fitting plaster cast is applied and immobilization is continued until union is demonstrated on the roentgenograms. The cast may be changed at intervals of six weeks at which times roentgenographic checks should be made to determine the degree of healing.

Fractures through the tubercle, or the extra-articular portion of the navicular bone, retain an adequate blood supply and union occurs in about six weeks. Fractures through the body or waist, of the bone usually require three to six months to unite. Practically all fresh fractures of the navicular bone will heal if uninterrupted immobilization of the wrist is continued long enough,

which may be up to twelve months. Loss of blood supply to the proximal fragment at the time of injury may prevent union and a satisfactory result in some cases (Fig. 19).

Nonunion may be due to one or more of the following factors: failure to recognize an acute fracture or to institute immediate cast immobilization and continue it until union is shown on the roentgenograms; an insufficient blood supply with resulting necrosis of the proximal fragment.

Nonunion with two viable fragments is compatible with normal functioning of the wrist. If pain is present, union may be achieved by means of an intramedullary bone graft obtained from the styloid process of the radius. The styloidectomy will at the same time relieve the navicular bone of pressure which, in some patients, may cause pain at the extremes of flexion and extension.

Nonunion which is accompanied by aseptic necrosis of the proximal fragment, offers a less favorable prognosis. The clinical diagnosis of the necrosis can be made only with the aid of roentgenograms. Immobilization of the fracture in a cast causes the viable distal fragment and the surrounding living bones to undergo atrophy of disuse, while the dead proximal navicular fragment will retain its original density. After eight to ten weeks of treatment in a cast, x-ray evidence will show the necrotic proximal fragment to have a relatively high and unchanged density, and the living distal frag-

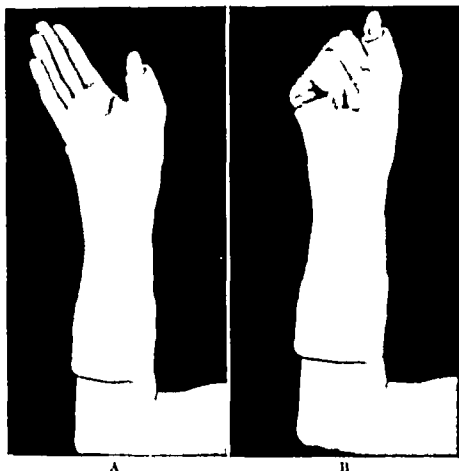


Figure 18. Photographs showing plaster cast use I in the treatment of fractures of the navicular bone. The plaster is made I well in the palm and permits full flexion of the fingers.

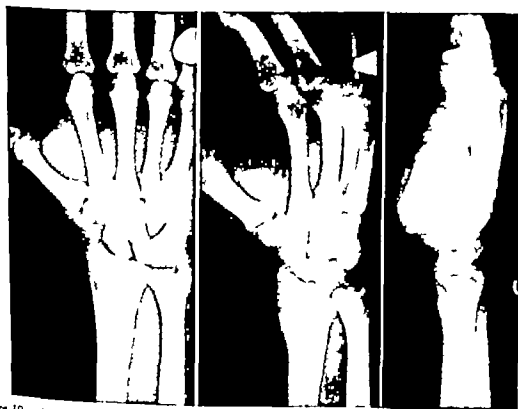


Figure 10. Aseptic necrosis of navicular bone following fracture and union. Note unchanged density of proximal half compared to atrophied distal portion and other carpal bones, due to osteoporosis from cast treatment of original fracture.

ment to have a low atrophic density (osteoporosis) In spite of this complication, cast immobilization must be continued without interruption, for consolidation might occur as blood vessels extend across the fracture site and gradually replace the dead fragment by new living bone through the process of creeping substitution. The end result may be very satisfactory.

If union does not occur between a live distal and a devitalized proximal fragment, disabling arthritis of the wrist, which is difficult to relieve, is sure to result. Of the numerous corrective procedures which have been recommended for the treatment of non union, radial styloidectomy without excision of the dead fragment combined with an intramedullary bone graft across the fracture site is preferable, provided the dead fragment is intact and the symptoms are relatively mild. If union occurs it will bring about gradual revitalization of the fragment; this may be checked by serial roentgenograms taken at three- to six month intervals. The bone fragment will gradually appear mottled or even cystic as the high density necrotic bone is replaced by low density atrophic living bone. This process spreads from the fracture site to the proximal extremity of the fragment. The replacement will be complete when the bone reassumes a more or less homogeneous appearance and trabecular structure, but, in spite of successful union and complete restitution of the

necrotic bone arthritis of the wrist joint may eventually occur.

In the presence of both nonunion and disabling arthritis (Fig. 20) excision of the devitalized fragment alone is rarely successful in relieving the wrist symptoms. The simple operation of styloidectomy offers a satisfactory prognosis, but arthrodesis is the only procedure which will definitely restore a painful wrist to satisfactory usefulness (Fig. 20).

Dislocation of the Lunate Bone. Although fractures of the carpal bones other than the navicular are uncommon, dislocations of the lunate bone are common (Fig. 21) This bone and the navicular form the junction between the radius and the hand. The proximal convex surface of the lunate articulates with the lower end of the radius medial to the navicular and with the cartilaginous disk of the distal radioulnar articulation. The distal surface of the lunate is deeply hollowed to accommodate the head of the capitate and the adjacent portion of the hamate bone.

Dislocation of the lunate results usually from a hyperextension injury such as may occur from a fall on the dorsiflexed hand. The injury may cause the ligaments between the capitate and lunate and lunate and radius to rupture, thus permitting the lunate bone to rotate forward. The latter may be partly or wholly dislocated and come to rest anterior to the distal end of the radius and



Figure 20 Advanced arthritis of wrist secondary to nonunion of navicular with necrosis of proximal fragment. Pain was relieved and motion improved by styloidectomy.

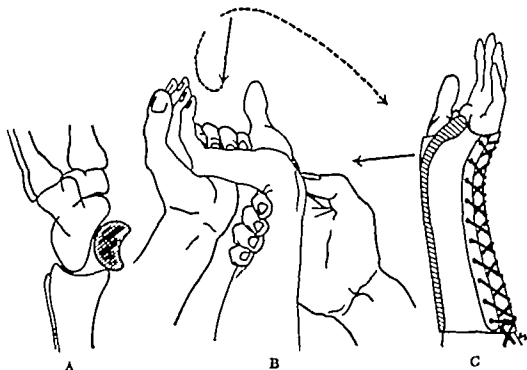


Figure 21. A, Drawing showing anterior dislocation of the lunate bone. B One method of closed reduction. C, Leather molded cock-up splint used during the convalescent period.

beneath the flexor tendons of the wrist and the median nerve.

Clinically the patient experiences great pain and swelling of the wrist with limitation of movement. The fingers are held in semiflexed position. A characteristic sign which may suggest the diagnosis is that of pain or tingling in the distribution of the median nerve. This is due to direct pressure on the nerve by the forwardly displaced lunate crowding the carpal tunnel. Another characteristic sign, but one which is not always present, is that of alteration in the relative positions of the heads of the second, third and fourth metacarpals. Normally the knuckle of the middle finger is more distal than the others but, with the dislocation of the lunate, this knuckle may lie on the same level as those of the index and ring fingers because of a proximal shift of the metacarpal bone. The diagnosis is confirmed by roentgenograms. Though the dislocation can be recognized in the anterior view it is shown more clearly in the lateral exposure. In the severe degrees, the articular surfaces of the lunate are completely separated from the radius and the surrounding carpal bones. In the less severe injuries the articular surface of the lunate and radius may still be in contact to some extent.

Early reduction is essential. Acute dislocations can be corrected by manipulation, that is, by strong downward traction on the hand and simultaneous countertraction on

the arm. This will pull the capitate bone away from the end of the radius where it has shifted, and restore space into which the lunate can be reduced by applying backward pressure with the fingers. Japanese traps on the fingers may be used to obtain strong traction (Fig. 22). Countertraction is produced by flexing the elbow at 90 degrees and placing a sling over the lower end of the humerus. The lunate can then be easily manipulated back into its normal position. A forearm cast which extends as far as the distal flexion crease of the hand maintains the reduction the wrist is placed in neutral position. The cast is removed at the end of four weeks, when motion can be resumed,



Figure 22. Photograph showing Japanese finger traps, useful in the reduction of fractures of the bones of the wrist and forearm.



Figure 23. Spontaneous aseptic necrosis of lunate, Kienbock's disease, with secondary deformity and fractures (A and B) treated by excision (C) with satisfactory result

and a removable leather molded splint is used for an additional six weeks to protect the wrist.

Dislocation of the lunate older than three weeks cannot ordinarily be reduced by manipulation although in injuries not yet three weeks old it may be worth while to apply the Japanese finger traps.

Persistent dislocations require excision of the bone. The first step is to make a transverse incision in the flexor crease of the wrist. The flexor tendons and median nerve are then retracted ulnarward after the volar carpal ligament has been sectioned. The lunate bone is isolated and removed because it is separated from its blood supply with resulting aseptic necrosis. A disabled wrist would eventuate if the lunate were retained.

The first symptoms of Kienbock's disease frequently follow trauma to the wrist with fracture of the lunate bone (Fig. 23). This condition is spontaneous aseptic necrosis which produces compression deformities, pain, limitation of motion and marked disability. A leather molded, metal-reinforced splint should be used in early cases to protect the wrist during replacement of the dead bone. Excision of the lunate is required when the bone is badly deformed.

Perilunar Dislocation of the Wrist. A perilunar dislocation of the wrist joint consists of posterior dislocation of all the carpal bones with the exception of the lunate, which retains its normal position. The navicular bone may be fractured at the same time, and in some cases the proximal half of the bone may remain in situ along with the lunate bone (Fig. 24). These injuries are frequently misdiagnosed and therefore are either inadequately treated or missed entirely resulting in grave crippling of the hand.

Physical signs include marked deformity of the wrist with swelling, pain and loss of function. Roentgenograms must be carefully studied for minute details.

Reduction of the dislocation is effected by strong traction and countertraction, with sudden forward flexion of the wrist. In some instances, open reduction is required, particularly if the proximal fragment of the navicular bone has become rotated and blocks closed reduction. Simple perilunar dislocations are immobilized in a forearm cast with the wrist held straight for a period of six weeks. If there is an associated fracture of the navicular bone immobilization in the cast must continue.

errupt

until roentgen ray evidence confirms the consolidation of the fracture. This may sometimes require from six to nine months.

Old and unrecognized perilunar dislocations of the wrist are best treated by open reduction and arthrodesis of the wrist joint.

Colles' Fracture. This is the most common of all fractures. It is frequently transverse and situated within an inch of the distal articular surface of the radius (Fig. 25) In the majority of cases either the triangular cartilage between the radius and

ulna is torn or the styloid process of the ulna is fractured. The injury results from indirect violence and most commonly from a fall on the outstretched hand. It may occur at any age but is common in persons of the older age groups. The same injury in young and active adults will more likely produce a fracture of the navicular bone and in adolescent children a fracture-dislocation of the distal radial epiphysis.

Clinically the typical Colles' fracture produces a so-called silver fork deformity and

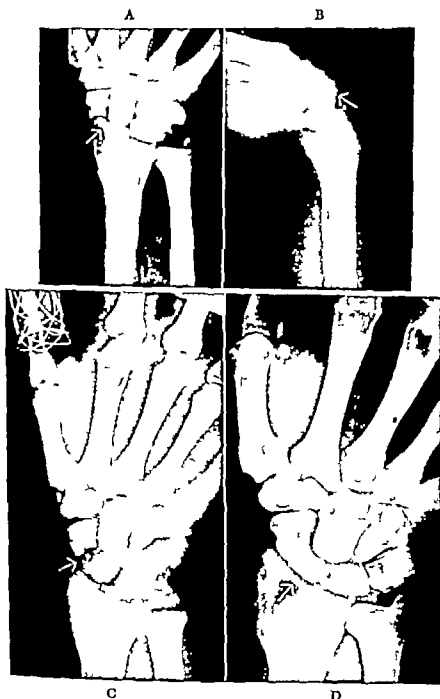
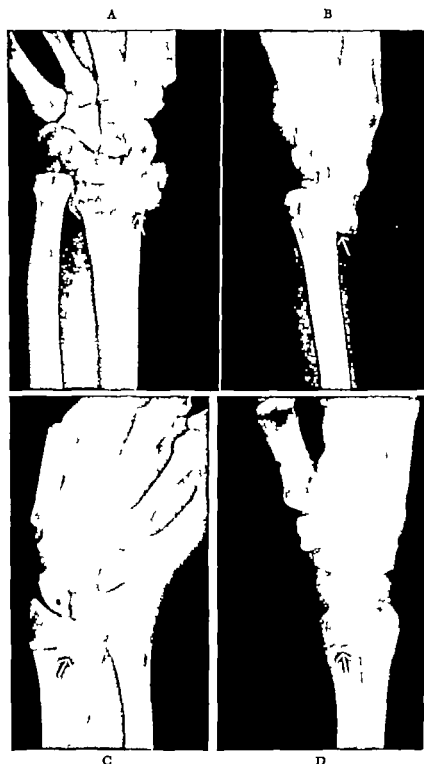


Figure 24. Posterior perilunar dislocation of the wrist with a simultaneous fracture of the navicular bone. A, Anterior view showing injury. B, Lateral view before treatment. C, Anterior view showing unsuccessful reduction by traction because of rotation of proximal fragment of navicular bone. D, Final result. Note excellent reduction and union of the navicular bone, but with a dead proximal fragment after open reduction.



Figures 25. Roentgenograms of a typical Colles fracture. A, Note the shortening and comminution of the distal fragment. B, Lateral view showing reversal of the distal articular surface. C and D, Excellent result obtained by closed reduction and continuous skeletal pin fixation in a forearm cast.

the usual symptoms of a fracture. The deformity is due to the posterior displacement of the distal fragment and is demonstrated clearly in lateral roentgenograms of the wrist. These lateral views also will show that the articular surface of the radius faces the dorsum of the hand, rather than being

directed slightly into the palm. An anteroposterior view will reveal, moreover, that the radius is shortened, as will be evident when the level of the styloid process of the radius is compared with that of the ulna. Normally the radial styloid should extend distally from $\frac{3}{8}$ to $\frac{7}{8}$ inch less than that

of the ulna. Not infrequently the distal fragment is displaced a little to the radial side and may be angulated in the direction of supination. Finally it should be noted whether or not the fracture lines have entered the wrist joint and also to what degree the posterior cortex of the radius at the fracture site has been comminuted.

Reduction of the Colles fracture may be performed with the patient under local or general anesthesia. The latter is preferable whenever possible, since it avoids the necessity of adding additional fluid to the already swollen fracture site. It also affords a better opportunity to obtain maximum reduction because of more complete muscle relaxation.

There is more than one way of reducing a Colles fracture, but there are certain definite steps which must be carried out. First, the fracture must be disimpacted which is done by forcible dorsiflexion of the distal fragment to effect its separation from the proximal one. Strong downward traction is simultaneously exerted on the hand and countertraction on the forearm. When the distal fragment has been brought down to length, the wrist is moved forward and ulward to engage the end of the proximal fragment. The wrist is next placed in moderate flexion to tighten the radial collateral ligament and the posterior capsule, which are attached to the distal fragment, and thus to safeguard against the possibility of having the deformity recur in the cast. Japanese finger traps offer one means of obtaining steady traction during the reduction and application of the cast.

The plaster dressing used for immobilization may be a circular cast or anterior and posterior plaster splints which have the additional advantage of being easily adapted to the degree of swelling (Fig. 26). If the fracture is comminuted or unstable, the plaster dressing should reach above the elbow. Roentgenograms must be made immediately to check the accuracy of the reduction. If it is unsatisfactory the cast must be removed and the manipulation repeated before the anesthesia wears off or the patient awakens. The aim should be to obtain as accurate a reduction as is possible and to do so with the least amount of trauma to the soft tissues.

Ordinarily it is best to hospitalize the patient overnight, for then the swelling can be kept down by elevating the arm and the state of circulation in the fingers can be checked. In case of marked swelling, it will be necessary to split the cast along the ulnar

side and to cut through the underlying dressings to the skin to restore adequate circulation. Roentgenograms must be repeated at the end of ten days and again after two weeks to determine whether reduction is being maintained so as to make remanipulation unnecessary. Union of the fracture ordinarily takes place in six weeks, at which time the cast may be removed and function resumed.

Fractures which alter the articular surface of the radius as well as those with severe comminution of the posterior cortex of the radius are best treated by continuous traction. This can be done while the patient is ambulatory by introducing a threaded wire through the midportion of the metacarpal bone of the thumb and another wire through the proximal crest of the ulna. The two wires are then incorporated in a forearm cast which is applied after the fracture is reduced by manipulation. Wrist and hand are immobilized with slight ulnar deviation but little flexion. This method avoids the Cotton Loader position of marked flexion and extreme ulnar deviation of the wrist, while at the same time maintaining a wide space be-

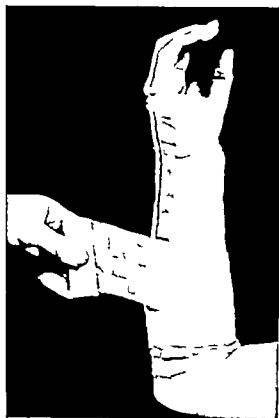


Figure 26 Method of applying anterior and posterior molded plaster splints. The splints of plaster are applied over several turns of sheet wadding after stockinet has been inserted on the arm. A wet plain gauze roll molds the plaster to the contour of the extremity.

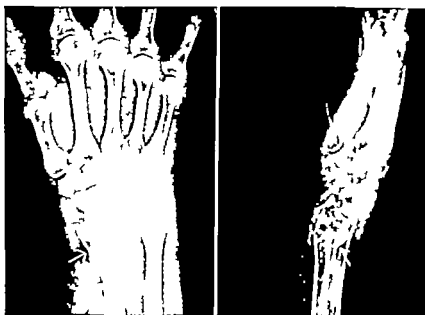


Figure 27 Malunited Colles fracture with permanent shortening of the radius and dorsal angulation of the distal fragment. Note the extreme osteoporosis of the bones. Motion of the wrist was limited and painful.

tween the fragmented surface of the radius and the articular surfaces of the proximal row of carpal bones. It is unlikely that fibrous adhesions which may grow across the joint surface will cause marked and permanent limitation of motion of the wrist joint.

Finally it should be noted that the Colles fracture is frequently considered to be a simple therapeutic problem and consequently is poorly treated (Fig. 27). This attitude is deplorable, because it is really a difficult fracture to treat successfully.

Smith Fracture (Reverse Colles) This interesting but rather rare fracture is most commonly the result of direct violence which forces the hand volarward. It may also be caused indirectly by a fall on the dorsum of the strongly flexed hand. The line of fracture is somewhat variable but is usually roughly similar to that of the Colles fracture, except that the distal fragment is displaced volarward and proximally (Fig. 28). The deformity is characteristic but in most instances it is less obvious than the silver fork appearance of the hand and wrist seen in Colles fracture (Fig. 29). Comminution is often present and makes it difficult to maintain the proper position of the fragments after reduction.

The signs and symptoms are much the same as for a Colles fracture and the general deformity has aptly been called "spade-handle deformity."

Reduction may be effected with the aid of local or general anesthesia. The treatment

follows principles which are similar to those employed for the Colles fracture, except that the movements of reduction are reversed. Strong traction is exerted on the hand and countertraction on the lower end of the humerus with the elbow in 90 degrees of flexion. Impaction, if present, is disengaged by exaggerating the flexion position of the small fragment, following which the fracture is reduced by strong traction. The wrist is next brought into dorsiflexion with ulnar deviation of the hand and is immobilized by means of anterior and posterior plaster splints or by a circular plaster dressing which extends from the elbow to the distal flexion crease of the hand, thus permitting full use of the fingers and thumb.

Roentgenograms should immediately be made to check the accuracy of the position of the fragments. They should be repeated after ten days. If no displacement has occurred, the cast is left in place for six weeks and then discarded. No sling is allowed during that time and full use of the fingers, elbow and shoulder is encouraged as in all wrist fractures. If on the other hand, reduction has not been maintained, as evidenced by roentgenograms taken at the end of ten days, the fracture must be remanipulated. In some of these fractures the difficulty encountered in maintaining good reduction is more troublesome than in Colles fractures. But, if in these patients the best alignment possible is obtained, if the wrist is put in extension with some ulnar deviation and if active use of the fingers is

and from

the first, a very good functional result will be obtained.

Marginal Fractures of the Distal End of the Radius. These fractures may occur along the anterior or the posterior margin of the distal end of the radius. They result from forceful flexion or extension of the hand

causing the carpal bones to impinge against one of the marginal ends just noted, and splitting off a segment. The fracture fragment whether from the anterior or the posterior margin always includes a portion of the joint surface. Upward displacement is rarely marked however. The injury is char

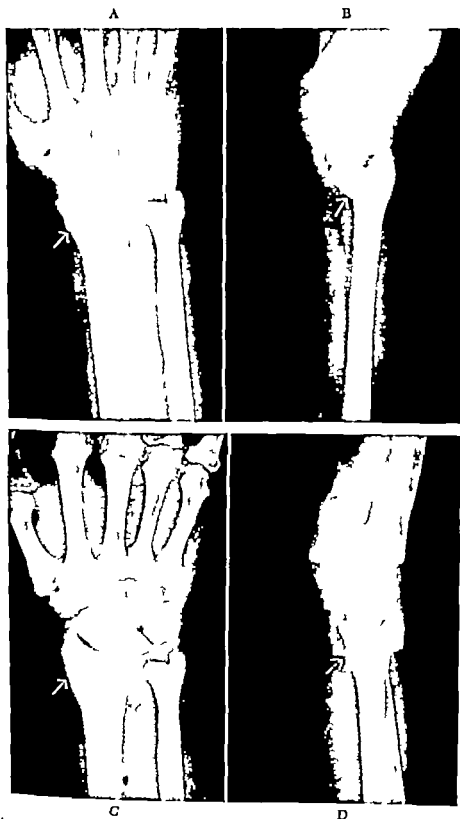


Figure 28. Anteroposterior and lateral roentgenograms of Smith fracture. A and B Before treatment, C and D After treatment.

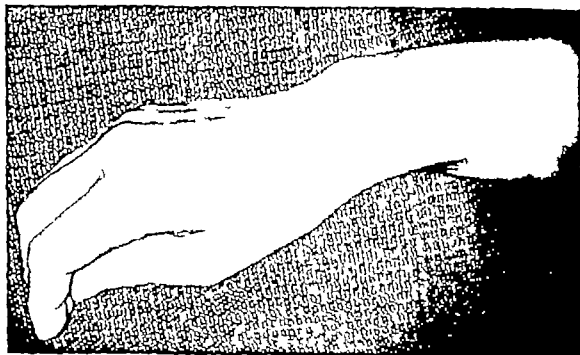


Figure 29 Photograph of unreduced Smith fracture showing reverse Colles' deformity. Note prominence of ulna.

acterized by pain, swelling, and limited motion of the wrist joint. Local tenderness and crepitus may also be present. Roentgenograms will confirm the diagnosis.

These fractures are not difficult to reduce but must not be neglected or treated lightly. The reduction is accomplished, with the patient under local anesthesia, by exerting moderate downward traction on the hand and countertraction on the elbow and by molding the fragment. Both anterior and posterior marginal fractures should be immobilized with the wrist held in slight dorsiflexion. Plaster splints applied along the front and back of the forearm from the elbow to the distal flexion crease of the hand offer adequate fixation. The plaster is molded from front to back at the site of the fracture to close such gap as may remain between the fragment and the radius. Union usually takes place within six weeks, at which time the cast may be discarded and full use of the arm resumed.

Fracture-Dislocation of the Distal Radial Epiphysis. An injury in adolescent children which is more or less comparable to the Colles' fracture in adults, is the fracture-dislocation of the distal radial epiphysis (Fig. 30). The injury results from forceful hyperextension of the wrist, such as in a fall on the hand. The fracture occurs through the radius just proximal to the distal epiphyseal growth plate. The fragment consists of the distal articular surface, the epiphyseal

ossification center, the cartilaginous growth plate and varying amounts of the adjacent metaphysis of the radius. The degree of dorsal displacement of the epiphysis varies from none in some cases to complete posterior dislocation in others.

The usual symptoms of a fracture are present. They consist of deformity, swelling, pain on attempted movement, muscle spasm and local tenderness.

A fracture-dislocation of the distal radial epiphysis is reduced by the same type of manipulation as that used for the Colles' or other extension fractures of the wrist. Downward traction is exerted on the fingers and countertraction is obtained at the elbow region. When the distal small fragment clears the lower end of the radius, the hand, including the distal fragment, is brought forward to re-align the radius. A long arm cast is applied with the wrist in moderate flexion and ulnar deviation.

The reduction is best performed with the patient under general anesthesia. Roentgenograms are obtained before the patient is allowed to awaken. If the reduction is unsatisfactory it is repeated. There should be no difficulty in obtaining accurate reduction of this fracture. It is however best to accept some degree of persistent displacement of the distal radial epiphysis rather than to perform an open reduction, for this provokes a premature closure of the epiphyseal growth zone with resultant shortening

of the radius and overgrowth of the ulna. Growth arrest rarely occurs when the fracture is reduced gently by closed methods even when the reposition is not complete.

Follow-up care is the same as for other fractures of the wrist. Sufficient union is present at the end of six weeks to allow the removal of the cast. No physical therapy is necessary. The patient should be followed

for several years to determine if arrest of growth of the radius has resulted from the fracture. Yearly roentgenograms will show whether longitudinal growth of the radius is occurring at the normal rate. Premature closure of the epiphysis may require surgical obliteration of the corresponding growth plate of the ulna and a shortening of the bone to the level of the radius de

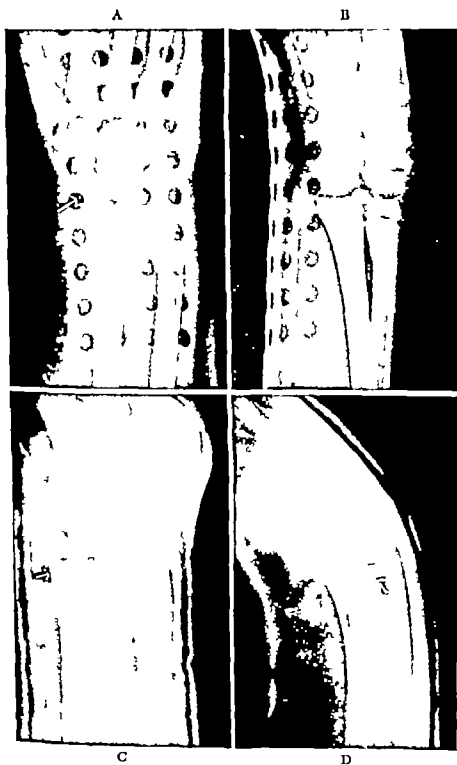


Figure 90. Fracture-dislocation of the distal radial epiphysis treated by manipulation and cast. A and B, Anteroposterior and lateral views showing the deformity. C and D, Similar views showing normal reposition of the fragment after closed reduction.

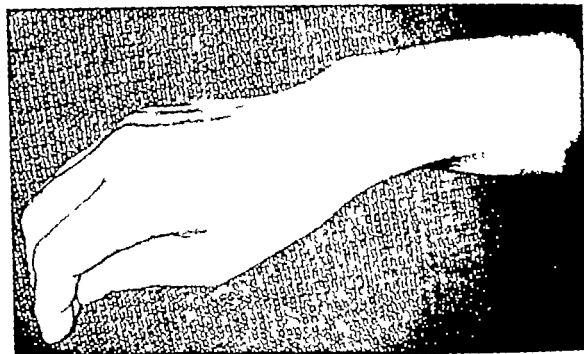


Figure 29 Photograph of unreduced Smith fracture showing reverse Colles deformity. Note prominence of ulna.

acterized by pain, swelling, and limited motion of the wrist joint. Local tenderness and crepitus may also be present. Roentgenograms will confirm the diagnosis.

These fractures are not difficult to reduce but must not be neglected or treated lightly. The reduction is accomplished, with the patient under local anesthesia, by exerting moderate downward traction on the hand and countertraction on the elbow and by molding the fragment. Both anterior and posterior marginal fractures should be immobilized with the wrist held in slight dorsiflexion. Plaster splints applied along the front and back of the forearm from the elbow to the distal flexion crease of the hand offer adequate fixation. The plaster is molded from front to back at the site of the fracture to close such gap as may remain between the fragment and the radius. Union usually takes place within six weeks at which time the cast may be discarded and full use of the arm resumed.

Fracture Dislocation of the Distal Radial Epiphysis. An injury in adolescent children, which is more or less comparable to the Colles fracture in adults, is the fracture-dislocation of the distal radial epiphysis (Fig. 30). The injury results from forceful hyperextension of the wrist, such as in a fall on the hand. The fracture occurs through the radius just proximal to the distal epiphyseal growth plate. The fragment consists of the distal articular surface, the epiphyseal

ossification center, the cartilaginous growth plate and varying amounts of the adjacent metaphysis of the radius. The degree of dorsal displacement of the epiphysis varies from none in some cases to complete posterior dislocation in others.

The usual symptoms of a fracture are present. They consist of deformity, swelling, pain on attempted movement, muscle spasm and local tenderness.

A fracture-dislocation of the distal radial epiphysis is reduced by the same type of manipulation as that used for the Colles or other extension fractures of the wrist. Downward traction is exerted on the fingers and countertraction is obtained at the elbow region. When the distal small fragment clears the lower end of the radius the hand, including the distal fragment, is brought forward to re-align the radius. A long arm cast is applied with the wrist in moderate flexion and ulnar deviation.

The reduction is best performed with the patient under general anesthesia. Roentgenograms are obtained before the patient is allowed to awaken. If the reduction is unsatisfactory it is repeated. There should be no difficulty in obtaining accurate reduction of this fracture. It is however best to accept some degree of persistent displacement of the distal radial epiphysis rather than to perform an open reduction, for this provokes a premature closure of the epiphyseal growth zone with resultant shortening

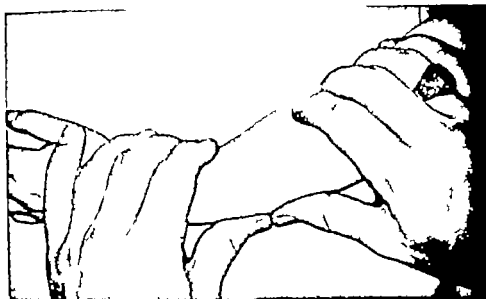


Figure 32. Photograph showing position of surgeon's hands during the manipulation of a green-stick fracture.

muscle, it is necessary to hold the forearm distal to the fracture in midposition between supination and pronation during the manipulation. In case of doubt as to the degree of supination of the proximal fragment of the radius, an anteroposterior x ray film of the forearm should be compared with films of the normal radius taken in known positions of rotation.

Fractures of the Radius and Ulna in Children. Most fractures of the radius and ulna in children can be treated effectively by manipulative reduction and application of a long arm cast. Green-stick fractures, most common in children, represent an incomplete break through the bone; they are characterized by extreme degrees of angulation which is most commonly in a volar direction (Fig. 31). Except for the presence of pain and the obvious angular deformity such common fracture signs as crepitus and false motion are absent. Roentgenograms in the anteroposterior and lateral projections should be taken prior to reduction.

The treatment of an incomplete or green-stick fracture of the radius and ulna consists essentially of re-aligning the distal fragment on the proximal one. No general or local anesthesia is necessary and sedation with codeine or morphine will suffice if properly adjusted to the child's age and temperament. The surgeon grasps the extremity distal and proximal to the fracture while placing his thumbs along its apex (Fig. 32). Reduction is accomplished by pushing forward firmly with the thumbs and by simultaneously levering the distal fragment backward until the bone snaps in two. The completely fractured bone can then be brought into normal

alignment. Care must be taken to avoid loss of the end-to-end position of the fragments. It is possible that the ulnar deformity may correct itself while the radius is being manipulated but more often it is necessary to repeat the maneuver just described. Immobilization is effected by means of a long arm cast with the elbow in 90 degrees of flexion and the wrist in moderate flexion and ulnar deviation. The cast should be made to fit snugly and yet allow for the swelling which occurs during the first twelve to twenty four hours. Angulation in the cast is less apt to take place if the plaster is well molded from front to back over the fracture site. Roentgenograms should check the accuracy of the reduction. If some angulation has occurred or the reduction has otherwise become unsatisfactory the cast may be wedged or if necessary removed and the manipulation repeated. An x ray examination must be made ten to fourteen days after reduction because of the tendency at that time for angulation to recur as the swelling subsides and the cast becomes loosened.

Wedging is accomplished by making parallel cuts spaced $\frac{3}{4}$ inch apart, through the sides of the cast, the cuts being centered over the fracture site. A window is then made in the front (or back) of the plaster between the adjacent cuts with its location on the side to which the cast is to be wedged. A single saw cut is finally made transversely across the cast opposite the window connecting with the parallel cuts. The distal portion of the cast which encases the lower fragment is then angulated sufficiently to open a wedge and to close the window at the same time that the alignment of the

pending upon the age of the patient at the time of injury. This fortunately is not a common injury but carries a guarded prognosis—a fact which should at once be explained to the parents.

THE RADIUS AND ULNA

Fractures of the shafts of the radius and ulna usually are the result of a direct blow to the bones or indirectly a fall on the outstretched hand. In adults the resulting fracture has a tendency to be comminuted and to be oblique with overlapping. The result is a fracture which is most difficult to maintain by closed manipulation. In children, the same fracture is frequently of the green stick variety although complete separation of the bone fragments is not uncommon and the combination of these two in one and the other bone is rather frequent.

The displacement of fragments following fractures of the forearm bones is dependent on muscle action as well as on the force of the injury. More specifically the nature of the deformity depends largely on the location of the break in relation to the pronator teres muscle. This muscle, it may be recalled, has its origin above the elbow medially and inserts near the middle of the radius. When the fracture occurs proximal

to the insertion of this muscle, the upper fragment of the radius assumes a position of supination, because of the unopposed action of the supinator muscle which passes from the ulna around the upper fourth of the radius. The distal fragment is then pronated by the pronator teres and the pronator quadratus. The latter muscle is located anteriorly in the distal fourth and its fibers pass transversely from ulna to radius. In order to re-align this fracture, the distal fragment must be strongly supinated with steady traction throughout the maneuver.

If the fracture is distal to the insertion of the pronator teres, the proximal fragment of the radius assumes a neutral position because the action of the supinator muscle is countered by that of the pronator teres. The distal end of the radius, on the other hand, is pronated by the pronator quadratus muscle and pulled toward the ulna. Reduction is accomplished by obtaining end-to-end alignment of the fragments, with the hand in neutral position between pronation and supination.

In summary when the fracture is proximal to the insertion of the pronator teres muscle, the distal fragment must be maintained in supination during reduction. When the fracture is distal to the insertion of this



Figure 31. Green-stick fractures of the midportion of the radius and ulna. A, Anterior view showing site of fracture but no deformity. B, Lateral roentgenogram showing marked volar angulation. C and D, Final result. Note some persisting angulation in the lateral view. The cosmetic and functional results were good.

sum of the forearm from the elbow to the metacarpal-phalangeal joints of the fingers. Several turns of sheet wadding are placed beneath the plaster and the splint is held in position by gauze. Roentgenograms are made in the anteroposterior and lateral positions and, if the reduction is shown to be satisfactory a circular long arm cast is applied to incorporate the dorsal plaster splint.

The patient should be hospitalized for at least twenty four hours to permit a close check of the circulation in the affected arm. An x-ray examination should be made at the end of ten days and again ten days later to make sure that the alignment has been maintained. Forearm fractures may angulate in the cast when the swelling subsides during the third week and as the child becomes increasingly active. Green-stick fractures will consolidate in six weeks, but displaced fractures of the radius and ulna require eight to ten weeks for sufficient callus to form so that the child, especially young ones, may be given full use of the extremity when the cast is removed.

Open reduction of a fracture of the shaft of the radius or ulna, or both, is rarely indicated in children. It is important, however, to explain to the parents that the persistence of some residual deformity until corrected by growth is preferable to subjecting the child to open reduction.

Transverse Fractures of the Shaft of the Radius and Ulna in Adults. These fractures are commonly accompanied by angulation and overriding. They may be reduced by

direct traction and countertraction or by the method of angulation just described for forearm fractures of children. For traction on the hand with the elbow flexed at 90 degrees Japanese finger traps may be used, and for countertraction a padded sling over the distal end of the arm. As much as 25 to 30 pounds of continuous traction can thus be exerted without the need of help from assistants. The arm is suspended from an overhead pulley by means of the finger traps which permits the operator to use both hands as he attempts to interlock the ends of the fragments to align the bones. With the arm in this position anteroposterior and lateral roentgenograms should be obtained to ascertain if reduction has been satisfactorily accomplished. A circular cast or anterior and posterior molded plaster splints are applied from below the axilla to the metacarpal phalangeal joints if the fragments are reduced. The postoperative care is the same as that for closed reduction of fractures in children. The patient is encouraged to flex and extend the fingers and to exercise the shoulder each day through the full range of motion.

Oblique or Comminuted Fractures of the Radius and Ulna. These fractures must be treated by open reduction or by continuous traction because they have a tendency to become displaced when in the cast (Fig. 35). Fixed traction may be obtained by means of two threaded wires which are inserted, respectively through the midshaft of the metacarpal bone of the thumb and



Figure 34. Incomplete reposition of bone ends but with good alignment is satisfactory treatment of forearm fractures in children, residual deformities correct with growth. A and B Before treatment, C and D following union.

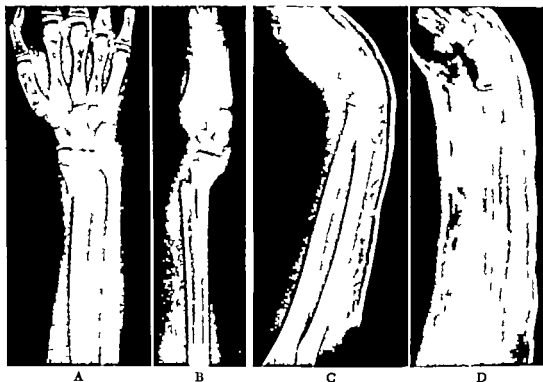


Figure 33. Fracture in the distal fourth of the radius and ulna in a child. A and B, Views showing displacement of radius and angulation of ulna. C, Lateral x-ray film, showing reduction and the dorsal plaster splint. D Lateral view showing good reduction and circular cast which included the splint.

arm is corrected. A piece of wood is placed in the resulting opening in the cast so as to maintain the corrected position. An x ray picture is taken immediately to determine the accuracy of the alignment. After final adjustment of the cast, the plaster is repaired.

Transverse fractures of the radius and ulna in children with complete separation and displacement of the fragments are best reduced by the angulation method rather than by strong traction and countertraction (Fig. 33). Satisfactory end-to-end position is difficult to obtain by traction alone, for the soft tissues become tense and appear to lock the fragments in their displaced positions.

The manipulation is performed with the patient under general anesthesia. If the distal fragment is dorsal to and overlaps the end of the proximal fragment, reduction is accomplished by angulating the distal fragment, wrist and hand into an exaggerated position which will then be considerably behind the plane of the upper segment of bone. At the same time, the proximal end of the distal fragment is pushed distally by the thumbs of the surgeon until it engages the lower end of the proximal one. The lower fragment is now brought into alignment with the upper one. If the degree of

overriding of the displaced fracture is marked, it may be helpful for an assistant to pull downward on the hand while a second assistant provides countertraction by pulling upward on the elbow as the surgeon manipulates the fracture. The reduction is tested by determining the degree of stability or instability of the fracture site. If no false motion is observed, it may warrant the conclusion that the reduction has been successful.

If there is an associated fracture of the ulna, it must also be reduced. Green-stick fractures should be corrected before manipulation of the radius. If the ulnar injury is a fracture-displacement, it should be manipulated by angulation in a manner similar to that described for the radius. Occasionally the reduced radius can serve as a fulcrum for manipulation of the ulna. End-to-end reposition of the radius is essential but is less exacting with the ulna. Anatomic reduction is not required of the radius and ulna in children as long as the alignment is satisfactory since growth will overcome imperfect end-to-end position (Fig. 34). In adults, however these fractures require the most accurate reduction if satisfactory results are to be obtained.

Following manipulation, a posterior molded plaster splint is applied to the dor-

artery or the superficial branch of the radial nerve. The ulna is exposed by a dorsal incision over its crest. No important structures will be encountered if the dissection is kept subperiosteally. For internal fixation, many surgeons are still using metal plates and screws, but the intramedullary rod is preferable because in addition to providing firm fixation, it often obviates the necessity for the additional protection of a plaster cast (Fig. 36). The intramedullary rod is inserted into the shaft of the radius by the direct method. For this purpose, the distal end of the bone is exposed dorsal and lateral to the radial styloid process. The rod is introduced through a hole drilled in the cortex and driven upward through the medullary canal to the fracture site and then continued into the proximal fragment after the fracture is reduced under direct vision. The length of the rod is determined by measuring the radius in the other arm.

Insertion of the rod into the ulna is performed by the retrograde technique. After exposure of the site of the fracture the rod is passed proximally into the medullary canal of the upper fragment through the end of the olecranon process and a small opening in the overlying soft tissues until the rod is flush with the distal end of the upper fragment. The fracture then is re-

duced and the rod is driven into the canal of the distal fragment. The end of the rod projects slightly beyond the olecranon but is buried under the soft tissue to minimize the possibility of infection. Bone grafts should be used for the added stimulus they give to callus formation when fractures four or more weeks old are subjected to open reduction. Finally a long arm cast should be applied for about one month if the fracture remains unstable in spite of the intramedullary rod. Complete union may not be achieved for four months or more but the patient may be able to resume suitable work soon after the cast is removed.

Fractures of the Shaft of the Radius. Either one of the two forearm bones may be broken. Fractures of the shaft of the radius alone are rare, however for this part of the bone is dense and compact. These fractures are usually the result of direct violence. In children they are very common and are most often of the green stick type. In adults, the fracture is as a rule, oblique or comminuted and often somewhat displaced toward the ulna. If the fracture is accompanied by overriding and shortening of the radius, it will be necessary to obtain roentgenograms of the entire forearm to confirm the presence of either an associated fracture or a dislocation of the ulna.



FIGURE 36. An unstable oblique fracture of the middle third of the radius and ulna in an adult treated by open reduction and intramedullary rods after unsuccessful closed manipulation. A, Before treatment. B, Soon after open reduction. C and D, State of healing after four months.



Figure 35 Oblique and comminuted fractures of the radius and ulna in an adult. A and B, Views before treatment. C, Reduction obtained by traction. D and E, Alignment soon after application of long arm cast with skeletal fixation. F Redisplacement of ulna after ten days, making open reduction necessary.

through the proximal end of the ulna. These wires then are incorporated in a long arm cast after the fracture has been manipulated. If in spite of accurate reduction, the radius or the ulna becomes displaced or angulated in the cast as the swelling subsides, then open reduction must be attempted. Many surgeons recommend recourse to operative reduction as the acceptable treatment for

all unstable fractures of the shaft of the radius and ulna in adults.

Open reduction must not be attempted by exposing both of these bones through one incision. It is moreover a difficult operation which should be performed only by an experienced surgeon. The shaft of the radius is exposed through an appropriate incision. Care must be taken not to injure the radial



Figure 37 Comminuted fracture of radius treated by manipulation and cast (A and B) Double pin fixation maintained alignment, although anterior part of cast was removed for skin grafting of extensive burn of forearm. Final result of fracture was excellent (C and D)

Closed reduction of fractures of the radius displaced ulnarward is very difficult. It may at least be attempted, however by lifting the radius manually away from the ulna and by using Japanese finger traps to reduce the fracture by strong traction, countertraction at the elbow and angulation of the bone fragments so that their ends may be locked. It remains, however that these fractures have a tendency to redisplace or angulate in the cast, thus making it necessary to check the reduction at frequent intervals with roentgenograms. If displacement occurs the cast must be removed and recourse must be had to continuous traction utilizing a thread wire through the metacarpal bone of the thumb and a second pin inserted across the proximal end of the ulna. These are incorporated in a long arm cast (Fig. 37) Open reduction followed by internal fixation with an intramedullary rod may be an alternative method of treatment. The result should prove completely satisfactory. Union of the fragments usually requires eight to twelve weeks. It is not exceptional to apply a bone graft at the time of surgery because of the high incidence of nonunion.

Fractures of the Shaft of the Ulna. These fractures are most often due to direct violence and occur usually in the lower third of the bone. They may be transverse oblique or comminuted, except in children, in whom they are usually of the green-stick variety. Closed reduction and immobilization in a long arm cast with the elbow in 90 degrees of flexion may be attempted however there is a tendency for the ulna to angulate toward the radius during immobilization in the cast. Fractures, therefore, which either cannot be reduced by manipulation or cannot be maintained after closed reduction must be treated by surgery preferably followed by fixation with an intramedullary rod. The rod is inserted by the retrograde technique. It may make a plaster cast unnecessary. A heavy Steinmann pin or a Rush rod may be used. Union occurs in about ten weeks. It need hardly be emphasized that malunited fractures of the ulna with bowing of the forearm are most disabling and must be avoided.

Fracture of the Shaft of the Ulna and Dislocation of the Head of the Radius (Monteggia Fracture) This is a fracture in the shaft of the ulna accompanied by either an anterior posterior or lateral displacement of the upper end of the radius. It is usually due to direct violence applied to the ulna. The injury is important because the disloca-

tion of the radius is often overlooked and only the obvious fracture of the ulna receives treatment. The inevitable result is a deformity with restriction of motion and disability. The accident is common in children, in whom closed reduction is usually satisfactory and a good position can be maintained with a plaster cast. In adults this is not the case and the need for perfect reposition of the ulna and radius often requires open reduction. The shaft of the ulna may fracture at any level from the lower third to just below the olecranon process.

There are in general two types of deformity: an anterior angulation of the ulnar fragments associated with anterior dislocation of the radial head, and posterior angulation of the ulnar fragments associated with posterior dislocation of the radial head. The fracture may be transverse, oblique or comminuted or of the green-stick variety in children. The commonest angulation of the ulna is with the apex of the angle pointing forward and with anterior dislocation of the radial head. The annular ligament is always torn as the radial head dislocates.

On physical examination, there is often a depression over the posterior aspect of the shaft of the ulna at the site of the fracture of the anterior type. Palpation of the radial head reveals this not to be in its normal position. The patient usually holds his elbow in partial flexion and in approximately mid-rotation. Examination for posterior interosseous nerve palsy should be made, since this is found in about one-fourth of the cases. Roentgenographic examination which shows the angulated fragments of the ulna only and does not include the elbow joint in lateral projection, is inadequate to rule out dislocation of the radial head. It is very easy to determine on a lateral view of the elbow whether the radial head is dislocated or not. A line drawn through the long axis of the radius and projected upward past the humerus normally passes through the center of the capitellum. If it does not, there is dislocation of the radial head.

Treatment of Monteggia injuries may be either conservative or surgical, depending upon the nature of the fracture and the reducibility of the radius. Many of these fractures can be satisfactorily treated by closed methods, for example, transverse fractures. Closed reduction of the ulna is accomplished by traction followed by manipulation of the head of the radius. Immobilization is obtained by means of a plaster cast extending from the metacarpals to the axilla.

is not, for it will delay recovery and lead to permanent loss of motion. The prognosis should always be guarded in patients in whom the head of the radius is excised for recovery of a painless joint with full flexion and extension capacity is difficult to accomplish.

In children fractures of the neck of the radius with severe lateral angulation or displacement of the head fragment require special treatment. In adults, these conditions should be treated by excision of the head of the radius; in children, the head must be replaced accurately by manipulation or surgery. Excision of the head will permit an undesirable upward displacement of the remaining portion of the radius during subsequent growth, with resulting subluxation of the distal radioulnar joint, accompanied by pain and marked disability of the wrist joint.

Fractures of the Olecranon Process. This injury occurs almost exclusively in adults. It usually follows a fall on the elbow resulting in a transverse, oblique or more often a comminuted fracture and occasionally with anterior dislocation of the elbow (Fig. 38). It may also occur from a violent pull of the triceps muscle, for its tendon is attached

to the tip of the olecranon process thus producing a true avulsion fracture. In the majority of subjects, there is some, and often considerable separation of the fragments.

The diagnosis is suggested by the history and the pain and swelling over the olecranon process. It is confirmed if a gap can be felt between the fragments or if the normal relationship between the process and epicondyles of the humerus is disturbed. Roentgenograms will reveal the exact nature of the fracture.

Fractures of the olecranon without separation of fragments should be immobilized in a long arm cast with the elbow at an angle of between 90 and 135 degrees and with the forearm in midpronation. Roentgenograms should be made at the end of the first and the second week to determine whether the smaller fragment has not been displaced by the triceps muscle. If no displacement has occurred, the cast can be discarded at the end of five weeks to permit gradual resumption of elbow motion.

Some fractures of the olecranon with separation of fragments may be satisfactorily reduced by complete extension of the elbow and can be maintained by the application

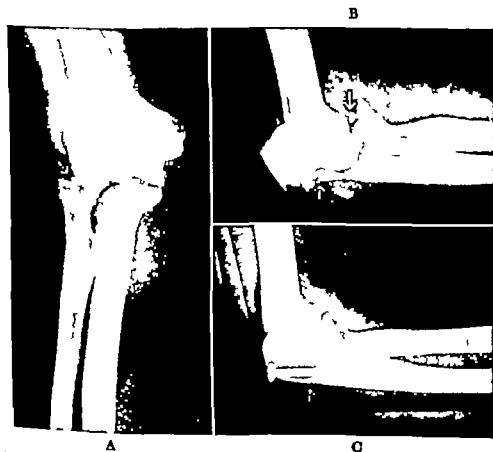


Figure 38. Anterior dislocation of the elbow and associated fractures of the olecranon process and head of the radius. A and B, Anteroposterior and lateral aspects before treatment. C, Lateral view following open reduction of dislocation, internal fixation of olecranon and excision of radial head.

In patients having anterior dislocation, the elbow should be put up in 90 degrees of flexion, while in those with posterior dislocation the elbow should be placed in incomplete extension (135 degrees or more). Frequent roentgenographic check ups must be made in the following two weeks to determine whether the ulnar fragments and the radial head are maintaining their reduced position. If the deformity recurs or the initial treatment is not satisfactory then open reduction must be performed.

Surgery is accomplished by exposure of the fracture site and the radial head through two separate incisions, except in the proximal third of the forearm where both may be made through a posterior incision, as described by Speed and Boyd. The fracture of the ulna is reduced and fixed by means of an intramedullary rod, following which the radial head is replaced and the annular ligament is repaired. A plaster cast is applied for six weeks to permit healing of the ligament, after which motion is resumed. At the same time, the intramedullary rod will maintain alignment of the ulna until final consolidation of the fracture has been achieved. If on the other hand, internal fixation has not been resorted to for treatment of the ulna it will be necessary to continue the use of the cast until roentgenograms confirm union of the fracture. This requires approximately ten to twelve weeks.

Neglected Monteggia fractures with disabling symptoms must be treated by surgery. In these patients, the fracture of the ulna is reduced, an intramedullary rod is utilized for fixation and bone grafts are placed about the fractures to stimulate union. The head of the radius is reduced and the annular ligament repaired with a strip of adjacent triceps tendon or of fascia lata. If this is not possible the radial head is resected. In some instances, satisfactory function of the elbow and forearm is present in spite of persistent dislocation of the upper end of the radius.

The prognosis of patients having Monteggia fractures is favorable provided the acute injuries are immediately recognized and treated. It is difficult to restore function of the radius in neglected cases although satisfactory union and alignment of the ulna may be obtained.

THE ELBOW

Fractures of the Head and Neck of the Radius. Anatomically the head and neck of the radius lie completely within the capsule

of the elbow joint. Clinically the importance of these fractures varies with the degree of their interference with the function of articulation between the radial head and the capitellum or between the radius and the ulna. Treatment must accordingly be adjusted to the extent of functional disability which may be expected in the elbow and radial-ulnar joints.

Fractures of the head and neck of the radius are extremely common in adults but less so in children. They usually result from a blow directed upward through the forearm, forcing the radial head to strike against the distal end of the humerus and producing one of the following main types of fracture: fissure, marginal, comminuted fractures of the head and fractures of the neck of the radius with impaction or displacement of the head.

The symptoms are usually characteristic. The patient complains of pain and swelling of the elbow local tenderness over the head of the radius and loss of the movements of pronation and supination, depending upon the degree of deformity of the head. An accurate diagnosis must make use of roentgenograms.

Linear cracks across the radial head or impaction of the head into the neck without significantly altering the position of the articular surface is not an important injury and may be treated adequately by putting the arm in a sling and early resumption of motion. On the other hand, fractures with marked comminution and separation of the fragments or with displacement of one or more fragments into the elbow joint should be treated by complete excision. The same treatment should be applied to impacted fractures in which the head of the radius is tilted to such a degree as to cause it to impinge upon the adjacent articular surfaces of the ulna and humerus.

Excision of the head of the radius is effected through an incision between the anconeus and the extensor carpi ulnaris muscles. The capsule of the elbow joint is exposed and the radial head is laid bare by an appropriate incision through it. The line of resection should be just proximal to the annular ligament. All loose fragments must be removed from the elbow joint. Trauma must be kept at a minimum, for otherwise considerable ossification will occur in the capsule and produce stiffness of the joint.

Elbow motion is permitted as soon as the wound is healed. Active exercise may be helpful, but passive stretch the elbow

wire suture. A cortical bone graft is applied on the ulnar side of the fracture while cancellous grafts are placed on the other side. The proximal end of the radius must not be exposed in the wound for otherwise cross-union and synostosis may result. Motion of the elbow is resumed after the wound has healed and internal fixation has been adequately procured. Union occurs in ten to twelve weeks with results which are as a rule, highly satisfactory.

Dislocation of the Elbow Joint. This injury is particularly frequent in children and adolescents, because of the undeveloped state of the coronoid process. The dislocation is most often in the posterior direction (Fig. 40) and usually is the result of a fall on the hand in which the forearm is forced backward while the elbow is flexed. The appearance of the arm, if not obscured by

swelling, is typical. The patient holds the forearm in incomplete extension. The elbow appears broadened from behind and the forearm is shortened. The olecranon may be palpated posterior to the medial and lateral epicondyles of the humerus. Normally the two humeral epicondyles and the tip of the olecranon process form an equilateral triangle when the elbow is flexed to a right angle and the three landmarks are at the same level when the elbow is in extension (Fig. 41). Crepitus is absent unless there is an associated fracture. Marked lateral (or medial) displacement is rare because of the strong collateral ligaments and the broad articular surface of the distal end of the humerus.

Reduction should always be preceded by a careful roentgenographic examination to rule out the possible presence of other in-

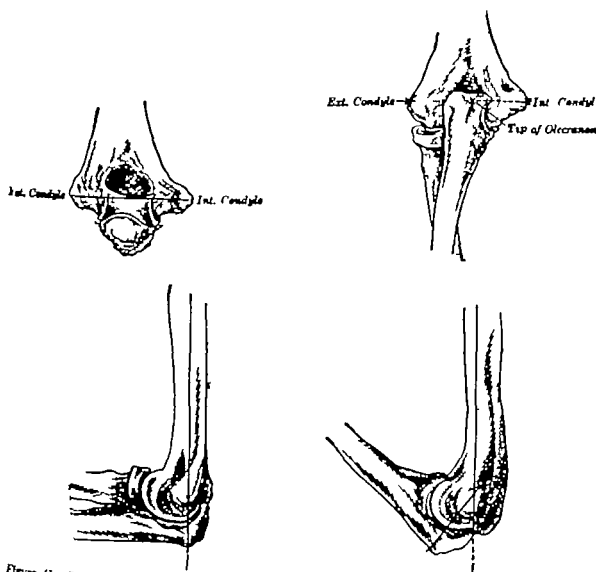


Figure 41. Drawings illustrating the relationship between the external and internal epicondyles of the humerus and the olecranon process when the elbow is in flexion and extension. (Ellison: Fractures of the Humerus, Radius and Ulna. D Appleton-Century Company Publishers.)

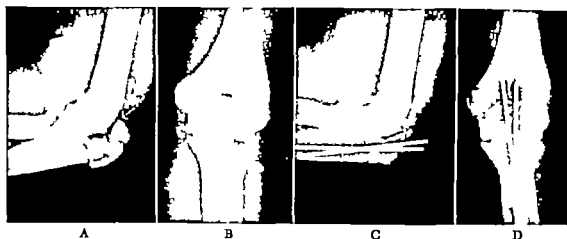


Figure 39 Fracture of the olecranon with separation of the fragments. Note the steplike defect of the articular surface of the olecranon which was corrected by open reduction and internal fixation using two threaded wires. A and B, Before surgery C and D Following union. Patient recovered full flexion and 170 degrees of extension.



Figure 40. Roentgenograms showing a posterior and lateral dislocation of the elbow joint.

of a cast for six weeks. But the position of the elbow is discomforting to the patient and the development of edema may pose a real problem. Another difficulty is in mobilizing the elbow after it has been in extension for six weeks. Manipulation with the patient under general anesthesia is frequently necessary.

By contrast, open reduction with adequate internal fixation, by use of multiple threaded wires, a long wood screw or wire suture, makes active motion of the elbow immediately possible and, as a rule, justifies the expectation of regaining a painless elbow with full flexion and extension.

Avulsed fragments of not more than one-third the length of the olecranon process are best treated by excision of the fragment and suture of the triceps tendon to the raw bone at the proximal end of the ulna. Motion can be resumed at the end of two weeks with the likelihood that full extension power of the elbow will be regained.

Comminuted fractures with separation should be re-aligned accurately by open reduction and fixed internally to maintain their normal relationship with the rest of the ulna (Fig. 39). Accurate restoration of the articular surface of the olecranon, even to the extent of replacing small fragments, is a requisite if arthritis due to incongruous joint surfaces is to be avoided.

Ununited fractures of the olecranon process are productive of pain and weakness of extension motion of the elbow and are best treated by open reduction and a bone graft. For this, a linear incision is made over the subcutaneous border of the olecranon process, beginning proximal to its tip and extending downward for about 3 inches. The bone is exposed subperiosteally. The ulnar nerve is left alone and preserved from injury. The fracture surfaces are freshened and reduced accurately if they are not in adequate alignment. Fixation is obtained with multiple threaded wires, screws.

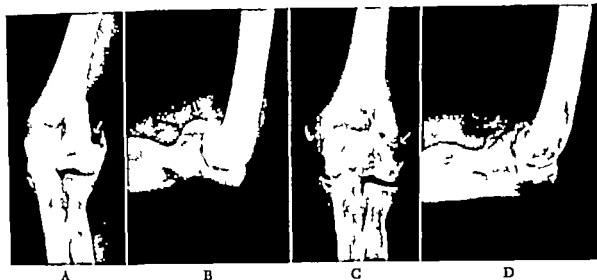


Figure 43. Fracture of the capitellum due to a force transmitted upward through the radius. A and B Note comminuted fracture with one large fragment displaced anteriorly. C and D Condition after manipulation and application of arm cast with elbow in extension for three weeks.

wrist and shoulder frequently. Six months more or less, may be required to regain maximum motion.

The prognosis should always be guarded, because it is not infrequent that deposition of bone in the collateral ligaments entails some permanent limitation of motion especially in extension. Old or neglected dislocations of the elbow must be treated by open reduction. Recurrent posterior dislocations may be stabilized by a bone graft which is inserted through a hole drilled from the posterior surface of the ulna. The end of the graft is made to project beyond the coronoid process to buttress the joint.

Fractures of the Capitellum of the Humerus. These fractures may be produced by a forceful impact of the head of the radius on the opposing articular surface of the humerus (capitellum) resulting in the breaking off of either a single fragment or multiple fragments of cartilage and bone (Fig. 43).

The immediate symptoms include swelling of the elbow due to hemorrhage accompanied by pain and limitation of motion of the elbow and forearm. Stereoscopic roentgenograms taken in the anteroposterior and lateral directions will disclose the location of the fragments.

Fractures which involve either a single or at most, two large segments of the capitellum should be reduced by open surgery and the articular surfaces restored as accurately as possible. Comminuted fractures which cannot be reassembled require excision of the fragments. Removal of the major portion of the capitellum, including the adjacent condylar bone, will result in lateral instabil-

ity of the elbow, permanent valgus deformity and functional disability.

Closed reduction of a large fragment with upward displacement, but without rotation, may be attempted in selected cases. This can be done with the patient either under local or general anesthesia. With the elbow in extension and the forearm adducted, pressure is exerted against the fragment to push it back in its normal position. Following this the elbow is placed in 90 degrees of flexion in a posterior molded splint. Roentgenograms are made immediately. If the reduction is not successful the splint must be removed and manipulation repeated. A posterior splint then is applied with the elbow in complete extension and roentgenograms are again made. If the reduction is now satisfactory the elbow is immobilized in extension for a period of three weeks, following which it is gradually brought into a flexed position in the course of the following two weeks.

Open reduction is performed through a lateral oblique incision between the anconeus and the extensor carpi ulnaris muscles. The incision is extended proximally along the epicondylar ridge. The anconeus is retracted posteriorly and the extensor muscles of the forearm anteriorly. The tendon of the anconeus is severed proximally and the soft tissues are stripped from the epicondylar ridge to gain access to the joint and to the adjacent posterior aspect of the humerus. The fracture then is reduced accurately and held in position by two screws which are inserted through the intact posterior portion of the condyle and into the fracture fragment. The screws must not protrude to the articular surface of the capitellum. Fol-



Figures 42. Posterior dislocation of the elbow with an associated comminuted fracture of the head of the radius. A and B Views showing the dislocation and fracture. C, Lateral roentgenogram showing reduction of dislocation but persistent displacement of head of radius. D and E, Final result after resection of radial head. Note ossification in capsule in E.

juries, such as a fracture of the head of the radius or the medial epicondyle of the humerus (Fig. 42). A general anesthetic should ordinarily be administered, although it may on rare occasion be necessary to attempt reduction by skillful manipulation following an adequate dose of a sedative.

Care must be exercised while effecting the reduction to avoid additional trauma. Downward traction is applied to the forearm while an assistant holds the upper arm. The medial or lateral displacement is corrected and then the forearm is extended to disengage the coronoid process from the back of the humerus. Further gentle downward traction on the forearm will bring the articular surface of the radius and ulna to the level of the distal end of the humerus, following which the elbow is flexed to effect final reduction. A posterior molded plaster

splint is then applied, with the elbow joint in 90 degrees or more of flexion. The flexion, as well as the swelling which ensues, must not be permitted to obliterate the radial pulse. Roentgenograms must be obtained before the anesthesia has subsided to determine the success of the reduction. If it is shown to be unsuccessful, the splint must be removed and manipulation repeated until a satisfactory result is obtained.

The adequacy of the circulation in the arm should be checked carefully for thirty-six to forty-eight hours and roentgenograms should be obtained again at the end of ten days and after two weeks. Active flexion motion may be resumed fourteen days from the time of injury when the arm is transferred from the cast to a sling. The patient is urged to close and open the hand vigorously immediately after reduction and to move the

ment. The fragment maintains a good blood supply through the soft tissues attached to it which facilitates rapid union. Aseptic necrosis of the avulsed portion of the humerus rarely occurs.

A posterior molded plaster splint is applied for four weeks, after which the patient is permitted active use of the elbow. The prognosis for recovery of full motion and maintenance of normal longitudinal growth should be guarded.

Adults are occasionally seen with untreated fractures of the lateral humeral condyle which had occurred in childhood. The function of the elbow joint is usually superior to that which the appearance of the roentgenograms would suggest and certainly is better than in those patients in whom the fragment was excised.

An increasing valgus deformity of the elbow occurs in children when the fracture is neglected or the distal lateral humeral epiphysis undergoes premature closure while growth of the trochlear epiphysis continues. This produces stretching of the ulnar nerve with partial or complete paralysis of those muscles of the hand supplied by it. The angular deformity of the bone can be corrected by supracondylar osteotomy. Transplantation of the ulnar nerve to the anterior surface of the elbow before irreparable damage to the nerve has occurred offers a good prognosis for recovery of function.

Fracture of the Medial Epicondyle of the

Humerus. The medial epicondyle epiphysis of the humerus may be avulsed (Fig. 45) and rotated downward by the pull of the flexor muscles of the wrist and fingers which are attached to it. The displacement may amount to a few millimeters in some patients and requires for treatment only a protective circular cast or a posterior plaster splint applied for four weeks. In others, the fragment may be significantly displaced downward along the side of the medial condyle of the humerus and in still others it may become incarcerated in the elbow when the joint is wedged open by a continuation of the force of the injury. This may likewise occur when the fragment fails to leave the joint as an associated dislocation is reduced (Fig. 46).

Open reduction of the epicondyle is indicated in all patients in whom there is a significant degree of displacement of the fragment. In this approach, a linear incision is centered over the medial side of the elbow. The deep fascia is opened and the ulnar nerve, which is stretched excessively at times by the injury is identified and protected from further damage. The avulsed fragment is then isolated outside or inside the joint, replaced in its normal position and fixed with two threaded wires. In some selected cases, the ulnar nerve is transplanted to the anterior aspect of the elbow during closure of the wound. A posterior molded splint is applied for three weeks and motion is re-

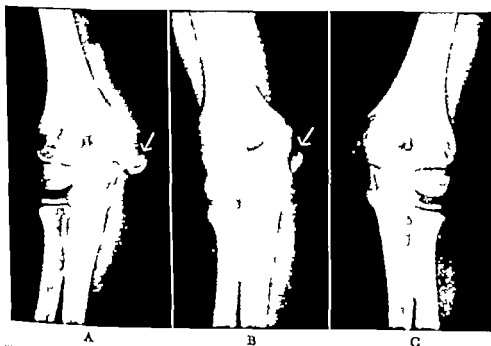


Figure 45. Avulsion fracture of the medial epicondyle of the humerus in a child with slight displacement treated by a long arm cast with the elbow in 90 degrees of flexion. A, Anteroposterior x ray film showing original condition. B, View of final result. C, Normal arm for comparative purposes.

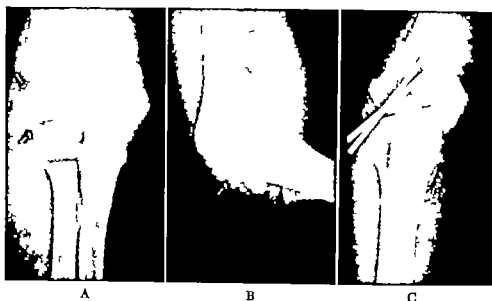


Figure 44 Fracture of the lateral condyle in a child. Wide displacement and rotation of the lateral condyle fragment is due to the attached extensor muscles of forearm. A and B, Anteroposterior and oblique views of original fracture. C, Accurate reposition by open reduction and fixation with two threaded wires.

lowing closure of the wound, the elbow is flexed to 90 degrees and immobilized in a posterior molded plaster splint for about three weeks. Full flexion and extension of the elbow joint may be recovered if the reduction has been performed accurately. In many instances, however, this goal is not attainable and some permanent decrease in the range of motion will persist.

Fractures of the Lateral Condyle of the Humerus in Children. This relatively rare fracture is produced by a valgus injury of the elbow. A comparable fracture is not seen in adults. The fracture extends from the lateral cortex of the humerus just above the epiphyseal plate obliquely downward across the growth cartilage plate and lateral condyle to emerge at the joint surface near the trochlea. The fragment may be pulled laterally and backward out of the joint by contraction of the attached extensor muscles. In some cases, the fragment is also rotated outward and forward through an arc of 90 degrees, thus causing its articular surface to face proximally and medially rather than distally.

Swelling and ecchymosis are confined more or less to the lateral half of the elbow joint. The articulation appears broader than normal and the abnormal relationship between the olecranon process and the epicondyles of the humerus at the lateral side of the joint should suggest the nature of the fracture. Roentgenograms made in the anteroposterior, lateral and oblique views will

show the details of the fracture and establish the diagnosis.

Closed reduction of this fracture is usually unsuccessful, although McLearn and Mereson have reported two successful cases. They believe that the elbow is dislocated posteriorly and laterally by the injury and carries the fractured lateral condyle fragment with it. The elbow dislocation, they assume, reduces spontaneously but leaves the fragment in its dislocated position. Therefore they redislocate the elbow laterally and the condyle fragment resumes its normal position as the dislocation is again reduced.

Surgery is indicated for those patients in whom the reduction cannot be accomplished manually (Fig. 44). The fragment and the elbow joint are exposed through an oblique incision between the anconeus and the extensor carpi ulnaris muscles. The distal, lateral aspect of the adjacent humerus is exposed subperiosteally as the incision is extended upward along the epicondylar ridge. All blood clots are removed from the joint and the fragment is rotated into its normal position where it is either sutured to the surrounding soft tissues or fixed to the humerus with two threaded wires. Accurate reduction must be maintained to prevent a bridge of bone from forming across the cartilaginous epiphyseal plate with resulting arrest of longitudinal growth. It is less dangerous to use two small-caliber threaded wires to secure accurate internal fixation than to permit the fragments to unite with displace-

diately of the accuracy of the reduction. If reduction is unsatisfactory the cast must be removed at once and the manipulation repeated until normal repositioning is obtained. If this cannot be achieved then continuous skeletal traction is necessary. Hospitalization after manipulative reduction is desirable for immediate detection of possible circulatory embarrassment.

If, as has been stated, the distal fragment cannot be held in position in the cast or if the swelling around the elbow is excessive and prevents immediate and adequate reduction, skeletal traction through the crest of the ulna may be used. An ordinary wood or eye screw is inserted into the olecranon just distal to the elbow joint. The injured arm is then extended over the side of the bed with the elbow at an angle of 90 degrees and 5 pounds of traction is applied to the screw attached to the olecranon process. The forearm is at the same time suspended overhead by means of 3 pounds of skin traction. A well-padded sling, to which a weight of 1 pound is attached, is placed over the lower end of the humerus to pull it backward to approximate the small condylar fragment as it comes forward. Medial or lateral displacement of the condyles is corrected by manual pressure if they do not assume a satisfactory position by traction. Pronation of the forearm may correct a medial shift of the small fragment.

Roentgenograms should be obtained at appropriate intervals during the first week to check the progress of the reduction. Sufficient callus will have developed in twelve to fourteen days to permit application of a

posterior molded splint with the elbow in as much flexion as can be tolerated. The patient then is discharged from the hospital.

Follow-up care is the same for both the manipulative and the continuous traction methods of reduction. Roentgenograms should be repeated at seven-day intervals on two or three occasions to check for possible rotation or posterior displacement of the distal fragment. The fracture may be re-manipulated at any time before much callus has formed if a change in alignment points to an unfavorable end result. Bony union should be sufficiently advanced in six to eight weeks to permit removal of the cast. Recovery of full motion may require several months. The elbow must not be stretched, for this will delay full recovery and may even lead to permanent fibrosis with limitation of joint extension.

United fractures with loss of the normal carrying angle are unlikely to correct with subsequent longitudinal growth of the humerus. In these patients it may be necessary to perform transverse supracondylar osteotomy to obtain proper functioning of the extremity. The procedure is carried out through a lateral epicondylar incision. The supracondylar portion of the humerus is exposed subperiosteally all around its circumference. A wedge of bone is then removed so that the distal fragment can be angulated outward into a corrected position. Six weeks are required for union.

It is of the utmost importance to observe the circulation in the fingers at frequent intervals for forty-eight to seventy-two hours after reduction, for this injury is most prone

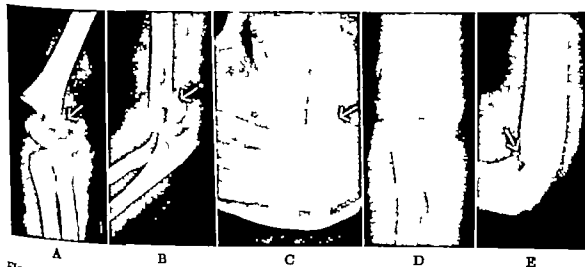


Figure 47. Roentgenograms of a typical supracondylar fracture of the humerus in a small child. A and B. Note the posterior and radial displacement of the distal fragment. C, Reduced fracture in a cast. D and E, Final result. Full function of the arm was recovered.

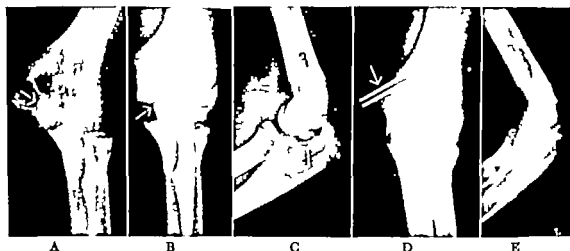


Figure 46 Fracture of the medial epicondyle of the humerus with posterior and lateral dislocation of the elbow joint. The medial epicondyle became incarcerated in the joint when the dislocation was reduced. A and B Prior to treatment. C, Reduction of dislocation with fragment in joint. D and E, Final result.

sumed. The pins are removed at a convenient time. Motion of the elbow joint should be fully recovered unless there is associated dislocation of the joint with fibrosis of the collateral ligaments and capsule.

Supracondylar Fractures of the Humerus in Children. These fractures (Fig. 47) occur through the thin transverse plate of bone between the condyles and the shaft of the humerus. They result from a fall on the outstretched hand with the elbow in varying degrees of flexion leading to anterior or posterior displacement of the condylar fragment as related to the shaft of the humerus. The posterior or extension type is most common because the fracturing force is usually upward and backward rather than upward and forward.

The diagnosis is based on physical examination and roentgen ray findings. There is diffuse swelling of the elbow with obvious shortening of the arm. The relationship between the epicondyles of the humerus and the olecranon process remains normal although they lie posterior to the shaft of the humerus, in contrast to their relationships in posterior dislocations of the elbow. In avulsion fractures of the epicondyles and in fractures of the olecranon process with upward displacement. The olecranon process can be palpated posteriorly to the condyles of the humerus in dislocations, whereas one epicondyle will be asymmetrically placed in fractures which involve it or one condyle. A careful neurologic examination is essential, because the radial, ulnar or medial nerves often show paralysis from which recovery is spontaneous in three or four months.

The circulatory status of the forearm and hand must also be studied prior to the re-

duction. Pressure upon or spasm of the brachial artery may present a serious therapeutic problem before or after reduction of the fracture, which may predispose to the development of a Volkmann's contracture.

Roentgenograms will disclose the direction of the fracture line and the degree of displacement of the distal fragment.

Some controversy exists as to which is the best method for the reduction of supracondylar fractures of the humerus, that is, whether reduction should be undertaken immediately after injury and the cast applied, or whether it is safer to apply continuous skeletal traction and reduce the fracture gradually.

Successive objectives in the manipulation of supracondylar fractures are correction of the proximal displacement of the condyles and of the medial and lateral shift, following which the distal fragment is brought forward to engage the end of the shaft of the humerus.

In patients with complete backward displacement with overlapping of the fragments, general anesthesia is necessary. Gentle downward traction is exerted on the forearm, while the distal fragment is aligned with the proximal one by the operator. After the condyles have cleared the end of the shaft, they are lifted forward by one hand, while the surgeon's other hand places the patient's forearm in moderate flexion. Reduction is maintained by the tension exerted by the triceps muscle attached to the olecranon process. The fracture is immobilized in a posterior molded plaster splint, with the elbow flexed at the maximum degree, which will leave the radial pulse intact. Roentgenographic checks should be made im-

by marked swelling of the elbow region and discoloration of the skin due to hemorrhage. The radial and ulnar nerves frequently become contused sufficiently to produce temporary paralysis. It is therefore important to check the condition of these nerves before proceeding with the reduction. If the surgical approach is contemplated the extremity should be immobilized for ten to fourteen days in a posterior metal or plaster splint with the elbow in 90 degrees of flexion and a large pressure dressing on the arm to give the swelling a chance to subside. If closed

reduction is to be tried skeletal traction can be instituted immediately after the accident.

For open reduction of comminuted fractures of the lower end of the humerus (Fig. 49) a posterior linear incision is made over the proximal end of the olecranon process and extended upward on the back of the arm for several inches. The olecranon process is then cut about 1 inch distal to its tip and the proximal segment is separated from the triceps tendon and removed. When the elbow is flexed an excellent exposure of

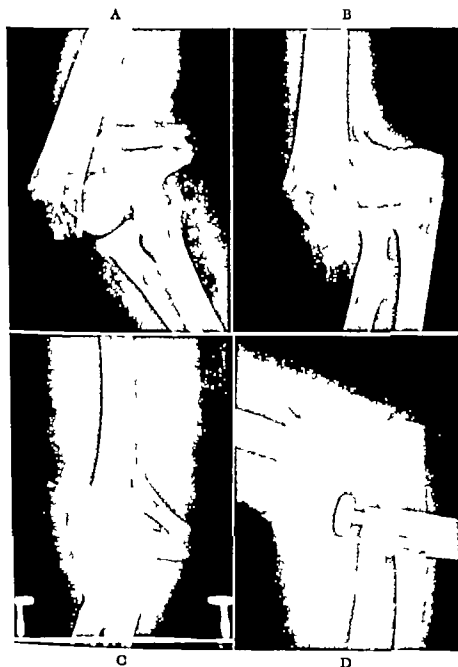


Figure 48. A serious comminuted fracture of the distal end of the humerus in a sixty-seven-year-old patient. Treatment by skeletal traction, using a threaded wire through the ulna, was unsuccessful. Open reduction was performed with recovery of full motion. A and B, Original fracture. C and D Unsatisfactory reduction by traction.

to be followed by *Volkman's ischemic contracture*. Immediate treatment is necessary in all patients and especially if poor circulation is due to spasm of the brachial artery which follows initial trauma or is induced by manipulation.

The earliest signs are those of obstruction to the arterial circulation, with pallor, absence of the distal pulse and lowered skin temperature, terminating in paralysis and contracture. When this condition has fully developed, the hand is so useless that little if any amelioration is possible. Therefore the importance of carefully observing the condition of the circulation cannot be too strongly emphasized.

At the first sign of an impending *Volkman's contracture* all dressings must be loosened down to the skin and, if necessary, the cast removed. The elbow should be extended sufficiently to permit recovery of the radial pulse. In some patients it may be necessary to allow the fracture to redisplace temporarily in order to restore adequate circulation. A superior cervical sympathetic ganglion block should be performed and, if successful, may be repeated at appropriate intervals to maintain an optimum degree of vasodilatation and blood flow.

If the condition remains refractory to these measures the surgeon must quickly proceed with an exploration of the brachial artery followed by correction of any abnormality that may be discovered. At the same time, the fascia of the forearm should be split longitudinally to relieve all pressure on muscles, blood vessels and nerves.

THE HUMERUS

Fractures of the Distal End of the Humerus in Adults. Some of the most difficult fracture problems are encountered in this region. There are four types: T fractures, transcondylar fractures of the medial or lateral condyles and comminuted fractures with wide displacement and rotation of the fragments. They may occur in the young, but are most frequent and common in older adults.

A T fracture is caused by a fall directly upon the elbow and consists of a transverse break across the supracondylar region of the humerus with an extension downward between the two condyles into the elbow joint. In the milder forms of displacement or deformity the fracture can be treated satisfactorily by means of a hanging cast left in place for four weeks. The arm is then carried

in a sling and a satisfactory range of motion is gradually recovered. Transcondylar fractures are treated similarly.

Displaced T fractures of the humerus require continuous traction for reduction. This can be obtained with the patient lying supine in bed by means of skeletal traction with a pin inserted through the proximal end of the ulna. Traction can also be carried out while the patient is ambulatory. The arm is then supported on an abduction splint to which the skeletal traction is attached. T fractures which do not adequately reduce by closed methods should be treated by open reduction and internal fixation.

Fractures of the medial or the lateral condyle of the humerus are infrequent. They result from shearing forces which produce a fracture which begins at the joint surface and extends upward to emerge at the outer aspect of the medial or lateral cortex of the humerus. The fracture can frequently be reduced by strong traction, although in many cases open reduction and internal fixation are necessary to restore alignment of the articular surface.

Comminuted fractures of the distal end of the humerus with wide displacement and rotation of fragments tax the ingenuity and skill of the surgeon to the utmost. The fracture frequently is of the comminuted variety and may include fragments of bone from the medial epicondylar ridge, the trochlea and medial condyle, varying portions of the capitellum and the lateral condyle and also pieces of the contiguous bone. Some of these fractures look hopeless in the roentgenograms but reduce quite accurately when skeletal traction is applied through the crest of the ulna with the elbow placed in 90 degrees of flexion. Recourse to open reduction may be advisable, however if the articular surface of the humerus is not satisfactorily restored by skeletal traction (Fig. 48). Highly fragmented fractures of the articular surface are almost impossible to reduce accurately even by surgery so that it may be wise to accept the best position which can be obtained by skeletal traction. On the other hand, when the fragments are large and not more than three or four in number open reduction offers a much better prognosis than an inaccurate closed one. Motion can be permitted three weeks after surgery if the fragments are rigidly secured by internal fixation with threaded wires, screws, bolts or plates.

Comminuted fractures are characterized

depends more upon the direction of the fracturing force than upon muscle action. Spasm of the biceps and triceps muscles pulls the distal fragment proximally and may produce shortening, but neither the distal nor proximal fragment is deviated significantly from the midline by muscle action.

The physical signs of a shaft fracture of the humerus consist of swelling, deformity, crepitation, false motion and, in some cases, wristdrop due to injury of the radial nerve (Fig. 51). Roentgenograms must be made not only in the anteroposterior but also in the lateral projection in order to obtain a complete understanding of the deformity.

Most fractures of the shaft of the humerus distal to the deltoid insertion can be treated successfully by the hanging cast (Fig. 52). This consists of application of a long arm cast with the elbow in 90 degrees of flexion, the forearm in supination and the wrist in a neutral position. The cast extends from the level of the fracture to the distal flexion crease of the palm. The thickness of the cast depends upon the amount of overriding of the fracture which must be corrected by the weight of the cast. If the cast is too heavy, it will distract the fragments—a complication which is difficult to correct. The cast must hang freely from a rope which passes around the patient's neck and is attached to the cast

near the wrist. It is important to caution the patient against resting the cast on a chair arm or other support for this will defeat the purpose of the cast by causing shortening or angulation of the fracture.

Roentgenograms should be made twenty-four hours after application of the cast, with the patient standing and the cast hanging down freely for in this way the optimum position of the reduction can be demonstrated. The radiologic check up is repeated as often as necessary for evaluating the course of the reduction and the degree of union.

The crepitus which is at first associated with movement of the bone fragments, should not be a cause of worry to the patient, since it disappears at the end of ten to fourteen days as fibrous tissue covers the end of the fragments. Union is achieved in six to twelve weeks.

If an acceptable reduction cannot be obtained with the hanging cast in seven to ten days, the fracture should be manipulated with the patient under general or local anesthesia. The hanging cast is continued in order to maintain the reduction.

Some fractures of the humerus distal to the deltoid muscle are best treated by a shoulder spica cast. This is especially applicable to patients in whom the proximal



Figure 50. Fracture of the shaft of the humerus distal to the deltoid tubercle treated by hanging cast.

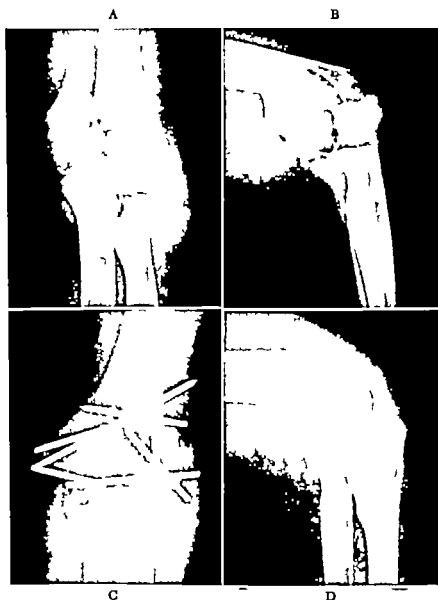


Figure 49 Comminuted fracture of the lower end of the humerus successfully treated by open reduction and multiple threaded wires. A and B Before treatment. C and D Following internal fixation. Wires were removed following union. Elbow demonstrated full flexion and 150 degrees of extension.

the interior of the joint can be obtained and as much of the lower fourth of the humerus as necessary can be uncovered by reflecting the triceps muscle and tendon upward. The ulnar nerve must be isolated at the beginning of the exposure. In some instances it may be desirable to transplant it to the front of the elbow at the time of closure. The radial nerve need not be exposed in the wound, although its location must be kept in mind to avoid its injury. The triceps tendon is sutured to the exposed end of the ulna at the end of the operation. The procedure is a highly specialized one and should not be undertaken by anyone except an experienced surgeon. It is important to understand that this fracture problem can be resolved in

spite of the hopeless appearance of the roentgenograms.

Fractures of the Shaft of the Humerus. Fractures of the shaft of the humerus may be grouped according to their location into those which occur between the humeral condyles and the insertion of the deltoid muscles, those between the insertions of the deltoid and the pectoralis major muscles and those which occur proximal to the attachment of the pectoralis major tendon.

Fractures of the shaft of the humerus distal to the deltoid tubercle (Fig 50) may be produced by direct or indirect violence. The fracture may be transverse, oblique, spiral or comminuted. They are rarely open fractures. Displacement of the fragments



A

B

Figure 53. Fracture of the humerus between the insertion of the deltoid and the pectoralis major. A, Original view of fracture. B, Note adduction of proximal fragment during hanging cast treatment. Open reduction was performed.



A

B

Figure 54. Fracture of shaft of humerus proximal to the attachment of the pectoralis major tendon with adduction of the proximal fragment by contraction of supraspinatus, infraspinatus and teres minor muscles. A, Roentgenogram showing deformity. B, Restoration of alignment by abduction of arm on splint.

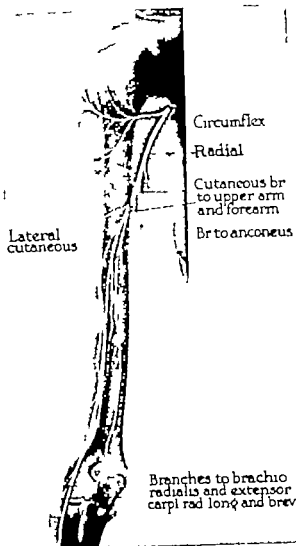


Figure 51 Anterior view of the right arm showing the relationship between the axillary and radial nerves and the humerus. The axillary nerve may be injured by dislocations at the shoulder and fractures of the surgical neck of the humerus. The radial nerve, because of its proximity to the shaft in its middle third, is particularly prone to injury when there is a fracture in this portion of the bone or by inclusion of the nerve in callus during the period of bone repair.

fragment remains abducted because of excessive obesity of the arm. Alignment, however, can be obtained in some of these patients with a hanging cast by placing enough padding on the inner side of the forearm to lever the lower fragment of the humerus away from the body and align it with the upper fragment. Tendencies toward adduction of the proximal fragment can be similarly corrected by padding the inner aspect of the arm proximal to the hanging cast, for in this way the upper fragment is levered outward.

Fractures located between the attachments of the deltoid muscle and the pec-



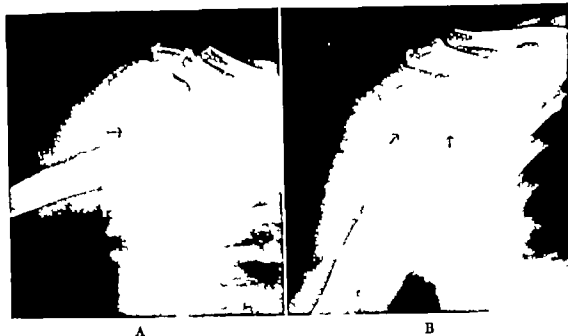
Figure 52 Photograph of hanging cast effectively used in the treatment of fractures of the surgical neck and the shaft of the humerus. The arm must hang freely at the side.

toralis major tendon may show considerable deformity. The distal fragment will be pulled proximally and become slightly abducted by the action of the deltoid, biceps and triceps muscles. The deforming force acting on the upper fragment is the unopposed pectoralis major muscle. This fragment assumes a position of adduction and slight internal rotation (Fig. 53).

Reduction of these fractures may be difficult because the proximal portion of the humerus must be abducted so that it can engage the end of the lower fragment. After the fracture is reduced, a hanging cast will usually maintain the corrected position. Open reduction is occasionally necessary.

Fractures of the shaft of the humerus proximal to the attachment of the pectoralis major tendon result in a deformity opposite to the one produced by a fracture distal to the pectoralis major insertion. The proximal fragment of the humerus, consisting of the head and that portion of the shaft between the neck of the humerus and the fracture site, is abducted and externally rotated by the supraspinatus, infraspinatus and teres minor muscles (Fig. 54). The distal fragment, consisting of the major portion of the shaft of the humerus, remains in a more or less neutral position. The adduction force of the pectoralis major balances the abduction action of the deltoid muscle.

For re-alignment of the humerus, the distal fragment must be brought into sufficient abduction to approximate the upper fragment. Some degree of external rotation o



A

B

Figure 57 Abduction fracture of the surgical neck of the humerus with separation and proximal displacement of the shaft treated by open reduction and wire fixation. A, Before reduction. B, Good anatomic result following surgery

of the humerus is characterized by varying degrees of separation and displacement of the shaft fragment. This type of fracture is divided into two subgroups, depending upon whether the shaft is in an abducted or adducted position in relation to the proximal fragment.

In abducted fractures the proximal end of the shaft of the humerus usually is displaced proximally and medially to its head and may encroach on the axilla. The head fragment may at times be broken.

Reduction is accomplished by strong downward traction on the arm while the proximal end of the shaft is manually lowered outward from the axilla so as to cause the humeral shaft to engage the end of the small head fragment. If the fracture is a transverse one, a hanging cast will or distally maintain end-to-end position until union occurs.

Oblique abduction fractures of the humerus may redisplace and require continuous traction, such as was described in the treatment of comminuted fractures of the distal end of the humerus or else the fracture must be surgically reduced and secured by internal fixation (Fig 57).

Adduction fractures of the humerus result in outward angulation of the proximal end of the shaft as well as of the head fragment. These fractures have a greater tendency to be impacted than do abduction fractures. Treatment consists of the application of a heavy hanging cast which will, as a rule,

correct the lateral angulation and maintain good relationship between the two portions of the bone. In some patients it may be necessary to manipulate the fracture in order to disimpact it. Union is achieved in six to eight weeks when the patient may resume abduction of the shoulder and use of the arm.

THE SHOULDER

Fractures of the Head of the Humerus.

Fractures of the head of the humerus may involve one or both tubercles or a combination in which the tubercles as well as the remaining portion of the head are fractured.

Fractures of the greater tubercle of the humerus may be of the avulsion type, due to contraction of the supraspinatus, infraspinatus, teres minor or a combination of these muscles. The fragment will usually be pulled upward and outward. The fracture is reduced by approximating the main portion of the humerus to the displaced small fragment. This is accomplished by abduction and external rotation of the humerus, so that the fragment resumes its normal relationship with the rest of the head. In some patients it may be necessary to change the degree of external rotation several times before stereoscopic roentgenograms show optimum reduction. Open reduction will occasionally be indicated.

In another type of fracture of the greater tubercle, the broken segment is impacted into the adjacent bone by forced abduction



Fig. 55 Compound fractures of the humerus with residual nonunion and partial defects (A) may be treated successfully by late application of bone grafts (B) across the pseudarthrosis.

the forearm and elbow is necessary since the small humeral fragment is usually externally rotated as well as abducted. The reduction is best performed with the patient under general anesthesia and is maintained by an abduction splint or a shoulder spica cast with the arm in 90 degrees of abduction and an appropriate degree of external rotation. If sufficient union is demonstrated by roentgenograms at the end of six weeks, the

spica cast can be discarded. Use of an adjustable abduction metal splint then will permit the arm to be brought gradually to the side of the trunk. Abduction exercises can at the same time be instituted, beginning with a small range of motion and gradually increasing it as the power of the deltoid muscle improves and the angle of the splint is decreased. Nonunion of this fracture is rare and the end results are very good (Fig. 55).

Fractures of the Surgical Neck of the Humerus. Fractures of the surgical neck of the humerus are of two general types. The most common is an impacted fracture (Fig. 56) in which the shaft is driven upward so that the head becomes impaled on the metaphysis as the force expends itself against the glenoid. Varying degrees of rotation of the head may be associated with this injury. Ordinarily it is not necessary to disimpact the fracture in an effort to correct the rotation unless the impaction is extreme.

Treatment should consist of immobilization of the arm at the side of the trunk for seven to ten days, following which it is placed in a sling and circumduction exercises are begun. These exercises are performed several times daily and the patient is permitted to resume active abduction motion of the shoulder against gravity as soon as he is able to do so. Sufficient union is present by the end of three weeks and precludes separation of the fragments.

The second type of surgical neck fracture

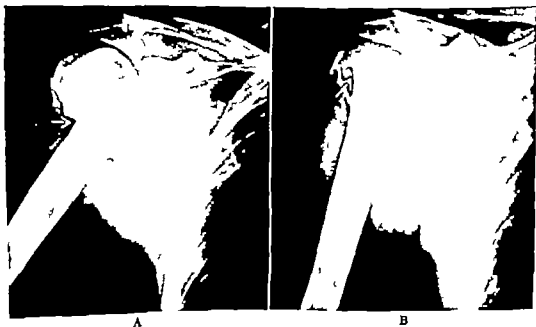


Figure 56. Fracture of the surgical neck of the humerus with backward rotation of the head and impaction into the shaft. The greater tubercle is fractured off as a third fragment. A, Before treatment. B, After therapy. Function was good in spite of persistent angulation.

greater and lesser tubercles should indicate the true nature of this interesting fracture.

With a few exceptions, full function of the shoulder cannot be obtained without recourse to open reduction. The head is disimpacted from the shaft and rotated upward to restore its proper angle with the shaft. The greater and lesser tubercles are brought into their normal position between the head fragment and the metaphysis. The tubercles are stabilized by being sutured together with heavy chromic catgut. This will in many cases offer sufficient stability between the fragment of the head and shaft without the necessity of internal fixation with metal. The arm is bandaged to the side of the trunk for three weeks, following which motion is permitted. A satisfactory range of shoulder motion is recovered in four to six months.

Comminuted fractures involving the head of the humerus may not always result in the three large fragments just described. It is considerably more difficult to reassemble multiple fragments representing the articular portion and the tubercles of the humerus and restore some of the normal anatomy. Fortunately the shoulder is not a weight bearing joint and the prognosis may be excellent even in patients in whom there is some deformity.

Dislocations of the Shoulder Joint The most frequently dislocated joint in the body is the shoulder for the shallowness of the glenoid cavity and the absence of strong ligaments evidently favor freedom of movement at the price of stability. The shoulder dislocates most frequently anteriorly although on occasion the displacement is in a posterior direction (Fig. 59). Forced ab-

duction of the arm causes the humerus to impinge against the acromion process and will in some cases, lever the head of the humerus out of the glenoid cavity and through the capsule of the shoulder joint. The head of the humerus comes to lie in front of the scapula beneath the subscapularis muscle. When the force is continued the displacement may carry the head of the humerus beneath the clavicle. On rare occasions the dislocation may be directly beneath the lower margin of the glenoid and the axillary border of the scapula. Sometimes the capsule is not only torn from its attachment to the scapula, but, in addition, the glenoid labrum is severed from the margin of the glenoid cavity.

The patient presents a typical appearance. The arm is held in a position of abduction and supported by the opposite hand. The shoulder appears flat and on palpation the proximal end of the humerus cannot be felt beneath the deltoid muscle. A marked prominence can usually be seen anterior to the shoulder and beneath the clavicle. It is caused by the displaced proximal end of the humerus.

A neurologic examination should precede reduction of the dislocation, as associated nerve injuries are not uncommon. Not infrequently the axillary nerve is injured and the patient loses active use of the deltoid muscle. This can be detected by requesting the patient to try to abduct the arm. Even faint contractions of the deltoid muscle revealed by palpation suffice to indicate that the axillary nerve is intact. Injury of the other components of the brachial plexus is less frequent.

Roentgenograms of the shoulder should



Figure 59 Roentgenogram showing a subglenoid dislocation of the shoulder

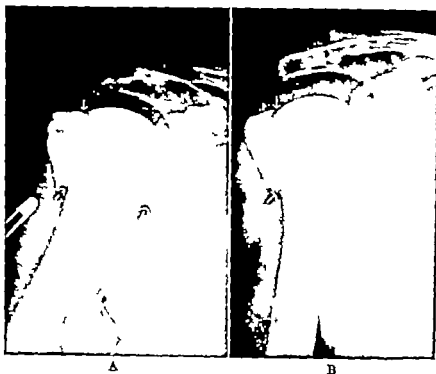


Figure 58 Roentgenograms showing impacted fracture of the humerus with backward displacement of the head fragment and fractures of greater and lesser tubercles. A, View showing deformity B, Final result. No reduction was performed but function was normal.

injury. There is pain and hesitancy to initiate shoulder motion. Roentgenograms will disclose the impacted nature of the fracture. No treatment other than the use of a sling is usually required. The patient is encouraged to perform swinging circumduction exercises immediately and to resume abduction motion as early as possible, for rarely will the fracture disimpact or fail to heal. Roentgenograms should be made frequently during the first three weeks to make sure the fragment has not displaced. If it has separated from the shaft, then the extremity should be immobilized in a cast in abduction and external rotation.

Fractures of the greater tubercle are also encountered in association with anterior dislocations of the shoulder. The fragment usually remains in its normal position and falls into its defect in the humeral head as the dislocation is reduced. Union occurs to a sufficient degree in four weeks to allow resumption of motion. In many patients the arm must be abducted and externally rotated in order to obtain reduction of the fracture, although this position carries within it the danger of recurrence of the dislocation. The arm should therefore be kept in as much adduction and forward flexion as possible to minimize the tension on the anterior capsule of the shoulder without jeopardizing the reduction.

Isolated fractures of the lesser tubercle of the humerus are infrequent and usually of the avulsion type, because of the subscapularis muscle. The fracture is treated by rotating the arm internally and keeping it mobilized at the side of the trunk for four weeks to permit the humeral head to approximate the lesser tubercle.

An injury of unusual interest is a comminuted fracture of the humerus in which the articular portion of the head is driven backward with such violence that the greater and lesser tubercles are forced to give way and the head becomes impacted on the upper end of the shaft (Fig. 59). The fracture results from an injury in which the humerus is forced violently against the glenoid portion of the scapula. The roentgenograms show a characteristic appearance. The articular cortex which outlines the head of the humerus in the roentgenogram will be found to lie horizontally across the end of the shaft instead of at an angle of 45 degrees to the longitudinal axis of the humerus. There is, moreover, considerable displacement between the head and the glenoid fossa.

The first interpretation of the roentgenogram showing marked displacement of the tubercles suggests dislocation of the shoulder but the characteristic displacement of the head and the associated fractures of the

muscle is next detached from the humerus. The humeral head is replaced and fixed to the shaft by loops of wire or staples. Following this, the capsule is repaired, the muscles are replaced and the wound is closed. The arm is then bandaged to the side of the trunk for three weeks, after which time motion is resumed. The fact that the humeral head undergoes aseptic necrosis from loss of the blood supply has prompted some surgeons to advocate its excision. However, since the head usually unites with the shaft and is in most instances gradually replaced by new bone, it is better to retain the head rather than to incur a flail shoulder which results from its removal.

THE SHOULDER GIRDLE

Fractures of the Clavicle. The clavicle is most frequently fractured in its middle third. The injury results from a fall onto the shoulder as well as a direct blow against the clavicle. It occurs most often in children.

The symptoms consist of pain, swelling and deformity at the site of the fracture. A fracture of the clavicle should always be suspected when a small child has sustained a fall from a chair or bed and subsequently hesitates to use the extremity. The presence of local tenderness over the clavicle, together with the demonstration of crepitus or deformity when present, should establish the diagnosis. The fracture is more easily recognized in adults who will refer the symptoms to the clavicle.

The deformity of displaced fractures of the clavicle is characteristic. The medial

fragment is pulled proximally and backward by the sternocleidomastoid muscle. The outer portion, the scapula and upper extremity fall forward, downward and toward the midline (Fig 60) because of the weight of the extremity and loss of the normal support provided by the intact clavicle. Stereoscopic roentgenograms will reveal the nature of the deformity.

Reduction of fractures of the clavicle is accomplished by approximating the displaced outer fragment to the medial one. This is achieved by elevation and retraction of the shoulder girdle which will mobilize the outer fragment. Strong backward pressure on both shoulder girdles is required to correct marked overriding. Numerous methods have been devised for immobilization of these fractures but a figure-of-eight plaster dressing is preferred. It is applied as follows: The patient is seated on a stool or a table and local anesthesia is induced at the fracture site. One assistant then elevates the patient's right arm and retracts it as far posteriorly as possible. At the same time a second assistant places the patient's left arm in a similar position. The operator stands between his assistants and behind the patient. Sheet wadding and then plaster are applied in a figure-of-eight manner crossing the clavicles passing under the armpits and crisscrossing posteriorly. As the plaster begins to set, the patient is placed in the supine position on a table with a small sandbag between the scapulae. Backward pressure is exerted on the shoulders by the surgeon to obtain maximum retraction,



Figure 60 Fracture of the clavicle treated by means of a figure-of-eight plaster dressing. A, Note the downward and forward displacement of the outer fragment and the proximal position of the outer end of the medial fragment. B, Union of the fracture occurred in six weeks.

be obtained in all these patients, since there are associated fractures of the head or neck of the humerus, the most common being avulsion of the greater tubercle.

Several methods are available for reduction of simple anterior dislocation of the shoulder. The oldest, often referred to as the hippocratic method, consists of downward traction on the arm while the unbooted foot of the physician is placed in the axilla. Gradual adduction of the extremity will usually effect a reduction. Care must be exercised that the pressure exerted in the axilla does not injure its contents.

The technique of the Kocher method is safe provided it is carried out gently. It is quite easy to fracture the proximal end of the humerus if the shoulder muscles are not relaxed or if too much force is applied. Manipulation is ordinarily carried out with the patient under general anesthesia. It can be done equally well following an adequate dose of morphine or Demerol, permitting the patient actively to perform the maneuvers as directed by the surgeon.

The patient is supine on the table and an assistant places an arm around the chest near the axilla so as to provide upward and outer pull for countertraction. The surgeon flexes the patient's elbow, obtains a strong hold on the arm just above the flare of the distal humeral condyles and pulls strongly downward. The arm is kept in about 20 degrees of abduction. The patient is instructed to rotate the arm externally. When his hand approximates the surface of the bed or table he is to move his elbow toward the front of his chest. As the elbow reaches the side of the trunk, his hand is placed on his opposite shoulder. Invariably the dislocation will be reduced on the first attempt. There is little pain during the maneuver because the muscle spasm which would ordinarily act to oppose the manipulation is absent. If the humerus is not replaced after two attempts, the patient should be given a general anesthetic and the manipulation repeated. Restoration of the normal contours of the shoulder will indicate that reduction has been successful. The arm is then bandaged to the trunk with the hand in front of the thorax. It should be held there for three weeks after roentgenograms have confirmed the accuracy of the reduction.

Patients with recurrent dislocations are permitted to use the extremity without an intermediate period of immobilization, for it will not preclude the possibility of subsequent dislocations.

Posterior dislocations of the shoulder are relatively rare. They are manifested by flattening of the shoulder, a prominence posterior to the joint and an adducted position of the arm. Reduction is accomplished by downward traction on the extremity and forward pressure on the head of the humerus.

Neglected dislocations of the shoulder cannot generally be reduced by closed manipulation. The head of the humerus should be replaced in the glenoid cavity by surgery even though the result is not wholly satisfactory because of persisting limitation of motion.

Fracture Dislocations of the Shoulder Joint. Anterior dislocations of the shoulder with simultaneous fracture of the surgical neck of the humerus occur most commonly. Dislocations accompanied by fractures of the coracoid process or the glenoid cavity also occur although very infrequently. A fracture-dislocation of the shoulder which involves the neck of the humerus occurs mainly in older people. The fracture takes place while the arm is in abduction, the shaft of the humerus then drops alongside the trunk while the head of the humerus remains displaced beneath the subscapularis muscle.

The diagnosis is usually suggested by the pain, loss of shoulder function, flattened deltoid region, palpable swelling under the clavicle and crepitus on passive motion. The exact level and type of fracture are not easy to gauge except by means of roentgenograms.

Replacement of the head of the humerus should be the first step in treatment. With the patient under general anesthesia, downward traction is made on the arm, and thereby on the humerus, to make room between it and the scapula, thus making possible the replacement of the humeral head into the glenoid cavity. Care must be taken that contents of the axilla are not injured during the manipulation. If manual reduction is not successful, traction is applied to the arm while it is in 90 degrees of abduction, and an attempt is made to pull the head into the capsule by way of the untorn soft tissues between the head and the shaft. Open reduction must be performed if the head is still dislocated.

The shoulder is exposed through an anterior deltoid incision. The short head of the biceps and the coracobrachialis muscles are reflected downward following osteotomy of the tip of the coracoid process. The subscap-

terposed between the articular surfaces of the outer end of the clavicle and the acromion gives additional stability to the articulation and permits greater range of motion.

Dislocation of the acromioclavicular joint (Fig. 62) may displace the outer end of the clavicle upward, backward or forward. The degree of displacement will depend upon the severity of the injury, to the restraining soft tissues. Rupture of the acromioclavicular ligament may permit the clavicle to dislocate, but it is only in the presence of rupture of the coracoclavicular ligament that wide displacement occurs.

The injury results either from a fall on the shoulder or from forces directed upward through the arm to the acromioclavicular joint. The patient usually experiences little pain, while the physical examination discloses local swelling and a palpable deformity at the acromioclavicular joint.

For minor subluxations of the acromioclavicular joint, adhesive dressing may suffice (Fig. 63). On the other hand, it is difficult to maintain adequate downward pressure on the outer end of the clavicle for six weeks by means of adhesive to correct major dislocations and to permit repair of the ligaments and capsule.

A satisfactory result can often be obtained by the use of a body and arm cast but this entails immobilization of the corresponding extremity for at least six weeks. A circular body cast which extends from the mid thorax to below the iliac crests is applied to

the trunk. It is joined with a second cast which encases the arm on the side of the injury with the elbow in 90 degrees of flexion. The dislocation is then reduced and held by a strap of webbing which crosses over the clavicle and is attached in front and back to the top of the body cast.

Some surgeons prefer open reduction for treatment of complete dislocations of the acromioclavicular joint. This is accomplished by a curved incision made along the outer portion of the clavicle and around the shoulder. The skin is reflected to expose the acromion and the adjacent clavicle. The dislocation is corrected and maintained by drilling two threaded wires through the acromion process into the outer fourth of the clavicle (Fig. 64). The capsule and ligaments are repaired and the wound is closed. The arm is bandaged to the side of the trunk for two weeks, after which cautious use of the extremity is allowed. The threaded wires are removed at the end of ten weeks.

Some chronic dislocations of the acromioclavicular joint are compatible with full use of the extremity. In the presence of pain and marked disability the symptoms may be relieved by resection of the clavicle lateral to the coracoclavicular ligaments, provided these are intact. This type of resection is particularly effective in relieving the pain.



Figure 62. Roentgenogram showing proximal dislocation of the outer end of the clavicle at the acromioclavicular joint.



Figure 63. Photograph of a patient with an acute acromioclavicular dislocation treated by adhesive dressing (Key J. A., and Conwell, H. E., *The Management of Fractures, Dislocations and Sprains*. C. V. Mosby Company.)



Figure 61 Intramedullary fixation of comminuted fracture of clavicle, which could not be reduced adequately by manipulation.

at the same time permitting the plaster dressing to mold itself over the sandbag and between the scapulae. The cast is trimmed to maintain a buttress in front of the outer end of the clavicle but is cut away sufficiently in the axillae to prevent excessive pressure upon their contents.

Roentgenograms are obtained following application of the cast and if reduction is not satisfactory it should be repeated. Accurate end-to-end repositioning of fractures of the clavicle is not necessary in the average patient. A considerable degree of persistent overriding may be accepted in young children, since the clavicle will be remodeled as growth continues, with a satisfactory end result. Some overriding may be accepted in adults, provided there is no undue prominence to cause pressure symptoms when the patient carries objects on his shoulder. A cosmetically acceptable result is, of course, important in women and may require continuous arm traction in abduction with the patient at bed rest.

Comminuted fractures of the clavicle can in most instances be reduced satisfactorily by the methods just described. In a few patients, open reduction is necessary espe-

cially when one of the lesser fragments rotates and becomes caught in the adjacent soft tissues. An appropriate incision is then made just below the site of fracture. The bone is exposed subperiosteally and internal fixation is obtained by means of a heavy intramedullary threaded wire (Fig. 61). The wire is first drilled outward through the medullary canal of the lateral fragment, permitting it to emerge through the skin in the suprascapular region. The fracture is then reduced and the threaded wire is drilled medially for a sufficient distance into the inner fragment. The smaller fragments are brought into their normal position and fixed with circular loops of heavy chromic catgut. The wound is closed and the arm is bandaged to the side of the trunk. Sutures are removed at the end of ten days, when the arm is placed in a sling and shoulder motion is resumed gradually. Union occurs in young children in about six weeks, in adults it may take eight to ten weeks.

Dislocations of the Acromioclavicular Joint. Stability of the acromioclavicular joint is dependent upon the intactness of the acromioclavicular and the coracoclavicular ligaments. A cartilaginous disk which is in-

crepitus of air in the subcutaneous tissues will be felt and the condition may spread over the whole of the chest even up to the neck and down to the abdomen. Muscular violence due to heavy lifting, sneezing or coughing may also cause rib fractures although the incidence is low. The upper two ribs are infrequently broken because of the protection afforded by the clavicle. The lowest, or floating, ribs are freely movable and difficult to break.

The fracture may be transverse, oblique or comminuted (Figs. 65 and 66). Displacement is usually slight. Open fractures, except those from gunshot wounds are rare.

Symptoms include severe pain in the chest, with aggravation by deep breathing and coughing or straining, swelling and tenderness will localize the site of the fracture. When crepitus cannot be detected by palpation, it may be heard at times with the stethoscope. Crepitus is ordinarily present for ten to fourteen days, by which time fibrous tissue and some callus will have covered the ends of the fragments and eliminated movement.

Röntgenograms must be taken after every chest injury. Portable films are satisfactory if they are taken with the patient in the upright position and are overexposed to demonstrate fractures, collapse of lung tis-

sue and a fluid level. Occasionally a linear fracture without displacement is not visible in the x-ray film thus leaving the diagnosis of fracture uncertain until callus is seen in a later roentgenogram or until palpable fracture or separation of a costal cartilage cannot be demonstrated on the x-ray film and its detection must be made by physical signs alone.

Multiple fractures with collapse of the chest wall may produce shock and dyspnea, the intrathoracic damage being so severe as to cause death. It is therefore necessary to begin immediate administration of intravenous fluids while the patient's blood is being crossmatched for transfusion. At the same time it is imperative to evaluate the status of the respiratory mechanism in these patients and to make every effort to restore its normal functioning, first by aspiration or intubation. Tracheotomy and skeletal traction should be employed to stabilize the chest wall if necessary. Steinmann pins can be passed parallel to the ribs beneath the pectoral muscles on one or both sides of the chest and attached by means of spreader bows to weights suspended from an overhead frame. Definitive treatment of other injuries must be delayed until this is effected.

Both single and multiple fractures, if uncomplicated may be treated by restricting the respiratory excursions, which can be



Figure 65. Roentgenogram showing fractures without displacement of the lower four ribs. This type of fracture can usually be treated by adhesive strapping or an Ace bandage which encircles the lower portion of the chest so as to restrict inspiration movements.



Figure 66. Crushing injury to chest with multiple fractures of ribs and collapse of lung frequently requires lifesaving measures in treatment.



Figure 64 Dislocation of the acromioclavicular joint unsuccessfully reduced by closed method and subsequently treated by open reduction and pin fixation. The ligament and capsule were repaired at the same time. Wires were removed at the end of three months. The result was good.

and limitation of motion associated with arthritis which often follows injuries to the acromioclavicular joint. Arthrodesis should never be performed since it restricts elevation of the arm.

Dislocation of the Sternoclavicular Joint. Dislocations of the clavicle occur much less frequently at its inner than at its outer end. The sternoclavicular joint is reinforced by the sternoclavicular and the costoclavicular ligaments. A cartilaginous disk is interposed between the articular surfaces of the joint and facilitates movement of the clavicle on the sternum.

In dislocations of this joint, the displacement is usually in an upward direction and is the result of a fall on the shoulder. The displacement may be forward. Little if any pain is associated with the dislocation and the patient notices only a firm prominence at the base of the neck. In many patients, early medical care is not sought because of the minimal disability produced by the dislocation.

The diagnosis is easily established by physical examination but is difficult to demonstrate by roentgenograms.

Acute dislocations of the sternoclavicular joint are hard to reduce by conservative means. There is no satisfactory method of

maintaining the clavicle in its normal relationship with the manubrium following closed reduction. Open reduction is probably the most satisfactory means of correcting the dislocation. Heavy wire must be used to anchor the inner end of the clavicle to an adjacent rib while at the same time the ligaments are repaired. The arm is immobilized in a Velpeau dressing for three weeks.

Chronic dislocations of the sternoclavicular joint are best left untreated unless they cause considerable disability in which case the inner end of the clavicle can be resected.

Fractures of the Scapula. Fractures of the scapula may involve its spine, the glenoid portion of the bone, the supraspinatus or infraspinatus fossae. They are the result of direct violence, such as a blow to the shoulder blade. Comminution may be marked, but the displacement is usually minimal.

No special treatment is required for these fractures other than the support of the arm in a sling and a bandage around the trunk to immobilize the scapula against the chest wall. Function of the extremity can be resumed in about three weeks.

On rare occasions, a fracture of the scapula is seen in which a portion of bone is angulated to such a degree that it cannot be reduced by manipulation. In these patients, open reduction is required to relieve the prominence; if the bone fragment is not too large or represents a sizable portion of the glenoid cavity it may be removed without impairing the normal function of the scapula or the shoulder.

Fractures which involve the neck or the glenoid cavity require more careful treatment. Frequently the fractures are impacted and require a sling only until the pain and reaction from the injury subside, following which function can be rapidly resumed. Skeletal traction with a pin through the proximal end of the ulna is necessary to reduce some displaced fractures involving the glenoid portion of the scapula. Open reduction is rarely indicated.

THE THORAX

Fractures of the Ribs. An injury to the front of the chest compressing the thoracic cage commonly results in fractures of the ribs along their axillary aspects. A severe direct blow which fractures the ribs may damage the pleura and the lungs, causing pneumothorax, hemothorax or surgical emphysema. With the latter the characteristic

be necessary to stop uncontrollable hemorrhage, relieve tension pneumothorax or to remove large blood clots. If empyema occurs, it must be treated either by antibiotics or surgery as may be indicated.

Open fractures of the chest wall require immediate debridement, closure of the wound, continuous aspiration and re-expansion of the lung. Elderly patients should be kept in the sitting position out of bed as soon as their condition will permit.

Pain usually disappears in two to three weeks. Rapid and satisfactory union occurs as a rule, in even the untreated patients in six to eight weeks. Nonunion rarely occurs and malunion hardly ever causes undesirable symptoms. Finally in the more severe chest injuries the prognosis must depend primarily on the complications.

THE SPINE

Any patient who complains of pain in the neck or back after a fall from a height a severe blow to the shoulders an automobile accident or similar injuries should be assumed to have sustained a fracture of the spine until examination proves otherwise. Fractures of the spine may be associated with dislocation of the facets while displacement of one vertebra on another may not be accompanied by a concomitant fracture. The spinal cord may be injured at the same time by displaced bone fragments by lacerations, hemorrhage or a complete transection as one vertebra dislocates forward on the one below.

Patients with fractures of the spine must

receive expert care from the moment of the accident until the fracture or dislocation has been diagnosed and appropriate treatment instituted.

Fractures and Dislocations of the Cervical Spine. Fractures of the cervical vertebrae are best treated by skeletal traction using Crutchfield tongs. The tongs can be inserted with the patient under local anesthesia and without placing him on the operating table, while gentle manual or halter traction is maintained on the head. Fifteen pounds of traction is adequate for the treatment of the average cervical fracture.

The head is kept in a neutral position at first, and then is gradually brought into hyperextension in order to obtain maximum reduction of the fracture. A Minerva jacket is applied at the appropriate time and its use is continued for a period of three months. A removable cervical collar is then substituted and motion of the head and neck is gradually resumed. The collar is discarded after the spine is mobilized and muscle tone has been recovered.

The best procedure for reduction of dislocations of the cervical spine is continuous skeletal traction (Fig. 69). Fifteen pounds of straight traction is applied and portable roentgenograms are obtained after thirty minutes. If the inferior articulating facet of the displaced vertebra has not been pulled upward beyond the level of the superior articulating facet of the body below an additional 5 pounds is added to the traction. Roentgenograms are repeated at intervals of thirty minutes and further weights are added

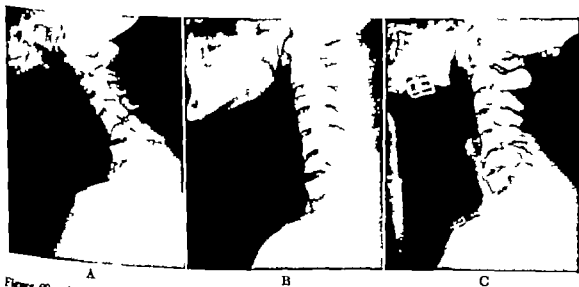


Figure 69 Anterior dislocation of the fifth cervical body successfully treated by Crutchfield tongs. A, Roentgenogram of dislocation. B, View showing reduction while traction was applied. C, Final result. Note bridge of bone anteriorly between the fifth and sixth bodies which increased the stability of the spine.

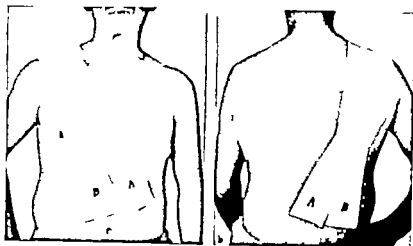


Figure 67 Front and back photographs of a patient, showing the vertical method of adhesive strapping for fractures of the upper four ribs. (Key J. A., and Corwell, H. E.: *The Management of Fractures, Dislocations and Sprains*. C. V. Mosby Company.)

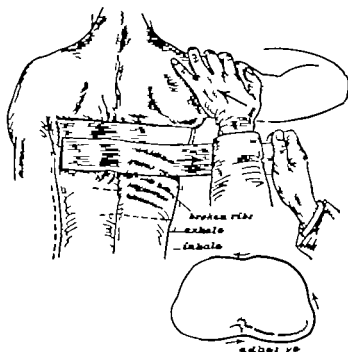


Figure 68 Strapping ribs. The adhesive extends from the opposite side of the chest posteriorly to well past the midline anteriorly. The skin is first painted with tincture of benzoin in order to avoid reaction from the adhesive tape.

done by a local strapping that crosses the midline in front and back or by a circular dressing around the lower portion of the thorax. The material used for this purpose may be adhesive (Figs. 67 and 68) elastic bandage or a rib belt. Adhesive strapping should not be too tightly applied in patients with multiple displaced fractures, so as not to increase the deformity. Instillation of 1 per cent Novocain into the hematoma at the fracture site will relieve pain and muscle spasm. Often a dry and persistent cough is

present from pleural irritation which may be relieved by a sedative cough mixture or codeine.

Emphysema usually does not require treatment and disappears in a few days. Pneumothorax with severe dyspnea can be relieved by aspiration of the air. If necessary a catheter may be inserted and an air seal and a water bottle used for continuous aspiration. Blood in the pleural cavity should be removed as demanded by its interference with lung function. Open surgery may

Open reduction of fractures and dislocations of the cervical spine must be performed while skeletal traction is maintained on the head. The laminae and facets are exposed in the usual manner; traction is then increased with the facets under direct vision. When the articulating processes clear each other the head is gently extended and traction is released as the upper vertebra is brought into proper alignment with the body below. A loop of wire is placed around the spinous processes of the two vertebrae to maintain the reduction. Many surgeons feel that a spinal fusion should be done at the same time.

Fractures of the odontoid process are usually associated with dislocation of the atlas on the axis (Fig. 70) and are produced by falls in which the head is thrown violently forward. Treatment consists of skeletal traction with the head in a neutral position. After the first cervical segment has cleared the axis, the head is gradually extended until reduction is accomplished and the odontoid fragments assume their normal relationship. Skeletal traction is maintained for four weeks, following which a head and body cast is applied for an additional eight weeks.

Dislocations of the atlas on the axis with or without an associated fracture of the odontoid process are of grave danger to the patient, because transection frequently occurs when the cervical cord is forced against the pointed end of the process as the atlas moves forward. Here, too, treatment consists in the immediate application of skeletal traction and gradual reduction of the dislocation by extension of the head. The subsequent treatment is the same as that for a fracture-dislocation of the odontoid process.

Fractures of the Dorsal Spine. Those which occur proximal to the seventh thoracic vertebra are difficult to reduce as is reflected by the conservative attitude of many surgeons. These patients are therefore provided with a Taylor-type back brace or a lumbodorsal corset, reinforced with heavy stays to relieve the strain on the fractured body and to maintain maximum posture of the spine during the three months required for healing. Some surgeons prefer a body cast which extends upward from the pelvis and over the tops of the shoulders.

Fractures of the Lumbodorsal Spine. Fractures of the lower thoracic vertebrae and those involving the upper lumbar bodies are due to severe flexion motion of the spine in which the patient assumes a so-called

jackknife position. These are the most common fractures which occur in the spine. An effort should be made to reduce the involved body accurately.

The resulting deformity of the vertebral body may be of two general types. If the pressure which is applied to the body is evenly distributed to the superior surface of the vertebra below the latter will become wedge shaped as its anterior height is shortened and the posterior portion remains unchanged (Fig. 71). If on the other hand the angulation of the spine at the time of the injury is more acute, the vertebral body will likely be broken into several segments and result in a comminuted fracture. The adjacent intervertebral disks are usually also injured and the prognosis is less favorable than in patients having simple wedge fractures.

Patients with deforming fractures of the lumbodorsal vertebrae must be treated in the hospital. If there are no associated injuries, the patient may be taken to surgery for reduction of the fracture and application of an adequate body cast. Many surgeons



Figure 71 Roentgenogram of the lumbodorsal spine showing depressed superior surface of the first lumbar vertebra. The fracture was treated by means of a lumbodorsal corset reinforced with heavy stays. No further collapse occurred.

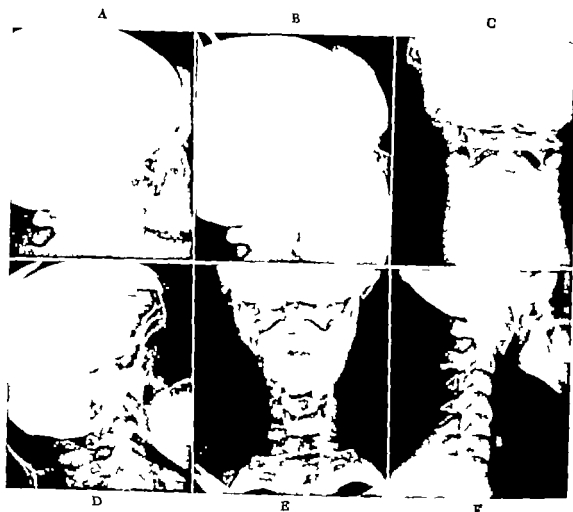


Figure 70 Fracture of the odontoid process with anterior displacement of the atlas on the axis. A, The condition at beginning of treatment. B, Reduction of dislocation by traction. C, View showing separation of atlas and axis with 10 pounds of traction. D, Maintenance of reduction in cast. E and F, Final anatomic result.

if indicated. As soon as the facets have cleared each other the head is placed in hyperextension and roentgenograms are again made. If the anteroposterior and lateral views show the facets to be in proper alignment, the traction is gradually decreased to permit reduction of the dislocation as the two vertebrae approximate each other and the facets lock. After accurate replacement has been accomplished, traction is continued, using 10 pounds of weight for three to four weeks. A Minerva plaster jacket is then applied for an additional eight weeks. The jacket should extend from the pelvis and include the head. Some surgeons apply the cast immediately after reduction of the dislocation with the head placed in moderate hyperextension, provided the reduction is stable. The patient can then become ambulatory.

Roentgenograms must be repeated at intervals of two to three weeks to make sure that the dislocation has not recurred. The

cast is discarded after three months, at the end of which period a cervical collar is used until painless motion has been restored.

Dislocations and fracture-dislocations of the cervical spine which cannot be adequately reduced by skeletal traction require open reduction. Manipulation of acute bilateral dislocations of the cervical vertebrae is not recommended, although there is no objection to careful manipulation of a unilateral dislocation of a cervical facet. This is accomplished by applying firm traction to the chin while angulating the head and neck toward the side of the intact joint. After the articulating process of the dislocated vertebra has cleared the one below the head is gently rotated toward the side of the dislocation so as to bring the facets in realignment. The traction is then released and reduction is completed. Roentgenograms will show whether manipulation has been successful. Persistent dislocations require surgery.

on the psoas muscles and may at the same time allow some reduction of the fractures. A plaster cast can be applied after seven to ten days if several of the transverse processes are fractured and the patient's condition warrants fixed immobilization. Most indi-

viduals with only involvement of one or two processes will recover satisfactorily after local infiltration of the fracture sites with Novocain and protection of the back from lifting and other strenuous activities for six weeks.

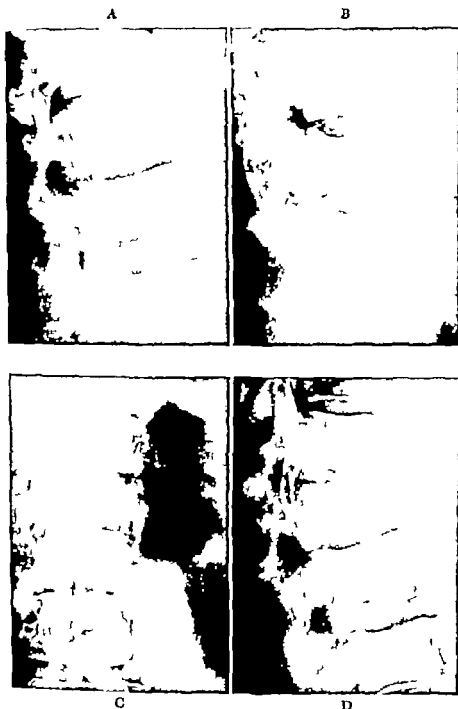


Figure 73. Comminuted fracture of the first lumbar vertebra carries a poor prognosis because of damage to the intervertebral disks and the tendency for the deformity to recur in spite of good reduction. A, Original fracture. B Following satisfactory reduction. C and D Condition after six months, showing further collapse of the body. Note the ossification of the ligaments which advantageously effected a local fusion.

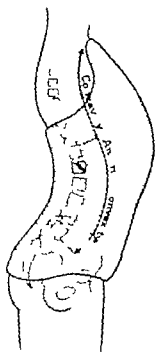


Figure 72. Diagram showing the type of plaster jacket used in fractures of the lumbar vertebral.

recommend bed rest for several days until the abdominal distention which invariably accompanies a fracture of the spine is relieved. Others believe that the distention is more easily controlled if the fracture is immediately reduced.

Numerous methods have been devised for reducing compression fractures of the lumbar region, but the two-table technique as described by Watson Jones is preferable. This consists in suspending the patient in the supine position between two tables. The table which holds the upper end of the body should be about 6 inches higher than the one which supports the legs. The patient is allowed to assume a position of maximum degree of hyperextension of the spine without actual manipulation of the back by the surgeon. Reduction of the fracture is accomplished by the corrective force exerted on the fracture by the tension generated in the anterior longitudinal ligament and the annulus fibrosus of the intervertebral disks. The patient is given an adequate dose of a sedative, but a general anesthetic should not be used. A plaster cast is finally applied which must extend from the sternal notch to the symphysis. The cast is trimmed posteriorly so that the scapulae are free but the lower margin of the cast should extend well down to the gluteal areas posteriorly and the symphysis anteriorly (Fig. 72).

The patient is then returned to bed. Some

surgeons favor a period of recumbency of from four to six weeks and others recommend ambulation as soon as the patient becomes adjusted to the cast and is relieved of pain in the spine and of abdominal distention.

Other methods of reducing fractures of the lumbodorsal region consist of suspension of the patient by the ankles in a supine position on the fracture table (Davis method) correction of the deformity by means of a hyperextension jack as described by Ryerson the use of the Goldthwaite trons or gradual correction of the deformity by progressive hyperextension of the spine with the Gatch bed in reverse.

The goal of treatment of fractures of the lumbodorsal spine is complete restitution of the normal height and shape of the vertebrae regardless of the way reduction is accomplished, immobilization in a properly fitting plaster cast, institution of a supervised exercise program to maintain good tone of the muscles of the back and abdomen and, after removal of the cast, a rehabilitation program to enable the individual to resume his usual form of employment.

Healing of wedge-shaped fractures of the lower thoracic and upper lumbar vertebrae is complete by the end of four months, at which time the cast is removed. Comminuted fractures unite more slowly and the deformity has a tendency to recur (Fig. 73) and, therefore, immobilization in a cast for four months should be followed by the use of a Taylor back brace for two months.

Fracture-Dislocations of the Lumbodorsal Region. These are best treated by open reduction. The facets are exposed through an adequate incision. The patient then is cautiously flexed on the table and longitudinal traction is applied in order to free the facets and to permit reduction of the dislocation by extension. A spinal fusion should be performed at the same time to insure stability of the back. A cast is applied and left in place for four months.

Fractures of Transverse Processes of the Lumbar Vertebrae. These fractures are common. They represent avulsion fractures in which the tips or other portions of the transverse processes are separated as a result of spasm of the psoas muscles. Following the injury these patients demonstrate the findings characteristic of acute low-back strain. The fractures are discovered on routine roentgen ray examination.

The patient is best treated by bed rest in a sitting-position, for this relieves the tension

on the psoas muscles and may at the same time allow some reduction of the fractures. A plaster cast can be applied after seven to ten days if several of the transverse processes are fractured and the patient's condition warrants fixed immobilization. Most indi-

viduals with only involvement of one or two processes will recover satisfactorily after local infiltration of the fracture sites with Novocain and protection of the back from lifting and other strenuous activities for six weeks.

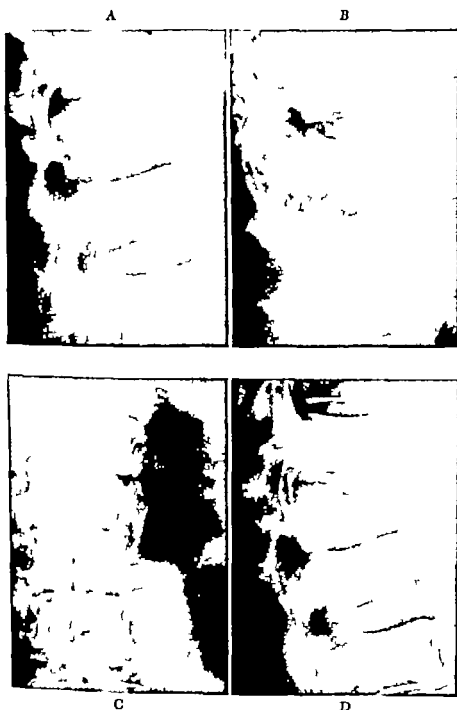


Figure 79. Comminuted fracture of the first lumbar vertebra carries a poor prognosis because of damage to the intervertebral disks and the tendency for the deformity to recur in spite of good reduction. A, Original fracture. B, Following satisfactory reduction. C and D, Condition after six months, showing further collapse of the body. Note the ossification of the ligaments which advantageously effected a local fusion.

Fractures and Dislocations of the Pelvis and the Lower Extremity

By HAROLD A. SOFIELD M D

HAROLD AUGUSTUS SOFIELD born in New Jersey received his education by geographically covering the United States. Columbia University Northwestern University and San Francisco City-County Hospital have contributed to his experience. He chose to receive his orthopedic training in the Midwest, where he has remained as Chief Surgeon Shriners Hospital for Crippled Children Chicago Unit. He is Professor of Orthopedic Surgery at Northwestern University Medical School.

FRACTURES OF THE PELVIS

Pelvic fractures fall into four general types: fractures of the rim of the pelvis, acetabular fractures, fractures of the pelvic ring, and fractures of the sacrum and coccyx.

These fractures are common and are increasing in number because so many injuries are received while persons are in sitting positions in automobiles and airplanes. The most serious fractures are those of the ring of the pelvis, practically all of which are compression fractures. When disruption of the pelvic ring, which is formed by the bones surrounding the pelvic opening, is severe viscera present inside the pelvic ring may be punctured by sharp spicules of the fractured bones. The bladder and the rectum are most commonly punctured, but tears of the urethra are frequent and injuries of the ureters are not uncommon. In all persons having fractures involving the pelvic ring in which significant displacement of fragments is present it is imperative that the condition of the urinary tract be ascertained as promptly as possible. If the patient voluntarily voids clear urine with no blood the chances are against any serious damage being present. A small amount of blood in the first specimen followed by clear, bloodless urine usually means injury to the mucous membranes, but no serious damage. When the patient cannot void, he should be carefully catheterized with a small, soft catheter. If the catheter cannot be introduced into the bladder and if blood is present at the tip of the catheter injury to the urethra may be suspected. If the catheter passes into the blad-

der and blood is persistently found in the urine injury to the bladder may be surmised. Once the catheter is in place, it should not be removed, for it may be very difficult to reinsert later. Whenever injury to the urethra, bladder or ureter is suspected, immediate steps should be taken to treat the injury. All patients with pelvic ring fractures should have a rectal examination to see whether a sharp fragment of bone may have punctured the lining or may be lying close to the rectum. Occasionally a sharp fragment may be moved away from danger by digital pressure. Shock accompanying violent fractures of the pelvic ring which produce severe displacement is often profound and sometimes fatal. Gentle manipulation to reduce marked displacement should be attempted because gross displacement adds to the shock.

Fractures of the Rim of the Pelvis. Almost the entire outer surface, or rim, of the pelvis serves for the attachment of muscles. Strong muscle contractions, particularly when the individual is participating in athletics, cause avulsion type fractures. Certain areas of the rim where strong muscle groups are attached to relatively small areas of bone are most vulnerable. Avulsion fractures of the rim occur most commonly at the anteroinferior iliac spine, the anterosuperior iliac spine, the crest of the ilium and the tuberosity of the ischium. Treatment consists of bed rest for a week or two until pain is reduced, then limited activities for several weeks. Even though displacement seems rather marked, closed manipulation is useless and open re-

placement with fixation is not required because none of these fractures seems to have any effect on ultimate function.

Acetabular Fractures. Two groups of fractures may be identified in consideration of fractures of the acetabulum: fractures of the rim of the acetabulum and fractures of the floor of the acetabulum.

Acetabular fractures are frequently grouped under dislocations of the hip because so-called central dislocations of the hip often accompany fractures of the acetabular floor. There are, however, many acetabular fractures in which there is no displacement of the head of the femur and, in common with pelvic fractures in general, the number of acetabular fractures is increasing. In about one-half of the patients with acetabular fractures, other pelvic fractures are present (Fig. 74). Marked displacement of the acetabular floor into the pelvis may in rare instances, cause injury to viscera or nerves occupying the pelvis. These fractures are most apt to appear in young and middle-aged adults; in older persons the same type of force is more likely to cause fractures of the upper end of the femur.

Fractures of the rim of the acetabulum may occur with or without dislocation of the hip. They are usually caused by the head of the femur being forcibly driven against the acetabular rim as is found in the mechanism producing posterior dislocation of the hip. Most frequently the rim fractures are at the posterosuperior border of the acetabulum, but, on rare occasions, practically the whole rim may break loose. Customarily the piece, or pieces, of the posterior rim are driven



Figure 74 Fracture of the floor of the acetabulum pushing the ischial portion inward. There is an associated fracture of the pubis near the symphysis.

backward and if the head of the femur follows, producing a posterior dislocation, the fractured portion of the rim may stay closely connected to the femur and may reduce satisfactorily when the dislocation is reduced. If however there is no dislocation and the fractured fragments are large and significantly displaced from their anatomic position, or if large fragments accompanying a dislocation do not come into place when the dislocation is reduced, open reduction with fixation by a screw or screws is indicated (Fig. 75). Provided the pieces are small and are not displaced into the acetabulum, they may be ignored.

If the fractured pieces of the rim are large, light traction on the leg should be instituted and continued for about four weeks. This is true whether there was no significant displacement to begin with or whether open



Figure 75 A, Fracture of posterior rim of the acetabulum with persistent displacement. B, Open reduction and screw fixation of fracture of acetabular rim. Traction should be continued for several weeks.

reduction and internal fixation were required. Every effort should be made to keep the acetabulum as near normal as possible. After removal of balanced Russell traction the patient is permitted to exercise in bed for one week and is then given crutches for two weeks. In most instances a good result can be expected.

Fractures of the floor of the acetabulum occur from force applied to the greater trochanter in line with the neck of the femur. If the floor of the acetabulum cannot resist the force, it bursts inward, usually splintering from the pubis and separating near the junction of the acetabular portions of the ilium and pubis. Most often the iliac portion of the acetabulum remains in place while the pubic portion is shoved inward. Occasionally both iliac and pubic portions are displaced inward and in very rare instances the pubic portion remains in place and the iliac portion pushes outward.

If there is a stellate type of fracture with no appreciable amount of displacement, balanced Russell traction may be applied for four to six weeks with moderate traction on the injured side and light traction on the other leg. The results in such patients are usually good although, if there is a fracture across the weight bearing portion of the acetabulum, disabling arthritis is apt to eventuate.

When moderate inward displacement of the acetabular fragments occurs heavy skeletal traction for five or six days with a wire through the lower femur or upper tibia, followed by moderate traction, is indicated. The leg should be in about 45 degrees of abduction and slight flexion at the hip. If after several days, x ray examination shows that the displaced acetabular floor has not returned to its normal location open reduction should be done. Surgical approach is made by an incision along the iliac rim near the anterosuperior iliac spine. By dissecting subperiosteally against the inner surface of the ilium and the pubis, the region of the acetabulum can be reached. The fragments are manipulated into place and fixed with wire or screws. Heavy traction is again continued for several days before moderate traction is used.

Grossly displaced acetabular fractures, in which the head of the femur has remained driven into the pelvis, require heavy skeletal traction for several days and, if reduction is not satisfactory and the head of the femur has not returned to its normal position, manipulation with the patient under anes-

thesia may be tried. Caution must be exercised not to displace further the fragments and cause intrapelvic damage. If the floor of the acetabulum does not follow the head of the femur into good reposition, open reduction as described above should be done. This likewise should be followed by heavy traction.

Traction outward in line with the neck of the femur by applying a sling or adhesive traction to the upper inner thigh combined with longitudinal traction is occasionally useful. Some surgeons prefer to use a pin or a screw through the greater trochanter fastened to a lateral traction device. Others find a well leg type of traction helpful. Regardless of the apparatus traction should be directed outward at 45 degrees or a little more.

Severely displaced acetabular floor fractures, even when adequately replaced, carry a doubtful prognosis (Fig. 76). Disabling arthritis is prone to intrude and it is wise to keep in mind that a hip fusion operation may be necessary later.

Fractures of the Pelvic Ring. The pelvic ring consists of a right and left pubis, ischium and ilium, and a single sacrum. The coccyx, while attached below the sacrum, carries no body weight and is not an integral functioning portion of the pelvic ring. The ring, made up of its various bony components transmits all the body weight from above to the lower extremities. Pelvic ring fractures may be divided into three types: incomplete fractures of the ring, complete fractures without displacement and complete fractures with displacement.

Incomplete fractures cause little trouble. This type is represented by isolated fracture of the superior ramus of the pubis, partial fracture in a vertical direction into the wing of the ilium, isolated fracture through the ramus of the ischium and isolated fracture of the inferior ramus of the pubis. In no instance is the pelvic ring entirely broken. Because of the strong enveloping muscles and ligaments, little displacement occurs and treatment consists of bed rest for one or two weeks followed by limited activity for another two weeks.

Complete fractures of the pelvic ring without displacement may be single or multiple. These are represented by fractures through both rami of the pubis, unilateral or bilateral (Fig. 77) fractures through the sacroiliac region on one side combined with fracture of the ischium, fractures through the region of the symphysis pubis either alone or com-



Figure 76. A, Fracture of acetabular floor with ischial portion displaced inward. Head of femur also remains displaced inward. B Condition of "central dislocation of hip" one year after injury. Note persistent displacement of head of femur and also complete obliteration of joint space. The patient has a painful hip and marked limp.

bited with fractures in or near the sacroiliac joint on one side, and fractures through the ilium. These are fairly common examples, but any single fracture or combination of pelvic fractures which severs the pelvic ring, but does not cause recognizable displacement, may be treated the same way. Patients having single fractures need only be put at bed rest for about three weeks, then be permitted limited activity for three more weeks. Comminuted fractures of the ring, in which there are two or more breaks, particularly if the combination is that of fracture near the pubic symphysis or across both pubic rami with fracture in or near the sacroiliac joint, require special attention. They must be guarded so that displacement does not occur. For patients having these fractures it is wise to use balanced traction with the leg on the uninjured side suspended without traction and with the leg on the side having the pelvic ring fractures pulled distally by moderate traction weights. The danger of displacement lies in the strong upward pull of the muscles, so that steps must be taken to keep the portion of the ring that has been freed of its support from becoming displaced proximally. Traction is continued for three or four weeks then bed rest for another two weeks, before permitting the patient to resume limited activity.

The third type, complete fracture of the pelvic ring with displacement, is a very serious type of injury. While visceral damage may occur in association with other types of fractures, in this type it is fraught with most serious consequences. There may be many combinations of fractures of the

components of the pelvic ring in this type of injury the most representative being fracture in or near the symphysis pubis associated with fracture in or near the sacroiliac joint on the same side (Fig. 78). This is termed the double vertical fracture of Malgaigne. The strong upward pull of muscles displaces the separated portion of the ring superiorly and, according to the force applied, the loose fragment may either be pushed outward, causing separation in the region of the symphysis pubis, or inward, causing overriding in the symphysis region. Shock is frequently dangerous to life and if the crushing force was severe enough to



Figure 77. Fractures of superior and inferior rami of pubis without significant displacement (anteroposterior view). The pelvic ring, though broken, had no displacement and healing occurred without trouble.

reduction and internal fixation were required. Every effort should be made to keep the acetabulum as near normal as possible. After removal of balanced Russell traction the patient is permitted to exercise in bed for one week and is then given crutches for two weeks. In most instances a good result can be expected.

Fractures of the floor of the acetabulum occur from force applied to the greater trochanter in line with the neck of the femur. If the floor of the acetabulum cannot resist the force it bursts inward, usually splintering from the pubis and separating near the junction of the acetabular portions of the ilium and pubis. Most often the iliac portion of the acetabulum remains in place while the pubic portion is shoved inward. Occasionally both iliac and pubic portions are displaced inward and in very rare instances the pubic portion remains in place and the iliac portion pushes outward.

If there is a stellate type of fracture with no appreciable amount of displacement, balanced Russell traction may be applied for four to six weeks with moderate traction on the injured side and light traction on the other leg. The results in such patients are usually good although, if there is a fracture across the weight bearing portion of the acetabulum, disabling arthritis is apt to eventuate.

When moderate inward displacement of the acetabular fragments occurs, heavy skeletal traction for five or six days with a wire through the lower femur or upper tibia, followed by moderate traction, is indicated. The leg should be in about 45 degrees of abduction and slight flexion at the hip. If after several days, x-ray examination shows that the displaced acetabular floor has not returned to its normal location, open reduction should be done. Surgical approach is made by an incision along the iliac rim near the anterosuperior iliac spine. By dissecting subperiosteally against the inner surface of the ilium and the pubis, the region of the acetabulum can be reached. The fragments are manipulated into place and fixed with wire or screws. Heavy traction is again continued for several days before moderate traction is used.

Grossly displaced acetabular fractures, in which the head of the femur has remained driven into the pelvis, require heavy skeletal traction for several days and, if reduction is not satisfactory and the head of the femur has not returned to its normal position, manipulation with the patient under anes-

thesia may be tried. Caution must be exercised not to displace further the fragments and cause intrapelvic damage. If the floor of the acetabulum does not follow the head of the femur into good reposition, open reduction as described above should be done. This likewise should be followed by heavy traction.

Traction outward in line with the neck of the femur by applying a sling or adhesive traction to the upper inner thigh combined with longitudinal traction is occasionally useful. Some surgeons prefer to use a pin or a screw through the greater trochanter fastened to a lateral traction device. Others find a well leg type of traction helpful. Regardless of the apparatus, traction should be directed outward at 45 degrees or a little more.

Severely displaced acetabular floor fractures even when adequately replaced, carry a doubtful prognosis (Fig. 76). Disabling arthritis is prone to intrude and it is wise to keep in mind that a hip fusion operation may be necessary later.

Fractures of the Pelvic Ring. The pelvic ring consists of a right and left pubis, ischium and ilium, and a single sacrum. The coccyx, while attached below the sacrum, carries no body weight and is not an integral functioning portion of the pelvic ring. The ring, made up of its various bony components, transmits all the body weight from above to the lower extremities. Pelvic ring fractures may be divided into three types: incomplete fractures of the ring, complete fractures without displacement and complete fractures with displacement.

Incomplete fractures cause little trouble. This type is represented by isolated fracture of the superior ramus of the pubis, partial fracture in a vertical direction into the wing of the ilium, isolated fracture through the ramus of the ischium and isolated fracture of the inferior ramus of the pubis. In no instance is the pelvic ring entirely broken. Because of the strong enveloping muscles and ligaments, little displacement occurs and treatment consists of bed rest for one or two weeks followed by limited activity for another two weeks.

Complete fractures of the pelvic ring without displacement may be single or multiple. These are represented by fractures through both rami of the pubis, unilateral or bilateral (Fig. 77) fractures through the sacroiliac region on one side combined with fracture of the ischium, fractures through the region of the symphysis pubis either alone or com-

mon but occur more frequently in women than in men. Displacement usually is minor but, if present, digital pressure in the rectum may effect some reduction of angulation. Adhesive strapping across the buttocks or a snug binder may give comfort. A supporting low-back belt is of some value in the relief of symptoms. Unless pain is present there is no reason to enforce bed rest. Occasionally severe injury to the sacrum may damage the lower sacral nerves and cause incontinence and local anesthesia.

Fractures of the coccyx are rare but painful. Adequate x-ray visualization is difficult and interpretation of the x-ray films is frequently a matter of conjecture. When fracture does occur displacement is usually slight, but in all patients having suspected fractures a rectal examination should be performed and, if displacement forward is found, efforts to reduce the forward angulation by digital pressure should be made. The treatment is the same as that used for fractures of the lower sacrum. In addition to support across the buttocks, the following measures are useful in relieving pain: an air pillow or ring to make sitting less painful, warm baths, and medication to keep the contents of the rectum soft so that straining at defecation may be minimized.

Symptoms of pain in the region of the coccyx may persist for many months following fracture but most cases of coccydynia are not due to fracture. Surgical removal of the coccyx to relieve pain is frequently in vain, because pain in the region may persist, may return or may increase after surgery. Certainly one should not resort to removal of the coccyx until more than a year has passed after fracture and all conservative measures to relieve pain have been exhausted.

FRACTURES OF THE FEMUR

Fractures of the femur may be divided into six types: fractures of the head of the femur, fractures of the neck of the femur, intertrochanteric fractures, trochanteric and subtrochanteric fractures, fractures of the shaft of the femur and fractures of the lower end of the femur.

The necessity of transmitting weight through the bones and joints of the lower extremities influences the conformations and functions of the joints and the shape of the bones. In the shoulder joint the head of the humerus fits against a shallow rim of fibrocartilage which encircles a flat disk of bone which acts as a buttress. Only a very small

area of the head of the humerus is ever in contact with buttress bone at any one time and when the shoulder is performing its most specialized functions the entire joint, with its ligaments and muscles is loosely held together so that maximum range and rapid changes of motion may be attained. On the contrary the hip joint has stability as one of its major attributes. When it is performing its most specialized functions it requires firm transmission of weight from one supporting bone to another weight transmitting bone. This necessitates relatively broad bone contact across joint surfaces and, in addition, it requires strong ligaments, muscles and tendons enveloping the joint in order to keep the articular surfaces within strict limits of motion so that the forces and pressures accompanying weight transmission can be confined to act only on those areas of the articular cartilages which are developed to sustain weight bearing without premature wearing of the cartilaginous surfaces. For this reason the hip joint has a much deeper socket than has the shoulder joint and relatively little leeway can be permitted in alterations of the opposing articular surfaces if smooth, durable function is to be expected. In treating fractures of the lower extremities it therefore becomes of prime importance to preserve or restore anatomic relationships to the highest degree possible.

Fractures of the Head of the Femur. Fractures of the head of the femur in which a portion is completely separated from the rest of the head, occur infrequently. Sometimes a violent force pushing the femur posteriorly shears off a portion of the head at the point where pressure is brought against the posterior rim of the acetabulum (Fig. 79). More frequently the posterior portion of the



Figure 79. Fracture of head of femur and of acetabular floor (anteroposterior view). This combination carries poor prognosis for function.



Figure 78. A, Markedly displaced fractures of pelvic ring, principally of the double vertical type (anteroposterior view) associated fractures including the upper shaft of the femur. The patient had profound shock. B, Partial reduction of severe pelvic ring fractures. In order to establish effective strong traction on the pelvis it was necessary to plate the fracture of the femur. The patient has no pain and only a slight limp. He plays football and participates in all other sports.

cause marked displacement, blood vessels, nerves and viscera in the region may be hopelessly damaged.

It is important to restore something approaching anatomic relationship if possible, although it is surprising to see what good function frequently results in patients in whom marked displacement was never corrected. Many patients retaining displacement subsequently develop pain and disability. In the female, it is necessary to restore the pelvic ring as accurately as possible so that difficulty in parturition may be minimized. The most useful method of reducing displaced pelvic ring fractures is continuous skeletal traction through the employment of a wire in the lower end of the femur or in the upper end of the tibia. Strong traction with heavy weights is indicated at the start, but if later x ray study reveals that the upward displacement has been corrected, the traction load may be reduced. In order to keep the heavy traction effective, it is often necessary to tip the foot of the bed well up in the air and pass a sheet between the legs, placing it against the perineum on the uninjured side and fastening it to the head of the bed. This provides good counter traction. It may also be necessary to place another sheet or a binder around the body and secure it to the bed frame on the uninjured side to keep the body straight and the traction functioning to maximum advantage. The use of pelvic hammocks or slings whereby the pelvic region is elevated from

the bed by attachment of the sling to the overhead frame is not as general as it was a few years ago. It still has a place in the treatment of fractures which are widely separated at the symphysis region, but it must be watched carefully so that displacement in the opposite direction does not occur. Experience has shown that the use of overhead pelvic slings frequently either checks the efficiency of longitudinal traction or causes even more displacement than existed previously. A snug binder around the pelvis may bring about improved position of fragments as well as maintenance of reduction. In addition, the patient often feels better with the support of a binder. In a few difficult fractures, well-leg traction applied by the method of Hoke or Roger Anderson may be effective when reduction is not accomplished by ordinary skeletal traction. Comminuted pelvic ring fractures, like practically all pelvic fractures, heal rapidly. Traction for four to six weeks followed by recumbency for three or four weeks is a good program.

Fractures of the Sacrum and Coccyx. The upper three segments of the sacrum are an integral part of the pelvic ring. The most common fracture of the lower sacrum is a transverse one at the level immediately below the third sacral segment. In this region the protection of the almost rigid sacroiliac joint is absent and the bone is most vulnerable to forces which may cause it to snap. Fractures of the lower sac are unco

and frequently more than ten years. This fact led Phemister and his associates to attempt to hasten replacement by inserting a bone graft through the viable neck into the necrotic head. Evidence shows that replacement is hastened by such method, but the time of complete resubstitution, so that safe weight bearing can be resumed, is still several years. It is difficult to keep persons in the advanced age group walking on crutches for several years and the wisdom of such a procedure may be in doubt. This fact has led many surgeons to discard the necrotic head as soon as definite evaluation of the condition can be made, which is usually between six and eighteen months. Some surgeons and some radiologists profess to be

able to assess the viability of the head before six months but the general opinion is that action taken on such assessments may lead to numerous mistakes. One may strongly suspect aseptic necrosis at three months in some patients and be quite correct but in general it is safer to make definite evaluations at a later date.

In the presence of a nonviable head and nonunion through the fracture area, one of several procedures may be employed. The insertion of a bone graft through the viable neck with the added agent of immobilization through the employment of multiple nails may be tried. Such operations, while frequently successful take a long time before weight bearing can be safely permitted.

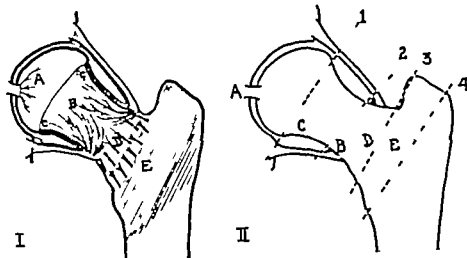


Figure 81 I, Sources of blood supply for the femoral neck. A, From the ligamentum teres, scant and sometimes missing; B, from the capsular attachments, numerous large vessels, C, from the capsular reflection and synovial, small short vessels, D nutrient vessels, numerous and good sized, E, from the extensive muscle attachments in intertrochanteric region (shaded area). In the intact femur these vessels anastomose freely II (Compare with I.) Effect of fracture on blood supply to the proximal fragment. Assuming the fracture to be complete in each instance, the proximal fragment retains after Fracture 1—blood supply A only Fracture 2—blood supply A plus C. Fracture 3—blood supply A plus B plus C plus D Fracture 4—blood supply practically intact.



Figure 82. A, Recent fracture of femoral neck. Head in moderate varus position. B Nonunion of femoral neck fracture. Aseptic necrosis of femoral head revealed by increased density of bone as compared to surrounding bone. Tri-flanged nail extruded and was removed.



Figure 80 A, Fracture of head of femur and posterior dislocation of hip (anteroposterior view). A portion of the head of the femur has remained in the acetabulum. B, Condition of fracture-dislocation of hip one year after open reduction and removal of fractured fragment of head of femur. The patient has marked pain and disability.

acetabulum is broken, but when it remains intact the head itself may break and leave a portion in the acetabulum while the rest of the head continues posteriorly with the neck and shaft and becomes dislocated (Fig. 80). There are numerous variations of fractures of the head, but they seem to have one feature in common and that is the rather extreme disability which results. It may be possible to bring about manipulative reduction or it may be necessary to open the hip joint widely in order to effect reduction of a dislocation. If the hip is opened, it seems wise to remove the isolated fragment from the joint, but then a rough surface is left to articulate against the acetabulum. The remaining head may be smoothed and, if it appears worthy of trial, nothing more need be done. If the remaining head is jagged and comminuted, some type of arthroplasty or replacement should be considered or a hip fusion may be judged the best solution. In practically all instances in which the remaining portion of the head reveals comminution, the prognosis for good, painless motion is very questionable. It is advisable to anticipate an unsatisfactory result unless some type of arthroplasty or fusion is performed.

Fractures of the Neck of the Femur There is little question but that fractures of the neck of the femur continue to be the most speculative and controversial of all fracture injuries. Entirely within the last half century the prognosis has changed from one of complete hopelessness for union of such fractures to anticipation of between 60 and 80 per cent solid bone unions. This has been

accomplished principally by the recognition that firm immobilization of fractures of the neck of the femur is the primary requisite for promotion of solid union of viable fragments. It has been known for some years that the blood supply to the head of the femur is rather precarious in the event of injury (Fig. 81). Fractures of the neck of the femur frequently cause sufficient damage to the blood vessels to impair seriously the circulation to the head and bring about a gradual aseptic necrosis (Fig. 82). This necrosis progresses slowly and invasion of the necrotic areas with new bone-forming elements starts rapidly. The two processes proceed simultaneously but the new bone-forming elements must grow upward from the viable neck, so it is only logical that the areas of bone close to the articulating surfaces of the head are the last to be revascularized and replaced with living bone. This process of revascularization and replacement with living bone appears to vary considerably as to its speed of occurrence. There is no doubt that replacement follows a definite time schedule which is under control of natural conditions, but there are so many variables in the assessment of such conditions that it is almost impossible at the present time to predict the speed of replacement or the time of completion of restoration of good bone. It may be stated that in fractures of the neck of the femur in the age group in which they are most common, namely in persons between sixty and seventy years old, replacement of necrotic bone with good bone in the head of the femur is usually a matter of more five years



A

B

Figure 85. A, Aseptic necrosis of head following femoral neck fracture. The multiple pins have been removed. Nonunion is evident. B, Necrotic head removed. An Escher prosthesis has been inserted.



Figure 86. Jodet type prosthesis in place. The plastic head occupies the acetabulum but does not show in x-ray film.

Subcapital fractures and vertical fracture lines through the neck seemed to indicate eventual bad results. Valgus position, either prevailing immediately after fracture or obtained in reduction, was considered a forerunner of good results. It now appears that the crux of the problem is not the position of the fragments or the angle of the fracture line, but the efficiency of the immobilization of the fragments. Subcapital fractures and vertical line fractures through the neck are difficult to immobilize securely while valgus fractures are more easily immobilized. Evidence now tends to show that true anatomic reduction with secure immobilization offers the best opportunity for successful results in the presence of a viable head.

Reduction of most fractures of the neck of the femur can be obtained by extension, moderate abduction and marked internal rotation. Occasionally when the neck of the

femur on the shaft side is displaced backward in relation to the head, it may be necessary to flex the thigh and knee to right angles and lift upward before resorting to extension, abduction and internal rotation. Ordinarily in the aged, a spring scale reading of between 40 and 50 pounds is sufficient to maintain reduction. This position may be maintained on a fracture table or by a Soutter type apparatus. Nailing or pinning is usually done by x-ray control, care being exercised to obtain satisfactory lateral views. The tri flanged, cannulated nail of Smith Petersen is most popularly employed (Fig. 87). Multiple nails of various sorts are frequently used (Fig. 88). There are many methods employing many types of screws, nails and bolts. All methods, if properly used, seem to give comparable results. In none of the methods does immediate, unassisted walking seem to be safe. The use of wheel chairs and walkers, followed by crutches, with full weight bearing withheld until evidence of beginning union is demonstrated by x-ray film, is a less dangerous procedure. One must study x-ray pictures taken at frequent intervals in order to judge the viability of the head, the maintenance of reduction and the progress of union.

The diagnosis of impacted fracture of the neck of the femur is made with more frequency than is justified by facts. There are numerous impacted fractures, but often the diagnosis is made without thorough x-ray study and the resulting dissolution of the fracture is almost inevitable when impaction is not actually present. In fact, even with



Figure 83. McMurphy osteotomy illustrating a medial shift of the upper shaft of the femur in an attempt to remedy nonunion of a femoral neck fracture with a viable head.

Osteotomies below the neck with medial shifting of the upper end of the shaft to a position under the necrotic head seem to improve the mechanics of weight distribution and the necrotic head seems to revascularize more rapidly (Fig. 83). Frequently however union does not occur between the neck and the head and the condition is not materially improved. Other forms of reconstruction are employed using the upper end of the shaft placed directly into the necrotic head after moving the muscular attachments of the greater trochanter distally. None of these methods is successful in a significantly high percentage of patients to indicate preference. The same methods are used when there is nonunion of a viable head. In these patients a higher percentage of successful results ensues, but there is still little choice between the various operations.

Because of the numerous failures of the above operations to produce painless, functioning hips for many patients in whom necrotic heads are present, metal replacements and metal cups or caps have become popular. Because of the usual reduction in size of the neck of the femur the placement of a metal cup on the stub of the neck does not produce good mechanical results and leads to instability (Fig. 84). The use of a cup on the end of a trimmed shaft, after moving the trochanteric muscular attach-

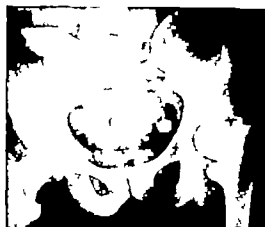


Figure 84. Smith-Petersen cup on fractured neck of femur. Instability due to short neck and varus position.

ments distally gives only fair stability and frequently limited motion in many patients. At present, the popular attack on the problem is removal of the necrotic head and replacement with an intramedullary type of stem prosthesis (Fig. 85). Previously a straight short stem type of prosthesis was used extensively (Fig. 86). While this seemed to work well for a short time, many of the stems did not maintain their original valgus positions, which resulted in unsatisfactory unstable varus positions. The intramedullary stem type prosthesis comes in many models and has many names, indicative of the fact that none is completely satisfactory and that no one model is significantly better than another type. The complications of such prostheses are multitudinous, yet there is gradually growing a substantial group of aged patients who, a few weeks after insertion of a prosthesis, walk with little pain and only moderate limp. When successful, the operations have the advantage of enabling the aged patients to walk in a shorter time than by any of the other procedures and, while the procedures may not stand the test of time, these aged patients frequently do not have a great deal of time to spend. The best advice is that each patient be judged individually and that all of the recognizable variables be taken into consideration before a plan of action is instituted. One should not become dogmatically addicted to one type of procedure.

With all possible complications in mind, treatment of fresh fractures of the neck of the femur takes on added significance. It has been thought that the position of the fracture or the angle of the fracture line entered into the prognosis of possible union.

offers the slightest chance of surgical survival, nail or pin fixation should be used.

Some surgeons, conscious of the appreciable number of nonunions which result with casual nailing in femoral neck fractures, prefer to remove the fractured heads immediately and substitute prostheses, others per-

form immediate osteotomies. In view of the relatively high percentage of unions obtained by adequate nailing and the uncertain results of prostheses and osteotomies it does not appear that such procedures, done immediately have sufficient percentages of success to justify their adoption.

Intertrochanteric Fractures. Any fracture or combination of fractures which interrupts the continuity of bone between the greater and the lesser trochanters may be termed intertrochanteric (Fig. 89). Such fractures are often found in combination with other fractures through the neck of the femur and with fractures of the subtrochanteric region. A great variety of intertrochanteric fractures occurs. In some, merely the lesser trochanter is broken off while, in others there are six or more complete fracture lines between the trochanters. Displacement may be slight or severe. It is no longer necessary to describe in detail the diagnostic differences of various types of fractures in clinical examinations. Whenever the possibility of a fracture exists today, an x ray study is indicated. Fractures are no longer diagnosed by clinical observations. Now even in the most remote hamlets and close to the battle lines in times of war x ray facilities are available.

The treatment of intertrochanteric fractures differing from that of femoral neck fractures may be successful either by opera-



Figure 89. Intertrochanteric fracture illustrating various position and comminution. Note fracture of lesser trochanter.

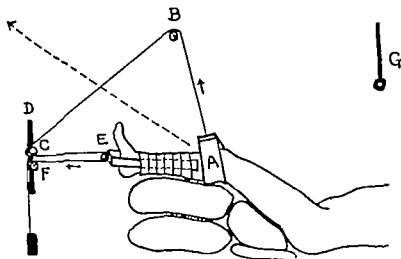


Figure 90. Russell traction. The thigh and leg are supported on pillows. A is a sling underneath the knee and upper calf. From this a rope passes through an overhead pulley B. Skin traction is applied to the leg, and incorporated in the spreader is a pulley E. Attached to the foot of the bed is a board holding a double pulley CF, or two pulleys close together. The traction cord from B passes through one side of the double pulley C, then through the pulley on the spreader E, and back through the other side of the double pulley F. This is the traction weight. The pull is a multiple of the weight, because of the double-pulley arrangement. The resultant of the two pulls is indicated by the large dotted arrow. Lowering the level of the pulley CF or placing the pulley B nearer the foot of the bed will alter the pull toward the horizontal. Raising the level of pulley CF or shifting pulley B toward the head of the bed will alter the pull toward the vertical. G is an overhead trapeze for use by the patient in shifting his position, in helping him to sit more or less upright and in making himself to use a bed pan, during bed changing, etc. Whereas skin traction is shown in this diagram, it is now felt that Kirschner wire traction through the fibula and tibia 1 inch above the ankle is preferable.

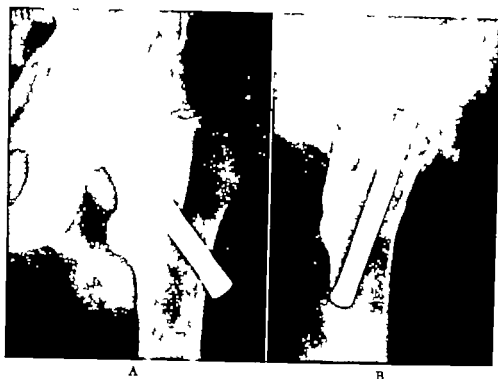


Figure 87 A, Smith-Petersen tri-flanged nail in place as fixation device for intracapsular fracture of the neck of the femur (anteroposterior view) B Lateral view of tri-flanged nail holding the fractured neck of femur fragments in position.



Figure 88 Femoral neck fracture. Fixation with Soefeld-Potts nails.

good impaction and bed confinement, the fracture fragments not infrequently fall apart. Such cases of failure become tragedies, because, subsequently nailing often does not result in promotion of union. The part of wisdom would seem to be primary nailing even in impacted fractures, for the

patient can then be permitted much more liberty of motion in and out of bed and the fear of late disruption of the so-called impacted fragments is minimized. Needless to state there is no place today for the treatment of neck fractures by casts or sandbag positioning. If the condition of the patient

markedly upward by its strong muscle attachment, it is probably the part of wisdom to perform an open reduction and maintain fixation with screws or small nails. One can then permit crutch walking in a few days. Markedly comminuted fractures of the greater trochanter are treated by keeping the limb abducted and extended until a safe degree of union exists.

Subtrochanteric fractures are often transverse and occur in combination with intertrochanteric or neck fractures. When iso-

lated, or when they occur in combination with other fractures which are amenable to treatment by nail plate fixation, they are treated by the same method (Fig. 93). The only difference is that the plate must be long enough to provide adequate support below the subtrochanteric region. These fractures may be treated by traction in younger adults, but often they resist reduction by any means short of open methods. If satisfactory reduction can be obtained in younger adults, continued traction until union occurs is an excellent method of treatment.

Fractures of the Femoral Shaft. The past ten years have seen what amounts to a revolutionary change in the treatment of femoral shaft fractures. Fifteen years ago Kuntscher started to popularize a method of intramedullary support for femoral shaft fractures and this method has made great strides. True it is that Rush and his followers had been using a type of intramedullary rod fixation for many years, but the method had not been widely adopted. The exigencies of the war years, the advances in metal alloys and the improvements of operative techniques combined to bring the method to the foreground. Numerous complications occurred and still prevail, but, in spite of difficulties, intramedullary fixation continues to



Figure 92. Recent fracture, top of greater trochanter. Displacement slight.

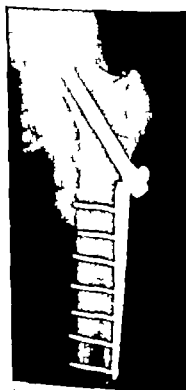


Figure 93. Subtrochanteric fracture treated by open reduction and fixation with McLaughlin nail-plate device.



A

B

Figure 94. A, Kuntscher intramedullary nail fixation for fracture of shaft of femur (anteroposterior view). Applied retrograde. B, Lateral view of intramedullary nail in femur. Callus frequently forms rapidly but firm union still requires nine to twelve months in most cases.

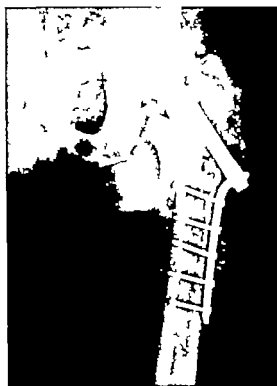


Figure 91 McLaughlin nail-plate fixation for intertrochanteric fracture.

tive or nonoperative methods. The chance of nonunion of intertrochanteric fractures, while present, is slight. Most intertrochanteric fractures progress to firm bony union and the only problem from the mechanical viewpoint is that of obtaining and maintaining satisfactory reduction of the fractures. Even this problem is not difficult. Traction, either of the Bucks type or by the Russell method (Fig. 90) or even by skeletal application, customarily brings about good reduction and satisfactory maintenance of position to permit good healing. In patients of the younger age group, treatment by traction may well be the method of choice. Certainly in children it is practically never necessary to resort to anything but traction for the treatment of intertrochanteric and shaft fractures of the femur. In persons of the military age group traction is still the method of choice, but as one approaches the older age group the dangers of long bed confinement necessitated by traction methods make one question the propriety of this type of treatment. It is well recognized that persons of advanced years react poorly to long confinement in bed, so, if a method of treatment which has a fair assurance of success and which permits the patient to get up in a wheel chair and then become ambulatory in a short time can be perfected, it should be

preferable to long traction treatment. Such a method has been successfully utilized in recent years and is particularly applicable to the treatment of the aged person. In its most common form, the method utilizes a triflanged or flat femoral neck nail to which is attached either integrally or by screw connection a plate which extends distally along the lateral side of the shaft for a distance of about 4 inches below the greater trochanter (Fig. 91). The firm attachment of the plate by screws through the bone provides strong support for the nail or blade which extends across the fractured fragments of the intertrochanteric region into the neck of the femur. Unless the fragments are very soft or the comminution extreme, the combination of nail or blade and plate gives adequate support. A few days of traction following the insertion of the intertrochanteric fixation apparatus are frequently employed in order to let the fresh fracture fragments adjust and become established in their positions. After a few days the patient is permitted up in a wheel chair and, if of sufficient physical and mental vigor he is soon allowed to use a walker and then crutches. Most intertrochanteric fractures become well united in from eight to twelve weeks. The common position for application of the nail plate is extension, moderate abduction and neutral rotation of the thigh. The use of a fracture table so that traction may be maintained during fixation is a requirement. Very frequently the lesser trochanter is completely separated from the shaft and occupies a place medial and proximal to its anatomic position. Seldom does reduction of the fractured lesser trochanter occur but experience has shown that bone usually bridges the gap to the displaced trochanter and function is satisfactorily restored.

Trochanteric and Subtrochanteric Fractures. Isolated fractures of the greater and lesser trochanters occur although single fracture of the lesser trochanter is not frequent. It is seldom necessary to perform open reduction and replacement of fractures of the lesser trochanter although some surgeons advocate such procedure when displacement is marked. In most instances, rest in bed with the thigh flexed and adducted and in neutral rotation suffices for treatment. Fractures of the greater trochanter are common and variegated (Fig. 92). They may be of a type that shears off the greater trochanter as a whole unit or they may splinter the trochanter into many pieces. When the greater trochanter is sheared off and pulled

viewing of the fractured bone ends. The fracture area is opened and customarily the rod or nail is inserted in a retrograde manner by starting it upward in the proximal fragment and permitting the upper end to emerge from the skin in the buttock area, then, when the fracture is reduced, the nail is driven downward to about 1 inch above the intercondylar notch. The nail should be only moderately snug, if the diameter is too large to pass the isthmus and wedging occurs, a most difficult situation of not being able to move the nail in any direction ensues. It is remarkable and discouraging to learn how firmly a nail may become jammed when improper choice of equipment has been made. Two general types of fairly rigid nails are in common use the clover leaf of Kilntschner and the diamond shape of Street. In the southern part of the United States the more flexible Rush rods enjoy considerable popularity (Fig. 96). All rods should be left in place until there is x ray and clinical evidence of firm bony union. Usually one year is considered a minimum time for removal of rods. There is usually no difficulty in extracting the rods after a year of use, even though they may have been fitted quite snugly at the time of fixation.

Fractures of the Lower End of the Femur. Condylar and supracondylar fractures in the lower one-fourth of the femur can often be treated by either adhesive traction or insertion of a skeletal pin through the upper or

lower end of the tibia. A frame holding the thigh upward at 45 degrees and the leg horizontal is useful. After adequate reduction and about six weeks of maintenance of position by traction a snug, long leg cast fitted well up into the groin may suffice to hold the fragments in good alignment. If satisfactory reduction cannot be had by traction and manipulation, open reduction may be necessary. Fixation with two Rush rods by starting the rods toward the medullary canal and upward from the lateral surfaces of both condyles may work well (Fig. 97). Bending a blade plate to right angles and molding the plate to fit the contour of the shaft then driving the blade across both condyles and screwing the plate to the shaft are useful. Sometimes screws may suffice, or a circular band of steel or wire, which is later removed so that ring sequestra may be avoided can be employed. In most instances good union is obtained in ten to fourteen weeks.

Fractures of the femur which split into the knee joint are frequently troublesome. If satisfactory reduction can be maintained by either adhesive or skeletal traction through the tibia, it is the method of choice. The use of screws, bolts, wires and other foreign materials in the lower femur close to the knee joint, does not encourage good joint function. Occasionally satisfactory reduction is not possible by traction and open reduction must be employed. In such cases, metal should be kept to a bare minimum and only the fewest required screws or bolts to maintain reduction should be used. Subsequent traction and plaster-cast support may be required. It is of course, important to restore proper joint conformation.

FRACTURES OF THE PATELLA

The important feature of patellar fractures is injury to the extensor mechanism of the knee. Actually the patella is a sesamoid bone in the quadriceps tendon and, while very useful for smooth knee joint function, it can be discarded when deemed necessary and joint function is only moderately impaired, although long term results of patellectomy are not as encouraging as might be desired. All efforts in treating fractures of the patella should be channeled into the task of restoring the damaged extensor mechanism and re-establishing a smooth gliding component. When it is possible to repair the fractured patella and restore a smooth articular surface, the result is better than when the patella is removed. Only in comminuted



Figure 97 Application of Rush rods in fracture of lower femoral shaft.

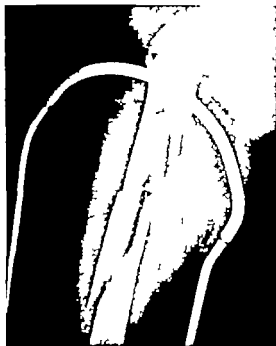


Figure 95 Comminuted fracture of the femoral shaft. Nail fixation is impractical. This is best treated by traction.

gain favor (Fig. 94). Not all femoral shaft fractures are amenable to treatment by rod fixation and, until the definite limits of proper application of such fixation are recognized, errors will continue to occur. An intramedullary rod can only be as large in diameter as the smallest area of the medullary canal of a bone. If one exaggerates the shape of a medullary canal and visualizes it as an hour glass, it can be realized that firm fixation can be had only near the narrow portion of the canal and the efficiency of fixation decreases rapidly as the extremities are reached. For this reason, intramedullary rod fixation is practically useless in the proximal

and distal fourths of the femoral shaft. Fixation is best at the junction of the middle and upper thirds of the shaft, where the medullary isthmus is located. The more comminuted the fractures and the more oblique the break, the poorer will be the case for rod fixation (Fig. 95). Traction for femoral shaft fractures is still an excellent method of treatment, but it suffers from the necessity of keeping the patient confined to bed for eight to twelve weeks with the attendant danger of stiffness of joints, pressure areas, atrophy of muscles and medical and surgical complications of long bed confinement. Young adults usually take bed confinement without too much trouble and children's femoral shaft fractures should practically always be treated by traction. Once again it is the aged who benefit most by methods designed for early ambulation and return to accustomed surroundings. Ideally one should not be influenced by economic or in some instances, military considerations. Still it is necessary to view conditions in their true perspective and, if so considered, the value of being able to apply a rod and get a patient ambulatory with crutches in a week or ten days, in comparison with being confined to bed with traction for two or three months makes a potent argument for rod fixation whenever applicable. The use of plates and screws in such fractures has largely been supplanted by the employment of rods, although, for certain types of fractures, plates may be superior. However it must be warned that two plates facing each other on opposite sides of a bone frequently lead to the tragedy of necrosis and nonunion.

On the North American continent, most intramedullary rods are applied with direct

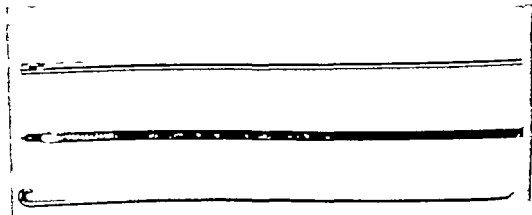


Figure 96. Intramedullary femoral rods. Top, Kirschner nail. Middle, Hansen-Street nail. Bottom, Rush rod.

dately. It usually takes six to ten weeks for safe union to occur and frequently the patella undergoes extensive atrophic changes during that time and has to rebuild itself into a solid-appearing bone over the course of months. On rare occasions a longitudinal fracture will occur in the patella. This seldom requires open reduction unless the split surfaces are widely separated and the longitudinal soft tissue tears are of slight consequence. Frequently the common transverse fractures are not complicated by adjacent tears into the ligaments and separation of the fragments is minimal. In such cases, open reduction is not indicated and cast support suffices.

Whenever the patella is split into more than three large pieces, it seems inevitable that considerable roughness of the articular surface will result and it is considered wise to perform primary patellectomy (Fig 99). When this excision is performed, one should avoid damaging the remaining soft tissue strands which preserve the integrity of the quadriceps-patellar ligament mechanism. If frequently happens the mechanism is severely damaged and appears quite loose and elongated after attempted approximation, it is better to take a tuck in the ligament and tendons so as to provide proper stability.

On many occasions fractures occur only at the proximal or distal portions of the patella where the quadriceps tendon inserts proximally or at the attachment of the patellar ligament distally. Such fractures are of the avulsion type and are frequently comminuted. The rule of restoration of the extensor mechanism as the primary object continues in force in such instances so it is best to remove and discard the small fracture fragments, leaving the main body of the patella, and then carefully repair the tendinous or ligamentous attachments. This may require drilling holes in the outer surface of the patella in order to obtain a firm anchor for the soft structures.

FRACTURES OF THE TIBIA AND FIBULA

Fractures of the tibia may be divided into three types: fractures of the anterior spine of the tibia and of the tubercle fractures of the tibial plateau and fractures of the shaft of the tibia.

Because the blood supply to the shaft of the tibia is apt to be seriously curtailed by fractures, delayed union and nonunion may occur in fractures anywhere in the shaft. However the most dangerous area by far

is the region of the junction of the middle and lower thirds of the shaft.

Fractures of the Anterior Spine of the Tibia and of the Tubercle. Fractures of the anterior spine of the tibia occur and, because of the danger of resulting ligamentous instability as well as malposition of the fractured bone open reduction is indicated when the piece of bone is of enough size to justify operative intervention. This is particularly true when the spine fractures at the base and has marked displacement. Antero-medial incision, extending from the upper pole of the patella and running close to the medial aspect of the patella straight distally to the upper rim of the tibia, then turning laterally to the region of the tibial tubercle, is made. The knee joint is entered and the anterior tibial spine becomes evident. A small hole is drilled through the base of the spine and then a hole is drilled from the region just above the tibial tubercle obliquely upward to emerge into the joint just anterior to the anatomic base of the tibial spine. Wire is passed upward through the main body of the tibia, then through the base of the loose fragment and back through the anterior area of the tibia to the initial insertion area near the tubercle. The wire is permitted to extrude through the skin during closure and then the knee is extended. The wire is then pulled taut and fastened to a button or other material outside the skin. After six or eight weeks, the wire is pulled out. This method usually effects satisfactory replacement of the tibial spine. The posterior tibial spine is seldom fractured.

Fractures of the tibial tubercle occur and are quite painful. The tubercle being the principal anchor of the patellar tendon, is subjected to almost continuous upward displacement force. When definite diagnosis of tubercle fracture is made and one must be careful not to confuse ununited tubercle epiphyses with fracture treatment for an undisplaced fracture consists of immobilization in extension for four to six weeks. If there is displacement of a sizable fragment, open reduction is performed and fixation with a screw or screws is necessary. When considerable comminution exists, the fragments should be removed and the patellar tendon firmly attached to the underlying bone.

Fractures of the Tibial Plateau. The articulating surface of the tibia at the knee joint is known as the tibial plateau. Its two major components are the medial and lateral tibial condyles. Fractures frequently

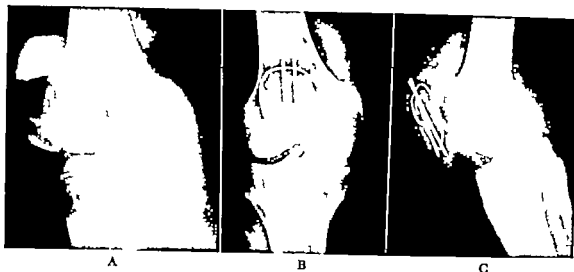


Figure 98 A, Transverse fracture of patella. The wide separation of fragments denotes extensive tears of the lateral expansions of the patellar tendon. Torn lateral expansions must always be repaired with strong sutures. B, Fixation of patellar fragments with wire and threaded pins. Anteroposterior x-ray view. C, Lateral x-ray view. The wire does not touch the articular surface of the patella.

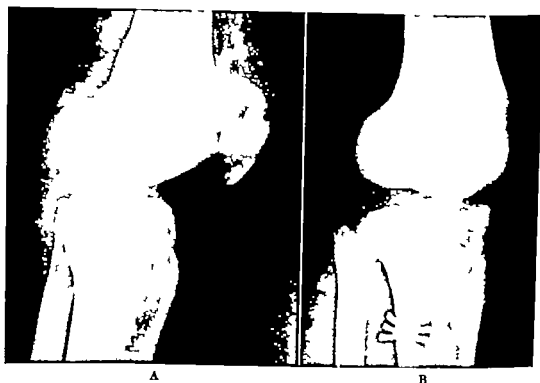


Figure 99 A, Comminuted fractures involving large portion of articular surface of patella. Patellectomy advised. B, Patella excised because of extensive comminution of fracture. Quadriceps tendon firmly sutured to patellar ligament. Lateral expansion tears repaired.

fractures, when it is evident that the multiplicity of articular fractures militates against the restoration of smooth function, should the patella be sacrificed.

Transverse fractures are most common in injuries to the patella. The disabling tears in the ligaments on both sides of the patella must be repaired and the fractured fragments carefully apposed. When it is evident

that good restoration of articular surfaces is possible, holes are drilled from the outer surfaces of the fragments into the fractured surfaces but not into the articular surfaces (Fig. 98). Stainless steel wire is applied in a manner to pull and hold the fractures snugly together and the wire is twisted securely. A posterior splint is applied for only a few days. Quadriceps exercises are started imme-



Figure 102. Severe "bumper" fracture with comminuted comminution of upper end of tibia and fibula. Prognosis for knee-joint function is poor

all the fragments and prognosis of a poor result is foreseen (Fig. 102). Initial knee fusion, however is seldom indicated because clinical trial in such cases has shown that occasionally fairly good joint function may result and last for a considerable time. Of course, the prognosis for long term function is poor and eventual knee fusion should be anticipated.

Fractures of the Shaft of the Tibia. Most single fractures in the upper two-thirds of the shaft of the tibia can be well treated by closed manipulation or by traction, when necessary followed by application of a plaster cast. Very oblique fractures may require traction to prevent shortening, but the more oblique the fracture, the more raw bleeding surface there is available for promoting successful union. In general, it may be stated that the chances of successful union in any given fracture are increased in proportion to the degree of obliquity of the fracture—the more transverse the fracture, the smaller the amount of raw surface there is available for promoting bony union.

Single fracture at the junction of the middle and lower thirds of the shaft of the tibia is usually only moderately oblique and is frequently transverse (Fig. 103). The blood supply to this region is seriously impaired by injury and nonunion or delayed union is common (Fig. 104). All efforts should be bent to secure good apposition and firm immobilization of the fragments. If the patient is in good general health and the fracture is

not compound, open reduction and internal fixation may be the treatment of choice. With oblique fractures in this region one or two screws may suffice for fixation. Up to the present time, a popular method of fixation has been the application of a metal plate to the lateral surface of the tibia. Slotted plates which theoretically permit the screws and bone fragments to move toward each other as the fragments are influenced by strong muscle pull, have acquired many advocates. Under ideal situations, such slotted plates keep the fractures in good alignment while permitting the fragments to be snugly pressed together. If plates are used, they should not be put on the anteromedial aspect of the tibia because of its relatively superficial position, which leads to the danger of breakdown of skin and poor healing. Most surgeons prefer to place their plates on the anterolateral surface of the tibia, although some believe that the posterior surface is better. In both instances, good coverage by muscles is obtained and the dangers of superficial irritation are minimized. Many surgeons have been disappointed with the results of plate fixation for fractures of the tibial shaft and some have

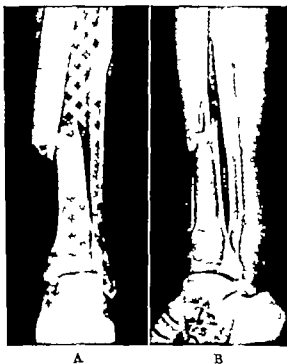


Figure 103. A, Comminuted fractures of lower shafts of tibia and fibula. At this region union is frequently slow. Firm immobilization is required for a long period. Anteroposterior view. B, Lateral x-ray view. Such fractures may be treated by closed reduction and plaster casts, traction followed by casts, open fixation with a plate and screws or by intramedullary fixation of the tibia.

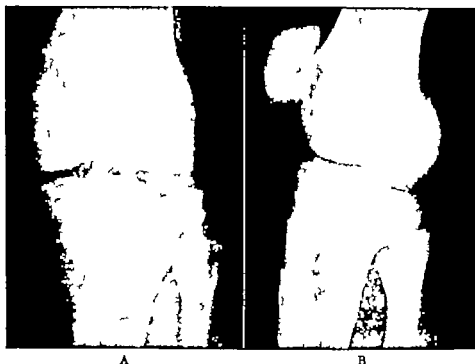


Figure 100 A, Comminuted fracture of lateral condyle of tibia. Anteroposterior x ray view B, Lateral x ray view. Closed reduction by compression made with the hands is satisfactory. Reduction is maintained by a snug plaster cast. A carpenter's clamp could have been used for compression.

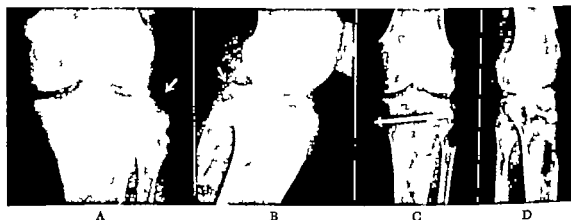


Figure 101 A, Comminuted depressed fracture of lateral condyle of tibia. B Lateral x-ray view. Closed manipulative reduction has been unsuccessful. C, Bone graft from 2 inches down on the tibia has been used as a prop to support the lateral articular surface of the tibia. A fracture bolt is used to hold the fragments together. D Lateral x-ray view. The defect below bolt is the area from which bone for supporting upper articular surface was removed.

extend distally from the region of the anterior tibial spine and extend medially or laterally to break through the cortices at about the level of the tubercle or below. The tubercle is usually left intact. The displacement of condylar fractures may be slight or marked. When displacement is slight, little need be done except elimination of weight bearing to prevent it. When displacement is severe restoration of anatomic position is required if good subsequent function is to be expected. Closed manipulative reduction of slightly displaced

fractures is frequently successful (Fig 100). If displacement is marked, open reduction should be attempted. Provided the fractures are not too comminuted, reposition can usually be obtained by prying the fragments up into place; then immobilization may be secured by long screws or fracture bolts (Fig. 101). Healing requires about ten to twelve weeks but knee-joint motion should be started in one or two weeks. No weight bearing should be allowed until healing occurs. Sometimes the comminution is so great that it is impossible to replace successfully

type and the vertical compression type. Internal and external rotation causes variations of the three types. Frequently a combination is present such as the abduction and external rotation fracture complex.

The important goal in treatment of fractures about the ankle is restoration of the anatomic mortise of the joint. This mortise is formed by the articular surface of the lower end of the fibula laterally and the articular surfaces of the lower end of the tibia superiorly and medially (Fig. 106). The lower projection of the tibia medially forms the medial malleolus and the lower extremity of the fibula forms the lateral malleolus. The talus fits into the mortise formed by the distal ends of the tibia and fibula. This fit must be preserved if full painless rocking motion is to be expected. Proper alignment of the ankle joint with the knee joint must be maintained.

The different varieties of fractures depend upon the position of the foot in relation to the leg when irresistible force is applied to the ankle joint. If the foot is everted beyond the ability of the ankle joint structures to resist disruption or if the foot is fixed to the ground in weight bearing and a strong force is applied to the outer aspect of the lower leg forcing the tibia medially, an abduction fracture may occur (Fig. 107). If a twisting motion is added to the force, a combination

type fracture may eventuate. The rupture of the ligaments and the degree of displacement of the fractures from minor to complete dislocation depend upon the amount of force applied. When vertical compression occurs with the foot in plantar flexion the posterior lip of the tibia may be fractured and, when the foot is in dorsiflexion vertical compression may cause fracture of the anterior lip of the tibia. Falling from a height or having a force suddenly applied upward, as from an explosion, may cause a compression fracture in which the main portion of the tibia breaks from the medial malleolus and the fibula fractures just above the lateral malleolus, while the horizontal portion of the lower tibial articular surface and the talus are pressed together so forcefully that displacement occurs. This may result in the talus being forced upward so strongly as to cause lower tibial shaft fractures and displacement of the talus to a position proximal to the malleoli. At other times the tibia is driven downward into the talus. If adduction or abduction is also a factor even more severe displacement may occur.

Ligaments are of the utmost importance in ankle injuries and efforts to insure repair in anatomic position must not be neglected. Inferior tibiofibular diastasis, if not reduced by manipulation and casting, should be reduced and immobilized by a long screw.

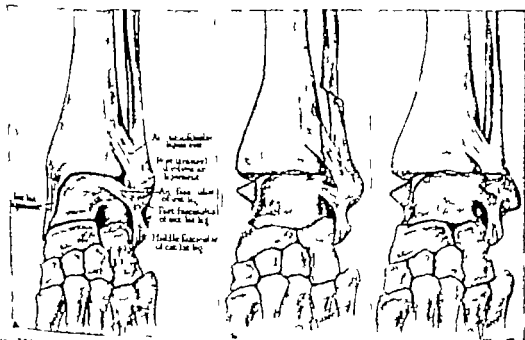


Figure 106. a, Diagrammatic illustration of the ankle joint and its ligaments. Note the position of the tibiofibular ligament. b, Abduction fracture with (1) fracture of internal malleolus; (2) laceration of tibiofibular ligament and separation of tibiofibular articulation; (3) fracture of shaft of fibula, and (4) lateral displacement of astragalus. c, Abduction fracture with intact tibiofibular ligament. Note (1) fracture of internal malleolus, (2) fracture of external malleolus near its base and (3) lateral displacement of astragalus.



Figure 104 A, Old nonunion of the shaft of the tibia under treatment with an onlay bone graft. Anteroposterior x-ray view. B, Lateral x-ray view. Note the atrophy of bone that usually occurs with restricted weight bearing due to nonunion.

turned to intramedullary fixation. The preliminary reports of the results obtained by intramedullary fixation have been encouraging for some types of nails, but sufficient time has not elapsed to permit mature evaluation. The Küntscher type tibial nail has not gained wide acceptance in the United States and the Rush tibial rods continue to enjoy their popularity principally in the South. A newer type of tibial nail by Lottes which is more rigid and provides firmer fixation, is now being given clinical trial in some surgical centers (Fig. 105). If this nail fulfills its early promise, it may in many cases supplant the use of plates and screws.

Markedly comminuted fractures of the tibia are frequently best treated by skeletal traction until sufficient union occurs to permit immobilization in a plaster cast. This often takes about six weeks. The use of the cast is continued until union is solid. There is no advantage in applying large amounts of various types of devices to a comminuted fracture of the tibia just to gain some degree of metal fixation, skeletal traction applied by a wire through the lower end of the tibia or

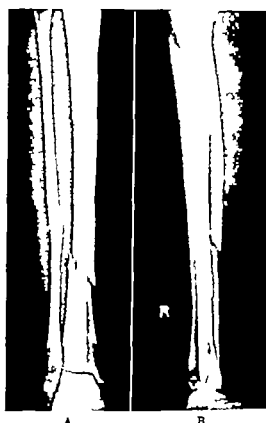


Figure 105 A, Fracture of tibia and fibula treated by Lottes intramedullary nail. Anteroposterior x-ray view. B, Lateral x-ray view showing Lottes nail in the tibia. If union is satisfactory the nail is usually removed at about the end of one year.

just above the calcaneus is a more satisfactory method of treatment.

Fractures of the shaft of the fibula usually occur in conjunction with fractures of the tibial shaft but also occur alone. If a fibular fracture accompanies fracture of the shaft of the tibia, it is usually satisfactorily reduced and immobilized during treatment of the fractured tibia. The shaft of the fibula is not a direct weight-bearing structure and therefore is not of prime importance in a weight-bearing extremity. Often a large portion of the shaft of the fibula is removed for grafting elsewhere and the absence of the shaft seems to make little difference. In patients having an isolated fracture of the fibular shaft, alignment and contact of the fragments is invariably good and treatment consists of relief of pain. A light plaster cast extending upward to just below the knee may be required, although frequently the use of crutches for about one week suffices.

FRACTURES OF THE ANKLE

There are three principal types of ankle fractures: the abduction type, the adduction

nation is graphic. When both malleoli are fractured, the condition is called a bimalleolar fracture and when the posterior lip of the tibia is added the condition is termed a trimalleolar fracture. Sometimes a pos

terior malleolar fracture is the only fracture accompanying a dislocation of the ankle joint.

There has been some difference of opinion as to the necessity for obtaining perfect reduction of posterior malleolar fractures. Usually displacement is upward and produces a condition of the articulating surface whereby the curved upper surface of the talus does not impinge on the displaced fragment although it comes into contact with the back edge of the main tibial fragment. The smaller the fragment the less chance there is of much weight bearing of the talus against a rough edge. Efforts should be made to manipulate the fragment into position by closed means but, in most instances perfect reduction cannot be accomplished. Whenever the posterior fragment is sizable and displaced downward, which is rare, it must be forced upward to its normal position. If this cannot be accomplished by the closed method, open reduction and fixation with a nail or screw are advisable. If, however the fragment is moderately displaced upward and is judged to be no greater than a fourth of the whole articulating surface, it can be left with a fair chance of successful ankle joint function. If the fragment is

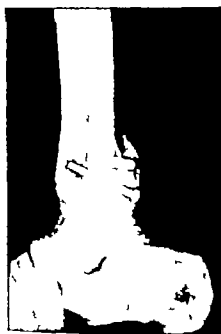


Figure 110. Fracture of the posterior lip of the lower tibia involving half of the articular surface. Open reduction and fixation with screws.



Figure 111. A, Compression fracture of ankle with marked comminution (anteroposterior view). Note skeletal traction applied by wire through calcaneus. B, Compression fracture (lateral view). Because of multiple fractures across the articular surface of the tibia the prognosis for painless motion must be guarded.



Figure 107 Abduction (Pott's) fracture of ankle. Note the transverse fracture of the medial malleolus at the level of the articular surface of the tibia, outward displacement of the lateral malleolus and lateral displacement with tipping of the talus. The inferior tibiofibular ligament is intact.

Abduction and Adduction Fractures. Knowledge of normal anatomy and careful study of x-ray films will assist in planning proper treatment of ankle fractures. It is self-evident that adduction fractures must be brought to normal position or slightly overcorrected and abduction fractures must be corrected in the opposite manner. Twisting and combination-type fractures require more study but the same reversal of direction of force which caused the displacement will usually reduce the major portion of the

displacement. In adduction and abduction fractures it should be emphasized that immobilization in markedly overcorrected positions in plaster casts should be avoided. Such treatment frequently reverses the original deformity and spreads the mortise unduly. Usually the best practice to follow in ankle fractures is to restore normal alignment or use only slight overcorrection. Molding inward over the felt padded malleoli with moderate pressure often tightens the mortise to its original position. Care must be taken not to cause pressure areas.

Adequate x-ray studies must be made for ankle region fractures. Anteroposterior, lateral and oblique views are desirable. The position of the medial malleolus is often a question and if x-ray films show significant displacement of a large fragment of the medial malleolus after attempted closed reduction, it is wise to perform open reduction and fixation with a nail or a screw (Figs. 108 and 109). This is necessary because large pieces of the malleoli, if displaced, endanger the stability of the ankle mortise. Small tip ends of the malleoli do not threaten the mortise and often fail to unite. They are of no great consequence, but occasionally result in some pain and, if it is desired, they may be removed.

The posterior lip of the lower tibial articulating area is now termed, in common parlance, the "posterior malleolus." While not anatomically accepted as yet, the design-



Fig. 108

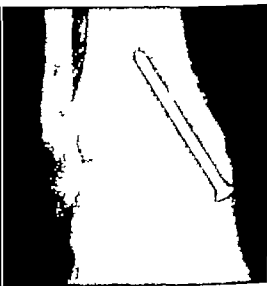


Fig. 109

Figure 108. Isolated fracture of medial malleolus. Because the fracture is at the level of the articular surface of the tibia, the mortise is disrupted. Perfect reposition is desirable. Open fixation with a nail or screw is the safest method of treatment.

Figure 109. Abduction fracture reduced (anteroposterior view). Medial malleolus secured in place with a screw.

flexion fractures, the posterior process or plantar flexion fractures, and body or compression fractures. Actually the posterior process and body fractures are no more common than the avulsion types from the neck or from the medial or lateral borders and about the only warning that need be heeded is that of not mistaking the common anomaly of a congenitally ununited posterior process, or os trigonum, for a posterior process fracture. Strong plantar flexion will, in rare instances, cause the posterior process to impinge against the posterior lip of the tibia and bring about fracture. In such cases, closed reduction can usually be

obtained by dorsiflexion and immobilization in a plaster boot for about six weeks suffices to restore continuity. Body and avulsion type fractures are also rare and require about the same type of plaster boot immobilization.

The most dangerous fracture of the talus is also the most common, namely, that of the neck of the bone. This is a dorsiflexion type of fracture in which the superior surface of the neck of the talus is driven forcibly against the anterior aspect of the tibia and, because the body is locked in the ankle mortise, fracture occurs across the neck in a vertical manner. Occasionally this fracture is associated with fractures of the an-

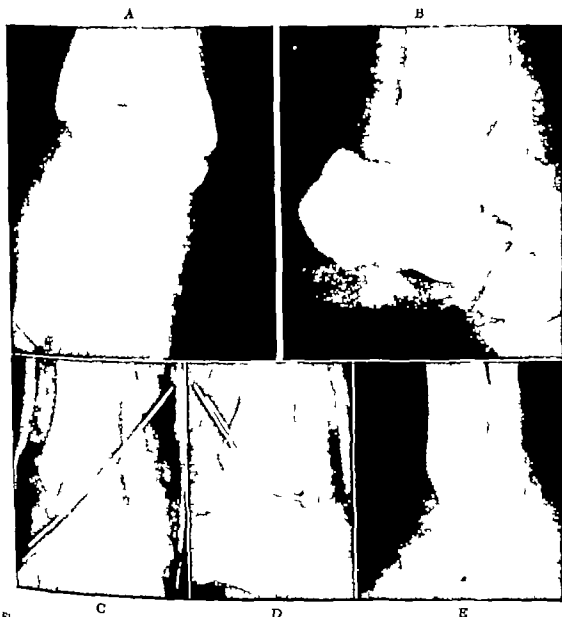


Figure 113. A, Fracture-dislocation of talus (anteroposterior view). The fracture is across the neck of the talus. The body of the talus remains in the mortise of the ankle joint. B, Lateral view showing fracture of the neck of the talus and forward displacement of the calcaneus at the talocalcaneal joint. C, Improved alignment of fragments of the fractured talus maintained by steel pins (anteroposterior view). D, Lateral view showing the position of fragments of the fractured talus, transfixed by pins. Note that some forward displacement of the calcaneus persists at the talocalcaneal joint. E, Lateral view taken later showing the usual aseptic necrosis of the body of the talus resulting from fracture-dislocation. The marked increase in the density of the bone indicates necrosis.



Figure 112. A, Fracture-dislocation of ankle, compression type (anteroposterior view) Tibia driven downward, forward and medially results in marked displacement. B Lateral view of fracture-dislocation of ankle. If reduction is delayed, permanent circulatory damage may occur

greater than a third of the whole articulating surface and does not reduce satisfactorily by closed methods, open reduction by posterior approach close to the Achilles tendon is desirable. Fixation with a nail or screw is sufficient (Fig. 110). The question of open reduction when the fragment is between one-third and one-quarter the size of the whole articulating surface remains a moot point. All ankle fractures that have much displacement before reduction are immobilized after reduction in plaster casts which extend from the toes to the upper thigh. The knee is flexed about 45 degrees. After three or four weeks, the casts are changed and knee-length casts are applied.

Compression Fractures. Compression fractures frequently cause marked comminution of the articular surfaces and guarded prognoses for painless joint function must be made (Fig. 111). Oftentimes it is necessary to resort to an ankle fusion when comminution has been so severe that good anatomic relationships could not be re-established (Fig. 112). Whenever possible, clinical trial of function should be given even though the chances of success appear minimal. Therefore, it is best to obtain everything possible

in the way of reduction of these fractures. Traction by means of a wire above the tubercle of the calcaneus or through the calcaneus is the method of choice. After a few days of traction, manual molding of the fragments may be helpful. A plaster cast is applied when union has progressed to a safe degree; this usually takes about four weeks. One should avoid trying to sort out many comminuted fragments and fix them with screws, wires or plates or other metal devices. These metal devices placed close to the ankle joint in severely comminuted fractures militate against the chances of a successful result.

FRACTURES OF THE FOOT

Fractures of the foot are considered under five divisions: the talus, the calcaneus, the navicular, cuboid and cuneiform bones, the metatarsals, and the phalanges of the toes.

Fractures of the Talus. All fractures of the talus are rare, but the most common fracture with its associated injuries is so disabling that careful attention must be paid to its diagnosis and treatment. It has been the usual practice to divide fractures of the talus into three groups: the neck or dorsal

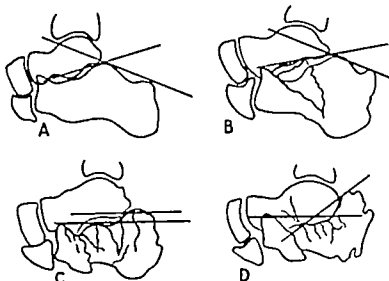


Figure 114 Tracings of roentgenograms of normal and fractured calcanei, showing alterations in the relationship of lines prolonging their superior surfaces. A, Normal B slight defect C, moderate defect, D very marked defect.

of early prognosis, because if we could definitely know the exact location of each fracture line and the degree of displacement present, we would then be able to foretell results with a high degree of accuracy. Whenever there is comminution into the articular surfaces of the calcaneus with displacement of fragments, a guarded prognosis is given. If the displacement is marked, operative fusion of the talocalcaneal, calcaneocuboid and talonavicular joints is indicated. This so-called triple arthrodesis is performed because if such fractures are treated by closed methods, painful arthritis practically always develops.

In general, fractures of the calcaneus can be separated into two large groups, which statistics show to be about evenly divided: those which involve a sizable portion of the articulating surfaces and those which do not involve articulating surfaces to any significant degree. In the first group the prognosis for good recovery following the use of closed methods for reduction is poor while in the second group the prognosis is excellent. The amount of comminution and the degree of displacement are not too significant. The important element is the degree of involvement of the articulating surfaces of the bone. The tuber joint angle formed by a line touching the anterior and posterior articulating surfaces intersecting a line drawn along the superior border of the tuberosity normally measures about 40 degrees (Fig. 114). Decrease of this angle is caused by upward displacement of the tuberosity from fractures, but the extent of decrease of the angle does not necessarily measure the se-

verity of the injury. Badly comminuted fractures with marked displacement may not involve articulating surfaces and, while it may be difficult to effect reduction of the fractures a good result should be expected (Fig. 115). In all these patients efforts should be made to restore the normal length of the Achilles tendon by replacing the tuberosity. If the tuberosity has been com-

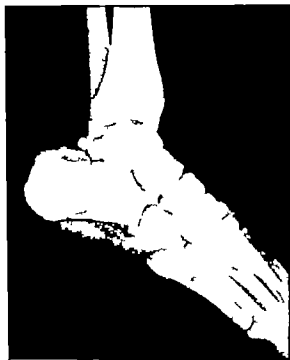


Figure 115. Markedly comminuted fractures of calcaneus with loss of tuber joint angle (lateral view). Fractures did not significantly involve the articular surfaces and after reduction the functional result was good. Note that the tuberosity is displaced upward with the large fragment.

terior lip of the lower tibia, the medial malleolus or the calcaneus. If the dorsiflexion force continues after fracture of the neck of the talus the rest of the foot may be driven forward in relation to the mortise and the anchored body of the talus and dislocation of the talocalcaneal joint, with the calcaneus forward in relation to the talus will occur (Fig. 113). In some instances the force continues even beyond this injury and forces the calcaneus further forward in relation to the body of the talus to the point where the sustentaculum tali becomes locked in front of the medial tubercle of the talus. When the dorsiflexion force is removed and the foot springs back toward a neutral position it is found that there is complete backward dislocation of the body of the talus, which lies rotated and locked with the main portion of the body lying medially and the fractured surface pointing upward and laterally. Frequently the disruption causing this marked dislocation is so violent that the skin below and behind the medial malleolus ruptures and the fracture becomes compound.

In all fractures of the neck of the talus injury to the blood supply of the body of the talus is to be feared. If the fracture occurs across the neck and there has been no subtalar subluxation or dislocation, the blood supply of the body of the talus will not be injured and union should occur without incident. Reduction is usually accomplished by plantar flexion and immobilization in that position for six to eight weeks is indicated.

Whenever subluxation or dislocation of the subtalar joint has occurred, the danger of aseptic necrosis of the body is present. If the dislocation is or was marked injury to the blood supply of the body is usually of sufficient severity to eventuate in aseptic necrosis. Whenever dislocation of the body occurs at both the tibiotalar and subtalar joints aseptic necrosis of the body is inevitable.

Treatment of fracture of the neck of the talus with dislocation of the subtalar joint should be accomplished by closed reduction. Plantar flexion should be maintained in a plaster cast for about eight weeks or longer if aseptic necrosis becomes evident.

Treatment of fracture of the neck of the talus with dislocation of the body from both the tibiotalar and subtalar joints is attempted by closed reduction, first manipulating in marked dorsiflexion to try to unlock the talus from behind the sustentaculum tali and then replacing the upper articulating surface in the ankle mortise. Open reduction must fre-

quently be done in order to accomplish reduction. Position is maintained by a boot cast in plantar flexion.

Because of the inevitability of aseptic necrosis of the body of the talus in severe fracture-dislocations many surgeons advocate immediate astragalectomy as the treatment of choice. In some clinics this treatment has produced satisfactory results, but in many other centers, the long-term results have been poor and pain has been a disabling factor. Differing from the head of the femur in aseptic necrosis, the body of the talus seems to have more favorable conditions to permit revascularization and consequently more rapid substitution of live bone for dead. It would appear that adequate replacement of bone may occur in from twelve to eighteen months. For this reason, it does not seem advisable to do astragalectomies routinely. Even though arthritis may be expected to develop in joints which have adjacent bone undergoing aseptic necrosis, better eventual results may be anticipated if ankle and subtalar fusions are brought about operatively when necessary.

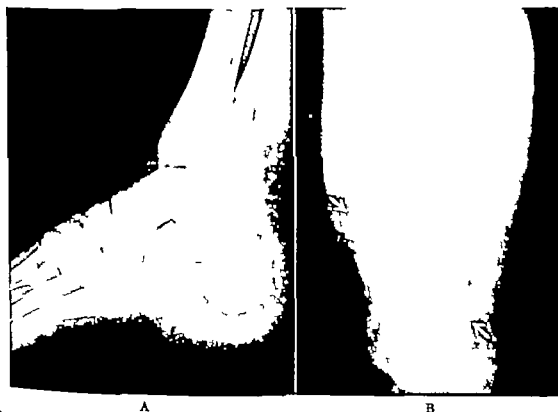
Fractures of the Calcaneus. The calcaneus is by far the most commonly fractured tarsal bone. It has been estimated that about 60 per cent of all tarsal fractures occur in this bone. In addition to its vulnerability to fracture, the calcaneus is also apt to be found fractured in association with compression fractures of the vertebral bodies. When a calcaneus fracture is occasioned by falling in an upright position so that the feet strike the ground first, or by an explosion forcing the deck of a ship or a floor suddenly upward, compression fractures of the vertebral bodies are likely to occur also. In such accidents the combination of compression fracture of the calcaneus with compression fracture of the spine is so common that it is advisable to x-ray both regions even though the symptoms may be confined to only the heel region.

A good deal of confusion has existed for years because of the difficulty in judging which calcaneal fractures were apt to heal with little or no residual pain and which ones would be painful to the point of incapacitation for work. One difficulty lay in inability to evaluate the extent of calcaneal fractures by clinical and ordinary x-ray examination. The advent of superoinferior or axial x-ray views of the bone has helped a great deal but it is still difficult to outline accurately the extent of many fractures. In this difficulty lies the source of our errors.

region opposite the sustentaculum tali while pulling strongly downward on the heel and the forefoot and then placing the foot in a cast in plantar flexion for six weeks, followed by neutral position for six weeks. Sometimes it is necessary to use a Böhler type apparatus which employs a Steinmann pin in the upper posterior portion of the calcaneus to effect longitudinal traction and then oblique downward traction (Fig. 110) Countertraction is obtained by the use of a pin through the tibia just above the ankle. After reduction is effected, a plaster cast embracing both pins is applied and left in place for eight to twelve weeks. As one becomes more efficient with the use of molding in plantar flexion

it will be found that fewer of the patients will require pin traction. There have been numerous serious infections around pins which are incorporated in plaster casts and, if possible such use of pins should be curtailed.

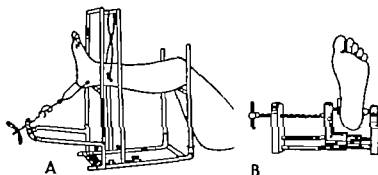
Fractures of the Navicular Cuboid and Cuneiform Bones. The navicular bone has somewhat the appearance and function of the keystone of an arch. It is fractured only occasionally, but severe fractures may result in long, painful disability. Among its more usual fractures three types may be recognized: tuberosity fractures in which the medial hooked area is fractured vertically but is seldom displaced, and requires casting for



A

B

Figure 118. A, Fracture of the body of the calcaneus with only moderate displacement. Note that the subtalar angle is not greatly reduced. The fracture did not involve the articular surfaces. The prognosis is good. B, Axial view of fractured calcaneus showing spreading of bone and fracture lines extending obliquely upward through bone.



A

B

Figure 119. A, Apparatus for reducing a fracture of the calcaneus by skeletal traction (after Böhler). B, special clamp designed by Böhler to correct lateral spreading of the calcaneus.



Figure 116 Fracture of the tuberosity of the calcaneus (lateral view). This type of fracture must be treated by open reduction and fixation.

pletely separated and displaced upward, open reduction and fixation with a nail or screw may be indicated (Fig. 116).

In the group of fractures which extend into the articulating surfaces are two main varieties: the type that runs upward and outward into the posterior portion of the subtalar joint and causes widening of the

articulating surface by displacing the lateral fragment outward and the type that by compression drives either or both the anterior and posterior articulating surfaces downward into the body of the calcaneus (Fig. 117). Some surgeons are now reporting success following open reduction of such fractures, but it seems likely that in most instances operative fusions will be required.

Of the fractures not involving articular surfaces, three principal varieties are common: tuberosity fractures, either vertical or the beak type involving the upper portion of the tubercle; fractures through the body, but not involving joint surfaces, and fractures of the sustentaculum tali. The tubercle and sustentaculum fractures can usually be treated by closed manipulation and the use of a plaster boot cast for four to six weeks. The fractures of the body frequently cause marked loss of the tuber joint angle and require more effort to effect reduction. In all fractures where there is spreading of the calcaneus laterally (Fig. 118) pressure by hand and knees or by a clamp should be used to squeeze the bony fragments together.

Whenever there is marked loss of the tuber joint angle regardless of whether articulating surfaces are involved, efforts should be made to restore the bone to normal contour. This may in some cases be effected by molding the foot while in plantar flexion by forcing upward on the sole in the

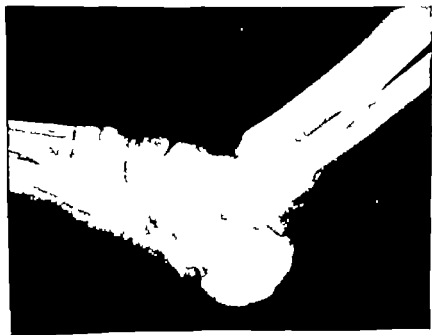


Figure 117 Multiple fractures of calcaneus extending into the articular surfaces. Note the loss of the tuber joint angle and upward displacement of the tuberosity. Because of involvement of the articular surfaces a poor result may be expected and arthrodesis may be indicated.

medial dislocation of the forefoot, the navicular may lock against the collar of the talus and defy all closed efforts to release it. A small incision permitting an elevator to pry the navicular loose from the back of the head allows subsequent reduction. A well molded cast for six to eight weeks followed by arch-support footwear is indicated.

Fractures of the Metatarsals. Fractures of the metatarsal bones are common the fifth metatarsal being most frequently fractured and the first metatarsal the next most frequently. Care must be taken not to mistake the normal epiphysis present in children at the lateral base of the fifth metatarsal for fracture. In addition to fractures due to direct violence, a variety of fatigue fractures, seen often in untrained military recruits required to take unaccustomed long walks, are recognized. Such march fractures occur at the necks of the second, third and fourth metatarsals, do not displace appreciably but cause considerable pain. For relief of pain

a cast may be applied for two or three weeks but in most instances a heavy soled, arch support shoe and limited activity for three weeks suffice for treatment.

The important feature of metatarsal fractures is the amount of plantar angulation of the fragments. Many metatarsal fractures have no significant displacement and many others have medial or lateral angulation or displacement with no plantar angulation (Fig. 123). Such fractures as a rule, heal without residual pain. The undisplaced fractures are treated by thick, leather-soled, arch-support footwear and limitation of activity. The angulated and displaced fractures of the second, third, fourth and fifth metatarsals with no plantar angulation (Fig. 124) should be manipulated in an effort to improve the alignment and accomplish reduction, if possible. When the best possible position has been accomplished and there is still no plantar angulation, treatment by application of a well molded plaster boot

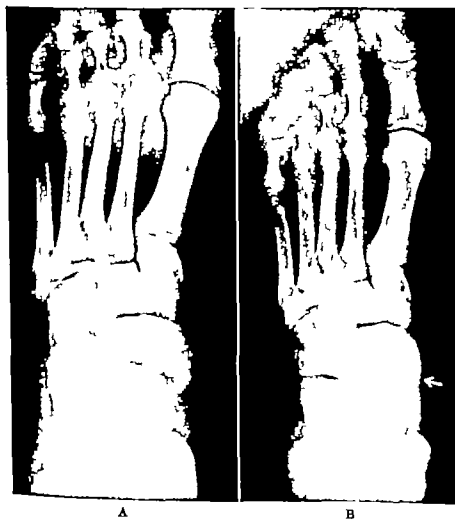


Figure 122. A, Metatarsal fracture-dislocation (anteroposterior view). Note the marked medial displacement of the navicular from the talonavicular joint. B, Metatarsal dislocation reduced. Note the fracture of tuberosity of the navicular bone.



Figure 120 Isolated fracture of the tuberosity of the navicular bone (anteroposterior view)

about four weeks (Fig. 120) dorsal chip fractures which are caused by direct trauma and seldom require more than elastic bandage support and some limitation of activity, and compression fractures which may result from an intolerable force applied over the crest of the longitudinal arch, causing the navicular bone to be violently squeezed between the head of the talus and the cuneiform bones. The compression force may

fracture the navicular transversely or in a stellate manner. Sometimes the dorsal fragment displaces upward and backward and, in rare instances, it cannot be reduced by closed methods. If open reduction of navicular fractures is required, it is advisable to take steps to promote arthrodesis of the talonavicular-cuneiform joints by removing the articular cartilages and packing many small bone chips or flakes all around the cartilage denuded joints. If fusion is not promoted, painful disability from the consequences of rough joint surfaces may be expected. A boot-type plaster cast, well molded upward in the region of the longitudinal arch, should be worn for ten to twelve weeks when fusion is occurring. This should be followed by use of a firm arch-support shoe or Oxford.

The cuboid and cuneiform bones are rarely fractured and, when fractured, they are seldom displaced significantly (Fig. 121). A well molded plaster cast supporting the longitudinal and transverse arches is indicated for comfort and support. It should be employed for about four to six weeks, following which a good arch-support shoe or Oxford should be worn.

An unusual type of fracture-dislocation involving the midtarsal area is occasionally seen (Fig. 122). The forefoot may be dislocated either laterally or medially with major displacement at the talonavicular and calcaneocuboid joints. The dislocation is almost invariably associated with fractures of some of the involved tarsal bones, although at times the fractures may be minimal. Reduction by closed manipulation is usually simple, but occasionally in patients with



Figure 121 Chip fractures of the cuboid bone (lateral view)

immobilization is used for about six to eight weeks. In all instances thick leather soled arch-support footwear is indicated following cast removal.

Fractures of the Phalanges of the Toes. One should be careful to secure good alignment of the great toe phalanges following fracture. Fortunately all phalangeal fractures, whether caused by stubbing the toes or having weights dropped on them, tend to remain in satisfactory alignment. They may be quite painful. In all patients, good alignment should be obtained, particular care being taken to avoid persistent plantar cocking or hammer toe deformity. Fractures of the great toe phalanges are best treated by employment of a metal splint or plaster cast. Fractures of the phalanges of all other toes can usually be treated by placing lamb's wool or cotton gauze between the adjacent toes and splinting to unbroken toes with adhesive strapping. Care must be taken not to have the strapping too tight or permit it to cut into the flesh. Sometimes a small dorsal splint is useful. Ambulation is permitted, but thick, leather soled, arch-support footwear is advised. If swelling is excessive, the tip of the shoe may have to be cut.

INTERNAL DERANGEMENTS OF THE KNEE JOINT

Internal derangements of the knee joint are commonly considered to be due to three mechanical causes: fractures of semilunar cartilages, tears of cruciate ligaments and osteoarthritic loose bodies. Closely akin to these agents, but separated because of anatomic position and pathologic differences, are two other major causes of knee joint disability: tears of the collateral ligaments and chondromalacia patellae.

Tears of the cruciate ligaments and fractures of the semilunar cartilages are frequently seen accompanying fractures of the knee joint region. Fractures of the plateau of the tibia are prone to cause injuries to the semilunar cartilages, the cruciate ligaments and the collateral ligaments. Dislocations of the knee joint always damage the menisci and all knee joint ligaments.

Osteoarthritic loose bodies are commonly seen in the knee joint as the result of three conditions: osteoarthritis, osteochondritis dissecans and osteochondromatosis, the last being rather rare. Internal derangement is caused by the loose bodies getting between the articulating joint surfaces.

The collateral ligaments are supporting structures to the knee joint, medially and

laterally. The internal semilunar cartilage is closely attached to the medial joint capsule which blends intimately with the internal collateral ligament. The external semilunar cartilage is more loosely connected with the lateral portion of the joint capsule and the blended external collateral ligament.

Chondromalacia patellae is a condition affecting the articular surface of the patella in which the surface becomes shredded and the hyaline cartilage degenerated. It is considered by some to be the result of trauma to the cartilage caused by the patella being driven against the femur. It gives symptoms of pain in the anterior compartment of the knee joint and usually produces crepitus on motion. If the symptoms become too troublesome surgery is performed with complete scraping off of the degenerated cartilage down to healthy bone. Some surgeons prefer to resort to patellectomy as an initial procedure.

Tears of the collateral ligaments are not really internal derangements of the knee joint but they are more common than any internal derangement. Tear of the internal collateral ligament at or near its femoral attachment is the most frequent of all major disabilities of the knee, and tear of the external collateral ligament at or near its femoral attachment follows. Injuries of the internal collateral ligament in order of frequency. A few surgeons taking care of highly skilled athletes operate on fresh collateral ligament tears if a diagnosis of complete rupture is made; their results are encouraging. The majority of surgeons utilize immobilization by a splint or a plaster cast for about four weeks, supplementing the therapy with good quadriceps exercises. Collateral ligament tears are diagnosed by pressing against the side of the patient's knee when his leg is extended. If abnormal motion is present, the joint opens on the side opposite the hand pressure and rupture of the ligament on that side is presumed. Chronic laxity of collateral ligaments due to old tears produces instability of the knee joint. Surgery for the repair of poor collateral ligaments or for substitution of collateral ligaments has not been uniformly successful and therefore the majority of surgeons prescribe a routine of quadriceps exercises in an effort to make the quadriceps mechanism strong enough to stabilize the knee joint.

In addition to tears of the menisci, mucoid cysts of the semilunar cartilages may cause symptoms of locking and pain. Cysts are more common in the external semilunar car-



Figure 123. Fracture of second metatarsal with slight displacement (anteroposterior view). No manipulation was necessary. Plaster boot cast provided satisfactory treatment.

extending from the tibial tubercle to the toes, to be worn for four to six weeks, is indicated, or thick, leather soled, arch-support footwear or an inserted rigid arch support may be used. Fractures of the first metatarsal should be reduced to anatomic position by closed manipulation when possible or by open reduction when necessary.

The only metatarsal fractures which require painstaking attention are those exhibiting significant plantar angulation. If the bones heal with marked plantar angulation, walking is often quite painful. In most of the patients closed manipulation is successful in eliminating the plantar angulation, but if not successful, or if improved position cannot be maintained in a plaster cast, open reduction and fixation with transfixion wires or plates and screws may be indicated. Skeletal traction applied by the use of small pins through the phalanges of the broken metatarsals is a useful method of treating difficult fractures. The pins are connected by rubber bands to a banjo splint arrangement formed by a loop of heavy wire extending beyond the toes and incorporated in a plaster cast. With the banjo splint arrangement,

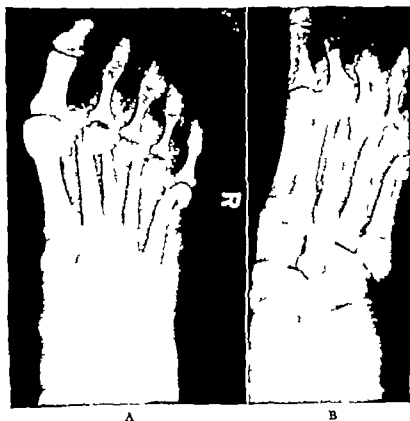


Figure 124. A, Fractures of necks of the second, third, fourth and fifth metatarsals (anteroposterior view). Because of serious associated head injuries, no definitive treatment could be given for these foot fractures. Plaster casts were applied for comfort. The patient recovered and walks with no pain and no limp. B, Lateral view of multiple metatarsal neck fractures. The plantar angulation is not marked.

notch and if it remains there it may produce minimal symptoms however it is more apt to slide out and lock between the articular surfaces, occasionally causing repeated episodes of disability. In such cases, there is always at least slight limitation of extension and of flexion.

Transverse and oblique tears may occur anywhere along the free edge of the cartilage and in some rare cases along the capsular edge, although the usual capsular tear is longitudinal and at the junction of the capsule and the cartilage. Oblique tears may produce a long tongue of cartilage fastened only at one end and the free end of the tongue may get caught between the articular surfaces. Quite a variety of tears may be produced in cartilages that undergo repeated fracture and one may have multiple bucket handle tears or Y or double oblique tears.

Initial treatment consists of making the patient comfortable and, if possible, reducing a displaced cartilage. Several methods of manipulation have been advocated for accomplishing replacement of torn cartilages which are caught in a position causing stretch on the capsule with the production of pain, but no certain manipulation is generally successful. Often, the patient is put to bed after manipulation with the knee still "locked" only to awaken the next morning with disappearance of symptoms. Perhaps it would be better to administer a sedative and let the patient spend a relaxed night in bed and if the knee is still very painfully locked the next morning, manipulation might then be considered.

With the first attack of meniscus fracture, immobilization of the knee in a splint or a cast with the knee in almost full extension is indicated. If the tear is close to the capsule, the blood supply is sufficient to heal the tear but if the tear is in the cartilage body it will never heal because of deficient circulation. Since one cannot accurately diagnose whether the tear is capsular it is better to give the meniscus an opportunity to heal, if perchance the tear is favorable. The cast or splint should remain on three or four weeks and quadriceps exercises should be done several times daily. If the attacks of knee disability recur and the diagnosis is firm, operative removal of the torn meniscus is indicated.

In diagnosis, several points need to be considered. The history of initial occurrence, the number and manner of recurrences, the length and severity of attacks and the findings on physical examinations. Anterior and

bucket handle tears usually show tenderness on pressure over the anterior horn of the cartilage just to the side of the patellar ligament. There is pain on rotating the tibia on the femur with the knee at right angle: there is loss of at least some degrees of extension and sometimes a "click" is evident on extension from a flexed position. X-ray studies in all patients with cartilage injuries are non-confirmatory. If the tear is lateral, there may be pain over the lateral aspect of the joint instead of anteriorly and if the tear is posterior there may be pain and limitation of motion on the extremes of knee flexion. Sometimes one can palpate the cartilage in different positions.

The question of the advisability of removing a torn cartilage which has caused several recurrent attacks of disability but which has gradually become less troublesome, is pertinent. Only by seeing the inside of a joint with an old badly torn cartilage and observing the damage to the articular cartilage of the femur can one be positive in advising removal of all torn cartilages which continue to give even minimal symptoms. The damage to the articular cartilage is sure to predispose to arthritic changes, and therefore, particularly in younger patients it is wise to remove all chronically torn menisci.

The technique of removing fractured semilunar cartilages varies somewhat between surgeons. The majority of surgeons prefer to keep the incision anterior to the collateral ligaments and vertical or oblique across the joint space. Some surgeons prefer an incision posterolaterally to remove all of the posterior horn of the meniscus. There is also difference of opinion as to whether the entire meniscus should always be removed or just the torn portion of a bucket handle tear. While, from the standpoint of eliminating all future chances of residual tearing it is probably safer to remove the total cartilage, statistics from some excellent clinics show very little difference in long term results. After removal of a semilunar cartilage a fibrous replacement forms that looks very much like the original meniscus.

Tears of Cruciate Ligaments. Cruciate ligament tears are frequently associated with fractures of the lower femur or upper tibia and always with dislocations of the knee joint. They may also occur in conjunction with fractures of the semilunar cartilages and are observed to happen in athletes participating in active contact sports.

Tear of the anterior cruciate ligament. The anterior cruciate ligament is fastened to

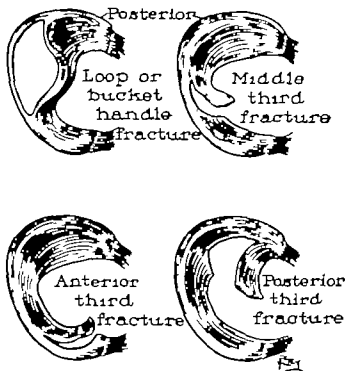


Figure 125 Types of fracture of the internal semilunar cartilage.

tilage and may cause the cartilage to protrude externally so that it can be seen and felt. Total removal of the cartilage is indicated when cysts are found.

In testing for range of motion and for stability of the knee joint, it is well to remember that great variations normally occur between individuals. The findings on the affected leg should always be compared with the findings on the other leg of the same individual.

Fractures of Semilunar Cartilages. Fractures or tears of semilunar cartilages are much more common in young men than in women and are frequently associated with forms of contact sports such as football, basketball and hockey. Tears of the medial semilunar cartilage are about ten times as common as tears of the lateral cartilage and "bucket handle" tears are more common than transverse, oblique or capsular tears.

Fracture of the medial meniscus occurs when the tibia is fixed to the ground and externally rotated on the femur. The knee is flexed and adducted when a force moves the leg into extension and the medial meniscus is caught between the tibia and the medial condyle of the femur causing the meniscus to tear. Fracture of the lateral meniscus occurs in a reverse position with the tibia internally rotated on the femur and the knee flexed and abducted when sudden extension is applied. A definite sensation of tearing may be noted by the patient and frequently

the knee joint becomes locked in partial flexion. In the majority of patients, the locking disappears when someone moves the leg or when the patient manipulates the leg. Occasionally the locking fails to disappear even after weeks have elapsed and only surgical removal of the cartilage gives relief.

In the average case, the limitation of motion preventing full extension and full flexion gradually becomes less and less until only by careful examination can any limitation be noted. Still, the patient may feel uncertain about the stability of his knee even though he may be able to participate in active sports. If the tear is in the cartilaginous portion of the meniscus, healing does not occur and after a while another episode of locking and disability will occur however often the trouble will not be as severe as the first attack. Subsequent episodes of displacement of the cartilage may cause less trouble until only minor difficulty may be experienced, however additional tears may occur causing increased symptoms.

Tears of the semilunar cartilages (Fig. 125) may be placed in four groups: bucket handle tears, transverse tears, oblique tears and capsular tears. The bucket-handle type of tear is the most common and is a longitudinal tear which does not extend all the way to either end. The movable "handle" portion of the torn cartilage frequently displaces all the way over into the intercondylar



Fig. 126



Fig. 127

Figure 126. Loose body in osteochondritis dissecans, arising from the internal condyle.

Figure 127. Osteochondromatosis of the knee with multiple loose bodies.

patella. Symptoms are vague until the body becomes entirely loose and happens to get between the articular surfaces. At first there is a feeling of uneasiness about the knee and occasionally a vague pain may be present. X-ray examination reveals a small defect just below the articular cartilage in a localized area. Since this trouble occurs primarily in adolescent boys and in young men it is wise to remove the body before it gets entirely loose and causes damage to the articular cartilage elsewhere in the joint. A word of caution is in order to remind surgeons that sometimes it is quite difficult to find the exact spot of the loosening body when the cartilage-covered area is examined. It may be wise to wait until the condition has progressed to the point where final loosening seems imminent and then it is not difficult to identify the region of softened, dull cartilage and minutely shredded surface. A sharp scalpel finds the line of demarcation between good surface cartilage and poor cartilage and, as the area is cut circularly the loose body below with its core of bone separates and is removed. If the body is discovered by x-ray examination to be free in the joint, it may be difficult to find when the joint is opened because it moves about quite freely and justifies its common name of "joint mouse."

Osteochondromatosis. The synovial membrane possesses the ability under certain benign pathologic processes to form numerous osteocartilaginous bodies which extend into the joint (Fig. 127). Many of the bodies arise from the region where the synovia is fastened to the bone and they may project inward as single bodies or in bunches like

grapes. They grow on pedicles which break and permit the bodies to become completely free in the joint. Hundreds of bodies of various sizes have been removed from a single joint, but unless synovectomy is done just as large a crop of bodies may be found the next time x-ray examination is made. Removal of the bodies and synovectomy are indicated in younger patients; in older patients, nothing need be done unless symptoms become troublesome. Symptoms are caused by the loose bodies getting between the articular surfaces.

DISLOCATIONS OF LOWER EXTREMITY JOINTS

Lower extremity joint dislocations may be divided into five categories: dislocations of the hip joint, dislocations of the knee joint, dislocations of the patella, dislocations of the ankle joint and dislocations of the joints of the foot.

It is not unusual to have dislocations occur either directly or indirectly because of infections of joints, Charcot joints, paraplegia, cerebral spastic palsy tumor formations and many other pathologic conditions. However consideration will be limited to dislocations caused by injuries of violence.

Dislocations of joints are severely traumatic episodes and in every instance there is damage to the capsule, the ligaments and the periosteum. Initial dislocations are usually very painful and may cause varying degrees of shock. Frequently there is considerable bleeding and this may produce a condition of myositis ossificans. Injury to the blood supply of the bone and to the surrounding soft tissues may lead to necrosis

the anterior portion of the tibial spine and to the back of the inner surface of the lateral femoral condyle in the intercondylar region. Thus it runs obliquely backward and laterally and limits the forward excursion of the tibia on the femur. A sudden forward push of the internally rotated leg with the femur stationary causes rupture of the anterior cruciate ligament and may also cause damage to the collateral ligaments. A strong thrust backward on the femur with the tibia stationary causes the same type of tear of the anterior cruciate ligament.

Evidence of rupture of the anterior cruciate ligament is shown by the drawer sign. With the patient supine and the knee flexed to 90 degrees the foot is rested against the table and the tibia is pulled forward just below the knee joint. Abnormal forward motion is indicative of injury to the anterior cruciate ligament. Rupture of this ligament is much more common than is rupture of the posterior cruciate ligament.

Tear of the posterior cruciate ligament. The posterior cruciate ligament is fastened to the posterior portion of the tibial spine and to the forward portion of the inner surface of the medial femoral condyle in the intercondylar region. Thus it runs forward and medially and limits the backward excursion of the tibia on the femur. Sudden backward thrust of the tibia with the femur fixed, or forward thrust of the femur with the tibia stationary causes rupture of the posterior cruciate ligament.

Rupture of the posterior cruciate ligament is demonstrated by putting the patient in the same position as for testing the anterior cruciate ligament and pushing backward on the tibia instead of pulling forward. If abnormal backward motion is found, it may be presumed that the posterior cruciate ligament is injured.

In some instances both anterior and posterior cruciate ligaments are found to be ruptured.

Treatment of cruciate ligament tears. The marked instability of the knee joint when both cruciate ligaments are ruptured produces a sensation of uncertainty on weight bearing. If only the anterior cruciate is ruptured and the patient has relatively strong muscles, there is not much disability. Rupture of the posterior cruciate ligament causes an increased feeling of insecurity, but strong musculature may overcome this difficulty.

If the patient is seen shortly after injury it is well to apply a long leg plaster cast

with the knee flexed about 30 degrees and pushed backward if the anterior cruciate ligament is torn and forward if the injury was to the posterior cruciate ligament. The cast should remain in place for eight to twelve weeks. It is of extreme importance to build up strong quadriceps power so that extra strength in that mechanism may substitute for injured ligaments.

Surgical repair of cruciate ligament tears is seldom of permanent value although numerous ingenious operations have been devised to assist this condition. Substitution operations are likewise disappointing. Development of strong muscle power about the knee joint is the best treatment at present.

Osteocartilaginous Loose Bodies. Loose bodies in the knee joint produce symptoms by irritating the synovial lining and by getting between the articular surfaces of the femur and the tibia. In some instances they closely simulate the symptoms of tear of the semilunar cartilages and only x ray examination gives definite differentiation.

Osteoarthritis. Osteophytes arising from the margins of the tibia, the femur and the patella may break off from their stems and become loose bodies in the knee joint. As long as the osteophytes remain attached and have a synovial covering, they are rough, but when they break off from their pedicles and wander about the joint, they become smooth and round. Any number may break off and become loose bodies and it is not unusual to see four or five such bodies in a knee joint. They produce symptoms by getting between the joint surfaces and if the symptoms are too troublesome, surgical removal is indicated. One must evaluate these cases however because the condition is more apt to occur in late middle age and old age when the arthritis present in the knees may be the major cause of symptoms. Only when firmly convinced that removal of the loose arthritic bodies will give relief should surgery be undertaken.

Osteochondritis dissecans. Loose bodies from osteochondritis dissecans may appear in the knee joint from the femur or from the patella. It is thought in some quarters that the condition of osteochondritis dissecans results from trauma and the local manifestation is that of avascular or aseptic necrosis of a small piece of bone and cartilage.

Most of the bodies arise from the inner portion of the medial femoral condyle (Fig. 128) but they may come from any part of the cartilage-covered femur in the knee joint. More rarely they may separate from the

As strong upward lift is exerted a gentle rocking motion is used to permit the head of the femur to find the tear in the capsule and return through the tear into the joint. Commonly a "pop" is elicited as the head snaps into the socket. The leg is then gently straightened.

The method of Bigelow (Fig. 131) uses

circumduction and requires the head to sweep around the outer rim of the acetabulum until it reaches the posteroinferior part of the joint where the rim is the lowest and then reduction is effected. The wide arc of travel of the head increases the danger of injuring adjacent tissues and also presents the hazard of the head continuing its upward



Figure 130 Grip used in reduction of a dislocated hip.

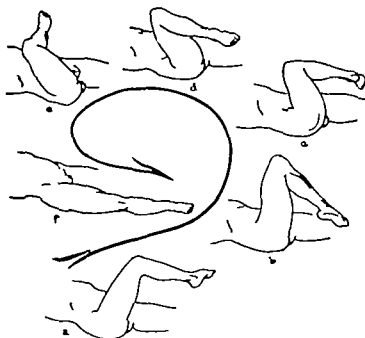


Figure 131 Bigelow method of reduction of a posterior dislocation of the hip (Cotton) a, The thigh is flexed and gently lifted in adduction with the leg in flexion, b, the thigh is rotated inward (foot outward) c, the thigh is adducted and rotated upward, d, the thigh is abducted, e, the thigh is rotated outward (foot inward) f, the leg is extended. This is done with a sweeping circular motion without force. In the diagram the heavy arrow indicates the sequence of positions.

and permanent disability and the longer the dislocation is permitted to remain unreduced, the greater the hazard of irreversible damage. Violent manipulative attempts at reduction may cause additional trauma and unnecessary complications.

Dislocations are often associated with fractures and it is not unusual to have unrecognized fractures because the massive displacement inherent in dislocations covers fractures which would otherwise be visualized on x-ray films. Occasionally fracture fragments are displaced into joints and prevent reduction of dislocations. Nerves are not infrequently damaged by dislocations and such damage may constitute the major residual disability following injury.

In recent years there has been a steady increase in the proportion of dislocations in bone and joint injuries. This is due to the increased violence which occurs in accidents of high-speed vehicles and also because the injuries happen when seated persons are hurled into projecting ledges and cramped corners.

Dislocations of the Hip Joint. Two principal types of hip joint dislocations occur posterior dislocations (Fig. 128) and anterior dislocations. Posterior dislocations are subdivided into two types upper or iliac dislocations and lower or sciatic dislocations. Anterior dislocations are subdivided into two types upper or pubic dislocations and lower or obturator dislocations. The subdivisions are descriptive of the regions occupied by the dislocated head of the femur iliac being on the ilium, sciatic at the sciatic notch, pubic near the os pubis, and obturator near the obturator foramen. Both anterior and posterior dislocations may be displaced even beyond the areas above noted, but such displacements are rare.

There is also a so-called central dislocation, but this can only occur in conjunction with fracture of the acetabulum.

Posterior dislocation of the hip is by far the most common. This usually occurs in automobile accidents and is brought about by the sudden forward thrust of a seated person striking the knee against the instrument panel or a seat. If the knees are crossed at the time, the dislocation occurs more easily. If the hip is markedly flexed at the time, the head of the femur is driven backward and downward toward the sciatic notch. If the hip is only moderately flexed, the femoral head is driven almost straight backward toward the ilium. Fracture of the posterior portion of the acetabulum may occur. The



Figure 128. Posterior and upward dislocation of the left hip.

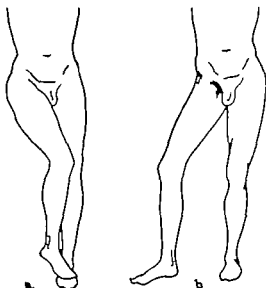


Figure 129 a, Posterior dislocation of the hip; b, anterior dislocation of the hip.

lower extremity on the affected side remains in a position of adduction, moderate hip and knee flexion and internal rotation (Fig. 129a.) Pain and shock may be severe.

Treatment of posterior dislocation of the hip consists of gentle manipulative reduction with the patient under general anesthesia. Three methods are in common use: the method of Allis, the method of Bigelow and the method of Stimson.

The method of Allis is probably the one most commonly used. The patient is placed on his back on the floor or on a low table and anesthetized. The pelvis is fastened down by a swath, or held down by an assistant, while the surgeon performs the manipulation. The hip and knee are flexed to right angles with the thigh adducted and slightly internally rotated. The surgeon then places his forearm up to the elbow under the bent knee and lifts upward (Fig. 130)

nology of knee joint dislocations is based on consideration of the femur as a fixed object and the tibia as the movable part. There are four types of dislocations of the knee joint: anterior dislocation (Fig 132), posterior dislocation, lateral dislocation and medial dislocation.

In all dislocations of the knee joint the capsule is torn, the cruciate ligaments are ruptured, the collateral ligaments are torn and the gastrocnemius and popliteus muscles are damaged. Other muscles may also be injured according to the type of dislocation. In posterior dislocations, the arteries and veins in the popliteal space may be injured and in medial dislocations the peroneal nerve is frequently damaged, causing peroneal nerve paralysis.

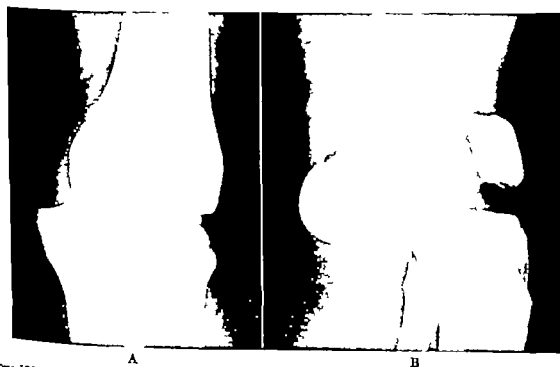
Dislocations of the knee joint are not common, but, like other disabilities due to severe trauma, they are undergoing a relative increase in occurrence.

The majority of knee joint dislocations are readily reducible by manipulation. Pulling down on the leg produces traction, and firm pressure over the protruding dislocated upper tibia pushes the bone back into place. In some dislocations, reduction is more easily accomplished by traction with the knee flexed to 90 degrees. Lateral dislocations may displace the semimembranosus, semitendinosus, sartorius and gracilis tendons into the intercondylar notch and in such patients it is impossible to reduce the dislo-

cation with the knee in extension. Flexion of the knee to 90 degrees or more relaxes these tendons and permits them to slide behind the condyle as reduction is accomplished.

Because of the instability of the knee joint resulting from any dislocation, it is necessary to immobilize the joint in a plaster cast for a period of six to twelve weeks. It is most important to institute quadriceps exercises as soon as the cast is applied and these exercises should be pursued diligently. After removal of the cast exercises should be continued. The inevitable multiple tearing of ligaments accompanying every knee joint dislocation produces a weak, unstable condition in a joint that largely depends upon ligaments for its stability. Only by attaining strong power in the quadriceps can good stability with fair function be expected.

Dislocations of the Patella. Dislocation of the patella is much more common than any type of knee dislocation. Invariably the dislocation is to the lateral side of the knee (Fig. 133). The patella may lock against the outer rim of the lateral condyle and stay with its anterior surface pointing forward or more frequently it may dislocate laterally and then rotate 90 degrees so that the articular surface lies against the lateral side of the lateral condyle. Spontaneous reduction is the rule rather than the exception and, for this reason, differential diagnosis is often difficult. Dislocation may only be momentary



Figures 132. A, Anteroposterior view of anterior dislocation of knee with some medial displacement of tibia. B, Lateral view of anterior dislocation of knee.

sweep and converting a posterior dislocation into an anterior one. With the hip and knee flexed, the thigh is brought into increased adduction and internal rotation. While strong upward lift is exerted, the knee is moved toward the umbilicus, then outward, downward and inward. The maneuver brings the thigh from marked internal rotation to marked external rotation. The final position after reduction is extension of the hip and knee.

The method of Stimson uses the force of gravity to aid in reduction. The patient is placed face down on an operating table with the pelvis at the lower break of the table. The lower table hinge is then released and an assistant holds the unaffected leg out straight to keep the patient from slipping. The thigh on the affected side is permitted to drop to a right angle to the body and then, with the knee kept in a bent position by holding the ankle downward, force is applied to the back of the calf just below the knee. This manipulation is frequently successful in reducing dislocation and actually is the same maneuver as the Allis method, but performed with the patient in a reversed position.

After reduction the patient is placed in bed on his back with his legs extended. The legs are kept in abducted positions by sandbags. Bed rest is enforced for three weeks, then crutch walking with little weight bearing on the affected side is advised for another three weeks.

Anterior dislocations of the hip occur as the lower extremities are forced into wide abduction and external rotation as in the position commonly known as "the split." Anterior dislocation causes the extremity to assume a position of moderate flexion of the knee and the hip with the extremity externally rotated and abducted (Fig. 129b). This type of dislocation is not as common as is posterior dislocation but it may present more difficulty in reduction. The degree of disability and shock is similar to that occurring in posterior dislocation and the dangers, while somewhat different, are no less.

Reduction of anterior dislocations is accomplished by one of two methods: the method of Allis or the method of Bigelow. The method of Allis is less traumatic and is in more common use. The method of Bigelow frequently results in converting an anterior dislocation into a posterior dislocation and, while in some patients such conversion seems necessary in order to accomplish reduction from the posterior position, the wide

excursion of the head of the femur around the outer rim of the acetabulum is not without danger of damage to important soft tissue structures.

In reducing anterior dislocations by the method of Allis, the patient is laid on his back and the pelvis is secured by a swath. The surgeon gently increases the abduction of the thigh to about 90 degrees with the hip flexed and the knee flexed to 90 degrees. Upward lift is constantly exerted by the surgeon while an assistant pushes firmly with his hands against the upper inner portion of the thigh in an effort to shove the head of the femur laterally. As the surgeon gently rocks the extremity the head finds the rent in the capsule and goes to the rim of the acetabulum. The extremity is then gently rotated inward and adducted while still being lifted. The head then goes over the rim of the acetabulum and reduction is completed as the extremity is straightened.

In the Bigelow method of reduction of anterior dislocations, the thigh and knee are flexed at 90 degrees and, as strong upward lift is exerted, the thigh is further abducted, further flexed and then adducted, internally rotated and finally straightened into extension.

Treatment after reduction consists of bed rest in the supine position with the legs tied together with a bandage so as to maintain an adducted position. After three weeks in bed, the patient is kept on crutches for another three weeks.

Muscles and capsule are always torn in dislocations of the hip but the two most serious frequent complications are injury to the sciatic nerve and injury to the blood supply of the head of the femur resulting in subsequent aseptic necrosis of the head. In posterior dislocations, the sciatic nerve may become stretched across the neck of the femur or may be injured by direct pressure of the head of the femur. If reduction is prompt, and not unnecessarily rough, the chances of complete recovery of sciatic nerve function are good but slow, sometimes requiring six to twelve months.

The incidence of aseptic necrosis of the head of the femur following hip dislocations is unfortunately quite high, being estimated at close to 25 per cent. Frequently x-ray and clinical evidences of necrosis fail to be confirmatory for almost a year.

Hip dislocations usually occur in vigorous young adults and prolonged disabilities from complications are of serious import.

Dislocations of the knee joint. Termi-

considered true dislocation and is better classed under injuries of ligaments.

The closely fitted mortise of the ankle joint with its posterior narrowing, and its lateral and medial malleoli projecting downward as cleats, makes true dislocation without fracture a rare occurrence. It is surprising that such pure dislocations ever occur because they necessitate very extensive rupture of ankle joint ligaments and, in the case of posterior dislocations, some spreading apart of the tibia and fibula. Yet a few true dislocations occur posteriorly and, extremely rarely, anteriorly.

In posterior dislocation the foot is pushed backward while the tibia and fibula are pushed forward. The astragalus locks behind the tibia and moves somewhat laterally. The dislocation occurs with the foot in plantar flexion, with the Achilles tendon in a shortened position.

In anterior dislocation, the heel is driven forward with the foot in dorsiflexion while the tibia and fibula are pushed backward. The strong Achilles tendon resists this displacement and is one reason for its rarity.

Nerves and blood vessels may be seriously damaged in such massive displacement.

Reduction of ankle dislocations is usually not difficult. With the patient anesthetized, the knee is flexed and traction exerted downward on the foot. With posterior dislocations, the foot is put in plantar flexion during traction and then pressure is exerted backward on the lower tibia and forward on the foot. With anterior dislocations, traction is made downward with the foot in dorsiflexion and then the foot is pushed backward while the tibia is pressed forward.

The extensive ligamentous damage necessitates cast immobilization for six to ten weeks, but after a few weeks a walking heel may be applied to the cast. The foot is placed in a neutral position during immobilization.

Dislocations of the Joints of the Foot. Dislocations of the joints of the foot may be divided into five types: dislocation of the talus, subtaloid dislocation, midtarsal dislocation, tarsometatarsal dislocation and metatarsophalangeal dislocation.

Dislocation of the talus. Complete dislocation of the talus without fracture is very unusual. Dislocation with fracture is discussed in the text concerned with fractures of the talus. Subluxations of the talus without fracture are concerned primarily with ligamentous injuries and are not true dislocations.

The talus may completely dislocate either forward and outward or inward and backward. Frequently the injury is compound and always there is marked circulatory damage. Reduction should be accomplished promptly in an effort to keep the soft tissue damage minimal and to release the pressure on the nerves and vessels. If manipulation with hand alone is not sufficient to pull the heel down and slip the talus back into its socket, a pin should be put through the calcaneus and strong skeletal traction exerted while manipulation of the protruding bone is carried out. Even though aseptic necrosis of the talus may result, it is better to go on to a pantalooid fusion later than to do a primary talectomy, however if the area is compound and dirty talectomy is the treatment of necessity.

After reduction, a plaster cast is applied with the foot in a neutral position and is left on for eight to ten weeks.

Subtaloid dislocation. On rare occasions, the talus may remain in its normal position while the foot below dislocates at the talocalcaneal and talonavicular joints (Fig. 134). Displacement may be forward, backward, lateral or medial. Disability is marked and circulatory impairment may be hazardous. Immediate reduction should be performed and ordinarily is not difficult. Traction downward on the foot to release the bones then manipulation to bring them back to their normal positions, ordinarily give prompt results. Plaster cast immobilization for four to eight weeks is indicated.

Midtarsal dislocation. Midtarsal dislocation is usually associated with one or more fractures. Rarely such a dislocation may occur without fracture and the position of displacement may be in any direction. Reduction should be prompt, but may be difficult. The anterior portion of the foot should be pulled forward, the displaced bones manipulated and attempts made to push them into place. The joints concerned are the talonavicular and the calcaneocuboid. If reduction cannot be accomplished by the closed method, open reduction should be performed.

Tarsometatarsal dislocation. Tarsometatarsal dislocation is the most common dislocation affecting the tarsal bones. The metatarsals may be displaced in almost any direction in relation to the tarsal bones and the dislocation may be single or multiple. The first and the fifth tarsometatarsal joints are most frequently dislocated.

Reduction is imperative if a painless foot is to be expected. Strong pull on the toes



Figure 133. A, Anteroposterior view of lateral dislocation of patella. B Lateral view of lateral dislocation of patella.

because extension of the knee usually causes reduction and the patient may never see the region of dislocation. Pain along the medial side of the patella and over the lower fibers of the vastus medialis may be confused with symptoms of a torn medial meniscus, and unusual mobility of the patella cannot always be evaluated because of the reaction of apprehension of the patient when the patella is pushed laterally.

Dislocation of the patella occurs when the foot is fixed on the ground with the tibia externally rotated and a sudden force pushes the knee inward. This brings the quadriceps tendon and the tibial tubercle on a line outside the lateral condyle and sudden contraction of the quadriceps causes tearing of the lower fibers of the vastus medialis and permits dislocation of the patella. A sudden force applied directly to the medial border of the patella when the quadriceps is relaxed may also cause dislocation of the patella.

Dislocations occur in adolescents and adults and should be distinguished from congenital dislocating patellae, in which the patellae dislocate almost every time the knees are flexed. It is considered that knock knees with their valgus positions are more liable to dislocations of the patella than are normal knees and it is further considered that congenital flattening of the anterior portion

of the lateral condyle permits dislocations to occur more readily than in normal states. The majority of dislocations occur in women.

Reduction of dislocation is simple. With the knee extended, the hip is flexed and the patella pushed upward and medially with the fingers.

Because of the marked tendency for recurrent dislocations, it is wise to immobilize the extremity in a plaster cast in almost full extension for eight weeks. Quadriceps exercises should be instituted as soon as the cast is applied and should be continued after removal of the cast.

If dislocations become recurrent, surgery is indicated. Provided the patient is old enough for the epiphyses to have closed, the most successful operation is transplantation medially of the tibial tubercle with its patellar ligament attachment. This brings the pull of the quadriceps more in line with the middle of the knee joint and prevents the patella from moving laterally.

Dislocations of the Ankle. Dislocations of the ankle are generally considered in five categories: posterior dislocation, anterior dislocation, medial dislocation, lateral dislocation and upward dislocation. Actually it is virtually impossible to produce medial, lateral or upward dislocations without fractures. Extensive rupture of ligaments, permitting subluxation of the ankle joint, is not



Figure 134 A, Anteroposterior view of ankle showing lateral subtaloid dislocation. B, Oblique view of lateral subtaloid dislocation. C, Lateral view after reduction of lateral subtaloid dislocation.

while the dislocation area is being manipulated usually accomplishes reduction if done early enough, but, if done late, it may fail to put the bones in place. Open reduction should be done if attempts at closed reduction are unsuccessful.

A plaster cast for four to six weeks is indicated following reduction.

Metatarsophalangeal dislocation. Dislocations at the metatarsophalangeal joints are rare but occur occasionally. The first metatarsophalangeal joint is the most frequently affected and displacement is with the phalanx lying on top of the head of the metatarsal. Strong pull on the toe while rocking it in dorsiflexion and sideways, usually accomplishes reduction. Immobilization for two or three weeks is recommended.

READING REFERENCES

- Böhler L. Medullary Nailing of Kuntscher Baltimore: Williams & Wilkins Company 1948.
- Böhler L.: The Treatment of Fractures. New York, Grune & Stratton, 3 vol., 1956-1958.
- Compere, E. L., and Banks, S. W.: Pictorial Handbook of Fracture Therapy. 3rd ed. Chicago, Year Book Publishers, 1952.
- Howarth, M. B.: A Textbook of Orthopedics. Philadelphia, W. B. Saunders Company 1951.
- Key J. A., and Corwell, H. E.: The Management of Fractures, Dislocations and Sprains, 2nd ed. St. Louis, C. V. Mosby Company 1956.
- Liebolt, F. L.: Illustrated Review of Fracture Treatment. Los Altos, Calif., Lange Medical Publications, 1954.
- Magnuson, P. B., and Stack, J. E.: Fractures, 5th ed. Philadelphia, J. B. Lippincott Company 1949.
- McMurray T. P.: A Practice of Orthopaedic Surgery 2nd ed. Baltimore, William Wood & Company 1943.
- Speed, J. S., and Knight, R. A., eds.: Campbell's Operative Orthopaedics, 3rd ed. St. Louis, C. V. Mosby Company 1956, 2 vol.
- Steadler A.: Orthopedic Operations. Springfield, Ill., Charles C Thomas, Publisher 1940.
- Watson-Jones, R.: Fractures and Joint Injuries, 4th ed. Baltimore, Williams & Wilkins Company 1953, 2 vol.

Muscles, Ligaments and Bursae

By JAMES K. STACK, M.D.

JAMES KEANE STACK is a graduate of Notre Dame and Northwestern University to which he came from Duluth, Minnesota. He has had a wide experience in traumatic injuries of the skeletal system as surgeon for a large railroad system. He is an Associate Professor of Orthopedic Surgery at Northwestern University.

The muscles, ligaments and bones, with accompanying joints and bursae, make up with their nerve and blood supply the motor-skeletal system. These structures are responsible for our ability to maintain an upright posture and to move agilely accurately and freely from place to place. Most of the structures in the motor-skeletal system differ from the other physical systems in that they are accessible to the sight and touch. The male of the species is especially attentive to his muscular development and the muscular system constitutes a substantial percentage of him. Ingrained in all persons is a pride of physique and this concept is so well established that compensation awards may be granted for its disturbance. For centuries the demonstrations of feats of strength, speed and agility have played an important role in our civilization. These demonstrations of the prowess of the motor-skeletal system are perhaps more frequent and accepted than those demonstrations concerning other vital but more concealed systems, such as digestive and respiratory.

Locomotive freedom is occasionally challenged by injury, bacterial invasion, neoplastic change or other disease and, for the surgeon to meet this challenge, certain established fundamentals must be available to him.

MUSCLES

In surgery involving muscle tissue, the primary concern is with skeletal or striated muscle. The muscles of the skeletal system are derived from the mesoderm. We are inclined to think of them as groups performing a certain function, as bending the knee or making a fist. However, such a function is accomplished by the simultaneous and synergistic action of myriads of muscle cells, each

equipped with the ability to contract, relax and to interchange with the capillary circulation those substances necessary for the function and survival of the cell. These cells are bound together in ever-enlarging systems until the point of the obvious and palpable muscle mass is reached. The power of contractility enhanced by the cooperative effort of the smaller muscle bundles is transmitted to bone by tendon or by a direct fibrous union. The cooperative or team effort, concept of muscle action can be taken a step farther. Each joint has antagonistic muscle groups moving it one way or the other and each group usually has protagonists to help. Seldom, however, is the struggle an even one, as most joints are more effectively used one way than another. Gravity also enters the game and by aiding one group or another may promote the production of contractures and deformities especially for the unwary surgeon.

The principles which govern the action of skeletal muscle have been established by those working in the fields of anatomy, neurology, physiology, chemistry, physics and mechanics and can be summed up briefly.

Each muscle cell, like all other living tissues, must have an arterial, a venous, lymphatic and nerve supply. The mass of cells making up the individual muscle bundles must have attachments called origins and insertions in order to function according to the laws of leverage. Some of the muscle, bone and joint arrangements in the body are very efficient levers while others leave something to be desired. The muscles consist of plasma, proteins, carbohydrates and acid and they give off measurable electrical potentials and measurable amounts of heat. The muscles have the property of, or are held in a state

of tone, which is responsible for our ability to maintain an erect posture without conscious effort. It is this same state of tone which holds the voluntary muscles in a state of readiness to act according to conscious impulse. This property of tone and this ability to respond are dependent, among other things, upon the intact nervous system. Studies in muscle kinetics tell us of the mechanics of muscle function and the relationships between them and the bone, joints and ligaments. They also explain the deformities and the functional disturbances which can be produced by paralysis, contractures and shortenings and faulty weight-bearing alignment caused by habitually bad postures and attitudes.

Muscle tissue like other highly specialized tissues in the body such as the brain and liver is not capable of regenerative repair. Any gross defect in a skeletal muscle such as is created by a rupture, incision or perforating wound, will heal by fibrous tissue invading the hematoma which is formed at the time of the insult. The basic muscle cell, with its highly integrated neurochemical contractile properties, will not heal by regeneration. However, after any but the greatest loss the fibrous tissue scar will unite the remaining muscle fibers and thus produce a functional result that may not greatly impair the use of the associated joints. In other words, an anatomic defect in voluntary muscle tissue need not create a significant functional defect. Voluntary muscle will atrophy when deprived of its nerve supply by direct nerve *section or by disease such as anterior poliomyelitis*. It will also atrophy by being subjected to unfavorable reflex stimuli. Perhaps the most common form of atrophy is that of disuse, when the muscle is deprived of the opportunity to carry out its normal function. An example is the shrinkage occurring after fracture and subsequent immobilization.

Voluntary muscle is also capable of hypertrophy and by progressive resistance exercises can be developed to even greater than normal size. Thus, when one muscle is paralyzed as for instance by poliomyelitis, antagonists can be strengthened and taught to compensate for this loss. Thus, the loss or impairment of a single muscle group does not mean necessarily the loss of all of the particular action in which that muscle group participates.

Skeletal muscle is heir to the ills which befall most other tissues. They run the gamut of life from congenital absence and anomalous origins and insertions to the lesions of

attrition that result in the fraying and ruptures of senescence and senility. In the years between, it is subjected to injury, infection, the vague inflammatory diseases that affect the collagen tissues, parasitic infestation and tumors that arise either in the muscle fibers proper or in the fibrous tissues that bind them. Peculiar to skeletal muscle are other conditions such as myasthenia gravis and the various dystrophies of which the basic causes are as yet unknown.

It is seldom that congenital absence of a skeletal muscle actually produces deformity or dysfunction enough to warrant transplantation of others to overcome contracture or to improve action. Usually the congenital absence is an academic curiosity and the attention of the patient is called to it because it involves gross defects which are unilateral. The muscle which is most commonly absent is the pectoralis major. This defect does not seem to affect the power of either adduction or internal rotation, as its antagonists seem to serve well.

Trauma to skeletal muscle tissue can be open or closed, e.g. the tissue can be punctured or lacerated, as by a gunshot or knife. It may be ruptured by nonpenetrating blows applied violently over, for instance, the calf, the anterior surface of the thigh and the anterior surface of the upper arm. The muscles of the abdominal wall may be ruptured, usually incompletely by hard coughing. The rather common fractures of the transverse processes of the lumbar vertebrae are due basically to muscle action and are associated with *muscle tearing at the point of insertion*. It is likely that some of the low back complaints following lifting and torsion strains are due to incomplete tearing of muscles within the substance or at the point of attachment to the ilium, sacrum or the lumbar vertebrae. Muscles may also be ruptured by longitudinal stresses which are beyond the tolerance of the elasticity of the fibers. The so-called Charley horse of the athlete is an incomplete muscular rupture caused by this particular violence and likely to occur in the early period of strenuous endeavor before the muscles have become accustomed to and are ready for the strains which will be put upon them. These incomplete ruptures are followed, of course by hemorrhage of varying degrees and the insult sets up a local spasm which accounts for the symptoms.

When it can be demonstrated that the rupture of a skeletal muscle is incomplete, rest in a position of relaxation is the treatment of choice. This may be followed by heat and

message and the gradual resumption of activity. If it is demonstrated that the rupture of the muscle is complete and there is no antagonist which can take over its duties to the point where there will be no functional disability an open repair is indicated. As stated before skeletal muscle tissue is not capable of regenerative repair and therefore the torn muscle ends should be anastomosed as accurately as possible so as to minimize the amount of scar and to prevent the healing of the muscle tissue in an elongated manner. In those instances in which the muscle has been parted by an open wound the repair should be preceded by a thorough cleansing and a wide and complete investigation to determine the viability of the muscle and the extent of its soiling. It is especially important to note its color and its response to delicate pinching. Lacerated or contused muscle tissue makes the ideal culture medium for those anaerobic pyogens and gas-forming bacilli which threaten first the muscle group and, later the entire limb or even the life of the patient.

Primary hematogenous infection of skeletal muscle is rare, because it has a copious blood supply which is principally anastomotic and devoid of the end arteries that make certain other tissues particularly vulnerable to this method of infection. Muscle will of course be involved secondarily in those suppurative infections that arise in the contiguous tissues, as in osteomyelitis, suppurative arthritis, infected open fractures and those abscesses which develop as the result of infected deep perforating wounds.

Skeletal muscle, and particularly the fibrous elements which form its interstitial parts, can be the site of nonbacterial inflammation. This condition is known as fibrositis, myositis, or muscular rheumatism. It may be temporarily acute and disabling and frequently is chronically uncomfortable, but it is not deforming or crippling. It is thought to be a mild variant of the group of collagen diseases, the more severe of which are rheumatoid arthritis, lupus erythematosus and periarteritis nodosa. The diagnosis of fibrositis or myositis should be made only after potentially serious, underlying lesions which produce lumbago, coccydynia, torticollis and pleurodynia have been ruled out. In other words, this diagnosis should not be a "work-ing diagnosis" but rather should be one that is made by exclusion. Fortunately this type of muscular inflammation responds to simple home measures, such as aspirin, heat, massage and exercise, though many people are

plagued with intermittent symptoms for discouragingly long periods.

Invasion of skeletal muscle by the *Trichinella spiralis* which enters the body by the ingestion of insufficiently cooked pork is occasionally seen. The inflammatory change developing around the encapsulated parasite will produce considerable local pain and tenderness during the acute phase and may in the chronic phase weaken muscle tissue and predispose it to rupture. Infections by the *Cysticercus* or by the *Echinococcus granulosus* are much rarer and in this era, almost surgical curiosities.

Myositis ossificans is another of the inflammations of muscle and is characterized by the laying down of new bone in the site of the hematoma following muscle rupture. In this instance, the rupture may be due to a single major violence or to repeated minimal tears which are said to be occupational in origin as in the adductor muscles of horsemen. This new bone is the end result of the metaplasia of the connective tissue which invades the hematoma. The process develops in the same basic manner that results in bone deposition between and around the ends of a fracture. The early phase of this condition is characterized by pain and dysfunction in the involved muscle group and, early in the development of the lesion, x-ray examination reveals no abnormality. There follows, in most instances rather rapidly, the development of a spotty or granular appearance in the affected muscle. This goes on to give the typical appearance of solidarity that makes the diagnosis possible on x-ray examination. If superficial, the bony mass may be palpable but, if deep its presence may not be suspected on clinical examination and will be seen only on x-ray films. In the x-ray study of such a lesion, it is important that additional tangential views be taken with the usual anteroposterior and lateral ones. This is necessary in order to establish the fact that the bony mass is not connected with the long bone in the affected limb. It is most rare for the ossified area to connect itself with the shaft of the bone, but, if it does so it then gives the appearance of a benign osteoma. It is not to be confused with those tumors arising in or invading muscle which are capable of laying down new bone.

Excision of an area of myositis ossificans is necessary only when it is of sufficient size to interfere with function, if it is excessively tender or if it jeopardizes major blood vessels and nerves in the area. It is important that the excision be carried out only after interval

x-ray studies show it to be mature. If excision is attempted in the immature phase of the development of this lesion a recurrence from the operative interference may follow. In the nonoperative management of myositis ossificans, the instillation of hydrocortone or hyaluronidase is an adjunct which may be of value in deterring the formation of bone in the hematoma or of hastening its absorption.

Tumors of skeletal muscle are relatively rare when compared with those arising from the glandular, osseous, nervous and lymphatic tissues of the body. However there are some, both benign and malignant, which bear mention.

Lipomas arise in the fatty areolar tissue lying between the muscle bundles. Attention is usually called to them by changes in the circumference or contour of the affected limb rather than by any interference with function or by pressure on nerves or important vessels. The x-ray examination discloses a typical density which makes the diagnosis possible. Fat does not absorb the x-rays as they pass through the affected limb and, therefore the lipoma gives the typical dark circumscribed appearance. It may appear globular if it is small and free or it may be elongated if compressed within fascial planes. In either case, its periphery is sharply demarcated and there is no suggestion of the invasion of contiguous parts.

Fibromas arising in the fibrous tissue between the muscle bundles are not as common as those arising in the subcutaneous fibrous tissue or in the fibrous tissue surrounding glandular structures, for instance, the breast. Here again attention is called to the lesion because of the alteration in contour. It is usually not painful or tender and generally does not develop sufficiently in size to interfere with function.

Hemangiomas, developing from congenitally abnormal arteriovenous communications, are also found within skeletal muscle. These can be identified by the change in size on elevation and depression in the limb by the compressibility on palpation if the tumor is superficial enough, by the appearance of calcified flecks within the suspected area on x-ray films and by venography or arteriography.

The acutely painful glomus tumor (myoneuroarterial glomus) has a muscle component but, as a rule, does not arise from skeletal muscle or from its fibrous interstices. Rhabdomyomas, which are the only true tumor derivative of the muscle cell, are extremely rare and the diagnosis can be made only by microscopic study.

All malignant tumors of muscle and the fibrous and fatty tissue within them are sarcomatous in nature and are accurately identified only by biopsy.

Metastatic tumors are rare, because no tumor has a predilection for skeletal muscle in its vascular or lymphatic spread. However tumors arising from the elements of bone or those from synovia will invariably invade or compress skeletal muscle in the course of their growth.

Herniation of muscle fibers through the containing fibrous sheath is occasionally seen in the anterior tibial, hamstring and lateral thigh areas. These are seldom capable of producing disability and rarely require surgical repair.

Lesions of attrition involving skeletal muscle and its associated tendon constitute a very important segment of the surgery of the skeletal system, first, because they are so common and, secondly, the nature of these lesions is frequently misunderstood. Normal muscle and tendon tissue will rupture only when subjected to substantial violence and this violence must be a specific stretching force applied longitudinally and with sufficient intensity to be beyond the limits of elasticity of the muscle or the tendon. These structures, because of their mobility and compressibility, can withstand all but the most powerful of direct and lateral forces. This is brought out by the rarity of muscle and tendon ruptures in instances of massive violence as in falls from heights or head-on collisions, in which the osseous system and the abdominal thoracic and cranial organs sustain severe damage. The history of most muscle and tendon ruptures suggests relatively trivial trauma, and some degenerative change either fibrous, fatty or xanthomatous, will be found to exist on microscopic study of the sections taken at the time of repair.

When a powerful young athlete ruptures his plantaris muscle on reaching for a shot in handball, or tears away the rectus femoris origin in starting a dash, or pulls apart his heel cord in making a turn in a downhill ski race, he is not at all appreciative of the diagnosis of rupture secondary to degenerative change. He cannot understand how such would happen in doing something he had done thousands of times before. The answer lies in the disturbing fact that we begin to show signs of wear and tear in our motor skeletal system, as well as in other body systems, much earlier in life than we may think. It has been said that our tissues in general will never be better than they are at the age of ten years.

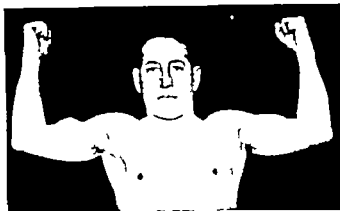


Figure 135. Partial rupture at proximal musculotendinous junction of the long head of the right biceps due to direct trauma. Note the characteristic bulging of the muscle belly

The bicipital tendon in its humeral groove is frequently frayed and will rupture with the slightest provocation. It will also part less frequently at its musculotendinous juncture and, still less frequently, it will be torn, usually by substantial violence, from its insertion on the bicipital tubercle of the radius. The proximal ruptures through the bicipital tendon and through the musculotendinous juncture will in a surprising number of instances cause no great disability and the patient's question will concern the change in contour of the biceps area as compared with the opposite side (Fig. 135). If excessive pain or disability is present, however satisfactory power can be restored by transplanting the tendon to the coracoid process of the scapula, thus having both heads of the biceps come from the same origin. Or in instances of lower tears, the muscle belly can be sutured to the humerus at the base of the bicipital groove. The rupture at the point of insertion in the bicipital tubercle of the radius has a much more disabling potential and should be replaced accurately by open surgical methods. Ruptures of the quadriceps muscle or tendon, which usually occur at the musculotendinous juncture or at the tendinous insertion onto the patella, require open repair and sufficient healing time in the position of relaxation. The Achilles tendon likewise lends itself to end-to-end anastomosis and the results are uniformly good by permitting healing with the foot held in the position characteristic of equinus, thereby relaxing the gastrocnemius group of muscles.

The plantaris muscle and tendon, because it plays a relatively minor role in the action of the calf muscle group need not be openly repaired and, following the initial pain and disability it will heal well by keeping the foot in an equinus position, usually by simply having the patient wear an elevated heel.

Those tendons which make up the rotator

cuff of the shoulder and join together the actions of the supraspinatus infraspinatus teres minor and subscapularis muscles are frequently the site of lesions of attrition. The tendon cuff and the bursa that lie immediately above them are impinged between the acromion process and the greater tuberosity of the humerus whenever the arm is elevated over 90 degrees. This wear and tear and the danger of subsequent rupture is the price we pay for brachiation. The deposition of calcium salts in the frayed fibers of these tendons is a common occurrence and is one of the common causes of shoulder pain (Fig. 136). When this calcium deposit reaches a size which permits it to irritate the floor of the bursa acute shoulder disability follows. A complete rupture of this tendon cuff makes it impossible for the patient to initiate abduction of the humerus because the mechanism has been disrupted which ordinarily would stabilize the humeral head against the gle-



Figure 136. Calcification within the tendons of the rotator cuff of the shoulder producing secondary bursitis in the subacromial bursa.

noid. In the absence of this stability the deltoid cannot get sufficient purchase to abduct the arm through the first 15 degrees. Open repair of the tendon cuff is necessary when these circumstances exist, but in the relatively minor tears which are one of the common causes of the painful shoulder operation is not indicated if abduction of the humerus can be initiated.

LIGAMENTS

Ligaments are basically of fibrous tissue structure, modified and strengthened to bind together and support all joints. They yield within the limits of their tolerance as the muscles move the joints or as the joints are moved passively by varying forms and degrees of violence and gravity. Many joints, especially the larger ones, have ligaments which are well defined, can be isolated and identified in the dissecting and the operating rooms and have specific names. Other joints, especially the smaller ones, are supported by blended reinforcement and expansions of the heavy fibrous layer of the capsule and these ligaments cannot, from a practical operative standpoint, be individualized. An example of the former would be the knee with its large and well-defined collateral and cruciate ligaments. An example of the latter would be the many reinforcements present throughout the multiple joints which make up the carpus. Some ligaments are relatively fragile and do not have the support of numbers. They are easily compromised and an example of this group is the fibulotalar ligament of the ankle. Others are heavy and numerous to the point where they will permit no functional motion and an example of this is the sacrotiliac.

Ligaments are highly innervated by fibers concerned with proprioception and pain and, because of the latter group, are capable of producing a degree of discomfort and disability beyond what one might expect on the basis of purely local findings on clinical examination. Because of their close relationship with the joints they support, they are endowed with a good blood supply which promotes prompt and efficient healing. If the principles of rest and relaxation of the injured structures are adhered to, the ligamentous structure, being basically a fibrous tissue, enables ligaments to reproduce a like tissue in the healing process and, if not permitted to heal in an elongated manner, the healed ligament will function as before.

Because ligaments are so intimately associated with joint structure and function, they have to be involved by association in dis-

eases, infections and neoplasms involving joints. Some injuries however may be selective enough to be confined to the ligaments alone, particularly when the ligament is not so intimately associated with the joint capsule, as in the case of the fibular collateral ligament of the knee and the nuchal ligament of the neck. The ligaments supporting a joint will be edematous in acute inflammatory diseases of that joint and thickened in the chronic conditions which affect it. The ligaments will be invaded and destroyed by tumors originating in the contiguous synovial membrane, periosteum or bone. They will be infiltrated, frayed and weakened by the sodium urate crystals deposited in gout, but seldom will the surgeon be dealing with serious conditions arising primarily in and involving solely a ligament.

The surgeon's principal contact with ligaments will be in the field of the surgery of trauma. Two terms used by the profession and the laity now enter this subject and, if used interchangeably they may be confusing. These are "strain" and "sprain." We should think of a strain as a stretching of a ligament within the tolerance of its tissue. There is no tissue in the body with the possible exception of adult cortical bone, that does not have some elasticity and yield. But if a persistent effort is made to strain a joint, for instance, to hyperextend the metacarpophalangeal joint forcefully and for several minutes, it will stretch and produce a very painful hand. Yet, on release, comfort will follow and no demonstrable tissue change will have taken place. As another example of strain, let us use the bamboo pole of the vaulter. It may bend to a frightening angle as he ascends to the crossbar but it shows no gross change when the vault has been completed. It is then used again and again.

A sprain, on the other hand, represents a definite rupture of ligamentous tissue, complete or incomplete. It may or may not permit the joint to dislocate. A sprain is caused by forces of violence which are beyond the limits of the tolerance of the tissue. A gross dislocation of the shoulder for instance, caused by substantial violence is permitted because the anteroinferior supporting ligaments and the capsule have given way either in their substance or at the attachment to the glenoid rim or humerus. This exemplifies an extreme form of sprain, permitting actual joint disruption.

Other sprains involving major ligaments may not be so obvious and may be missed unless thought about.

ulotalar liga

ment on the anterolateral surface of the ankle, when it is torn by inversion or adduction violence, will permit the talus to tilt on the distal articular surface of the tibia when the ankle is forced into inversion. This can be demonstrated by x-ray study after local infiltration with Novocain or with the patient under a light general anesthesia. When it is present, it is known as an occult dislocation of the ankle (Fig. 137). Normally, one is unable to change the relatively parallel relationship between the articular surfaces of the tibia and the talus by forced inversion. If such a sprain is not immobilized for from four to six weeks to permit solid healing, ankle instability may result and the patient may then become one who recurrently "sprains" his ankle. Plaster immobilization in a neutral position usually suffices and open repair of this ligament is seldom necessary.

On the opposite or medial side of the ankle is another important ligament, the complete and unrecognized sprain of which can also produce recurring disability. This is the deltoid ligament, so named because of its roughly triangular shape. This ligament is sprained by eversion or abduction violence—the same mechanism that produces the common Pott's fracture. The talus is forced laterally and by pressure against the fibula breaks it, usually an inch or two above the articular surface. If the force is continued, the talus

moves farther laterally and the medial malleolus of the tibia is then pulled off thus producing the typical bimalleolar fracture. If on the other hand, the medial malleolus remains intact, then the deltoid ligament will tear completely or incompletely. Whether the tear is complete or incomplete will be determined by the amount of lateral displacement of the talus by the condition of the medial malleolus and by the amount of local reaction about the medial side of the ankle. If this tear is thought to be complete an open exploration and repair should be executed. If this is not done under such circumstances ankle instability will result and the stage will be set for future development of traumatic arthritis.

A third joint, the ligaments of which are subject to serious sprain, is the knee. The medial collateral or tibial collateral ligament is commonly sprained in twisting injuries and by forces tending to push the knee into a valgus or knock-knee position. Because of its attachment to the medial meniscus it is likely to be involved, at least in some manner when this meniscus is torn. However this ligament can also be completely torn either in its substance at the level of the joint or from its femoral or tibial attachments. When torn completely the resultant instability of the knee can be demonstrated by an anteroposterior x-ray exposure. If the knee can be



Figure 137 The talus can be tilted on the articular surface of the tibia by forceful manipulation with the patient under general or local anesthesia, indicating a rupture of the fibulotalar ligament. The shadows above and below are the lead gloves protecting the hands of the demonstrator.

forced into a knock knee deformity of 15 degrees or more, then open repair of this ligament is indicated.

The cruciate ligaments lying within the knee joint are also subject to sprain and strain, particularly the anterior cruciate. It is the function of the anterior cruciate ligament to prevent the anterior displacement of the tibia on the femur and this ligament is taut when the knee is in extension or in mildly flexed positions. For this reason, it is more likely to be damaged than the posterior cruciate ligament which prevents posterior displacement of the tibia on the femur and is taut when the knee is completely flexed, as in the squatting position. The posterior cruciate ligament is seldom torn alone but gives way in complete disruptions of the knee. The anterior cruciate ligament, on the other hand, is not uncommonly torn without other damage to the joint and is frequently associated with tears of the medial meniscus with or without accompanying disruption of the medial collateral ligament. Fortunately an open repair of the anterior cruciate ligament is not, as a rule, necessary because the quadriceps muscle can be developed by progressive resistance exercises to stabilize the knee and effectively take over the function of the anterior cruciate ligament.

There are dangers inherent in the diagnosis of sprain in certain joints. When applied to the knee, one must be sure there is no underlying internal derangement such as a torn meniscus or no disease such as osteochondritis dissecans. One also must be sure that the patella did not dislocate and return to its normal position spontaneously. When applied to the lower back it must be determined that there is no congenital structural weakness or neurologic evidence of nerve root involvement or early manifestations of rheumatoid spondylitis. The diagnosis of a sprained wrist should not be made until all possibility of a fracture of the navicular bone has been ruled out by x ray films exposed in oblique positions with the wrist held in ulnar deviation. Such x ray study should be repeated ten to fourteen days after the injury as the hairline fracture through the wrist or the proximal pole of the bone may not be seen until sufficient absorption has taken place at the fracture line to widen it. The "sprained neck" can be a unilateral subluxation and the "sprained elbow" can mask an impacted fracture of the radial head. The "sprained hip" can be an impacted fracture of the femoral neck which may come apart after absorption and attempted use.

The diagnosis of "sprain" is used loosely by laymen and doctors alike and the failure to recognize underlying lesions which are part of the sprain picture, or independent of it, will lead to serious diagnostic errors.

BURSAE

"Bursa" is a Latin term meaning purse or pouch. Its plural form is "bursae." Bursae are most important in the musculoskeletal system and there are literally dozens of them. They are sacs containing a small amount of lubricating fluid which physically and chemically is the same as joint fluid. It is the same because the bursa is lined with a secretory membrane which, for all practical purposes, is the same as synovial membrane. The outer wall of a bursa is of fibrous tissue inseparably attached to the inner serous surface. Most bursae have but a single cavity i.e., they are monolocular or unicameral, but the demands of function or the residuum of disease or injury may cause the development of membranes which will divide them into two or more compartments. It is pointless to number the bursae and useless to name them, except for those that are of practical surgical interest.

It has been suggested that we are born without bursae because, in infancy the physical pressures and frictions of this rather passive phase of our existence are not great enough to warrant lubrication other than that provided by the joints. Certainly babies in the crawling stage do not develop prepatellar bursitis or housemaid's knee, presumably because there is no prepatellar bursa at this stage in life. At any rate, the bursae either are formed or develop from a basic potential structure in response to the demands which are put upon the involved areas. During the course of human growth, bursae develop at all points where any appreciable friction or motion exists. They are found where muscles and tendons must work around corners, where muscles cross to work in opposite directions and at those points between the skin and the underlying bone where frequent pressures and frictions are exerted. They are actually only potential cavities and under normal conditions are collapsed so that the serous surfaces are in contact. They are usually attached above and below and the gliding mechanism is achieved much like moving the palms of the hands back and forth with a collapsed lubricated balloon interposed. The rubber surfaces move on each other while the palms are adherent to the outer surfaces. In order to glide, there

must be a certain amount of redundancy in the bursa so that in some positions one surface will be taut and the other in folds. We know that in certain areas of the body adventitious bursae develop and are well known for the lesions they produce. They develop in response to structural change or to the need for additional protection to a part. Perhaps the best known of the adventitious bursae is that occurring on the medial side of the first metatarsophalangeal joint in association with a bunion deformity. The pressure and friction of the shoe are responsible. An ill fitting slipper may cause a bursa at the back of the heel and a bursa may form over any arthritic outgrowth on the dorsum of the foot where the tongue and the lacing of the shoe constantly press against it.

Bursae can be the site of local infection or injury and they can be involved in systemic disorders. The superficial bursae are most likely to be injured by direct violence, either by a single substantial blow or by repeated minor traumas. Examples of this are the chronic swelling of the prepatellar bursa in front of the knee—housemaid's knee—that over the tip of the olecranon process—miner's elbow—and that over the ischial tuberosities—tailor's bottom. The superficial bursae are also the most likely to be involved in puncture wounds and, if infection intervenes a true suppurative bursitis will result.

The deeper bursae are inaccessible to the usual direct violence and are more likely to become inflamed from chronic strain or a wearing and fraying process in the tendon sheaths contiguous to them. This, for instance, is the most common cause of pain arising from the subacromial bursa in the shoulder and the trochanteric and iliopectineal bursae about the hip. The irritation produces hyperemia and causes the development of an effusion within the closed sac and it is this tension which can be so excruciatingly painful.

The diagnosis and treatment of bursal conditions is based upon the same orderly concept as that of any other tissue. We may list the various types of involvement, such as congenital, infectious, neoplastic, metabolic, traumatic and attritious.

The congenital category is not an important one because there is a possibility that bursae are either absent or only potentially developed during the period of infancy. From a surgical point of view this may be helpful in a negative way because the possibility of a primary bursal lesion can be ruled out during the neonatal and infantile periods.

Bursae can be infected by perforation and contamination and subsequently involved in cellulitic processes, abscess formation and local gangrene as can any other soft tissue. The bursae may be invaded by the spread of infection from a contiguous tissue and, in theory at least, they can be involved by hematogenous infection. Primary tuberculosis or tuberculous involvement of a bursa by any means is not common but a bursa may be involved in tuberculous infection if it lies adjacent to an involved bone joint or tendon sheath. The nonbacterial inflammations, such as rheumatoid disease frequently involve bursae because they have a predilection for synovial surfaces. Hypertrophic villous bursitis of unknown cause does occur and this may result in the formation of rice bodies and, later in the osteochondral foreign bodies which are also seen in joints (Fig. 138).

Tumor formation is not common in bursal tissue in the primary phase and there is no tumor that has any special affinity for bursal metastases. Like all fibrous tissue, the outer wall of the bursa can undergo sarcomatous change and a fibrosarcoma can originate there. Also the highly malignant synoviomias can have their origin from the serous lining of a bursal sac just as readily as from the serous lining of a synovial surface.

The outstanding metabolic disease with a predilection for bursal involvement is gout. Gout causes the deposition of crystals of so-



Figure 138 Osteochondral foreign bodies in the superficial infrapatellar bursa. Many rice bodies were also present, and hypertrophic villous bursitis was present throughout the lining.

dium urate in the synovial lining of joints, bursae and tendon sheaths. The amount of this urate may be so great as to destroy the structure by pressure, followed by rupture and invasion of the contiguous soft tissues. It is not uncommon for a superficial bursa, such as the olecranon or prepatellar to be filled with solid crystals, and occasionally it will rupture and drain and become secondarily infected.

The management of bursal disease will be dictated by the nature of the involvement and the location and will range from simple advice as to home remedies to amputation of the limb of which the bursa is a part. The milder forms of superficial bursitis have a strong tendency toward spontaneous recovery with or without the help of rest, protection and moist heat. Usually the olecranon and the prepatellar types will regress with one or more aspirations and the application of a pressure dressing. The instillation of 1 cc. of hydrocortone within the sac after aspiration is also an established method of treatment because of the local anti-inflammatory properties of this drug. In the chronic stage of bursitis, however in which the hypertrophic villus formation has taken place, pain will be present on pressure and a crepitant sensation will be obtained on palpation of the involved bursa. Such a bursa should be excised. Most patients will ask about the function of the extremity without the bursa and they should be told that a new one will form in response to the demand. Excision is also the treatment of choice in those bursae containing chondral or osseous foreign bodies or a gouty mass which threatens to rupture and become secondarily infected.

Substantial enlargements of the anserine bursa on the medial side of the upper tibia between the collateral ligament of the knee and the gracilis and semitendinosus insertions will have to be excised because of pain and interference with knee function. In the milder forms of this type of bursitis, multiple needle punctures using 1 per cent Novocain and 1 cc. of hydrocortone will decompress the bursa and bring relief. When the bursa is small it is difficult to actually aspirate material from it, but the excess fluid can be dispersed into the surrounding tissues by the needle punctures and it will ultimately be absorbed. Affections of the greater trochanteric bursa at the insertion of the gluteus maximus muscle and the lesser trochanteric bursa at the insertion of the iliopsoas muscle can be treated in a similar way.

The bursal lesion which will receive the most attention, however will be the subdel-

toid, or subacromial. This is located immediately superficial to the rotator tendon cuff of the shoulder and it usually extends from an area just distal to the tuberosities to a point about 1 inch under the acromion process of the scapula. It lies on the tendon cuff and is covered distally by the deltoid muscle and proximally by the coracoacromial ligament which forms the roof of the humeral-scapular joint. Because of the proximity of the long tendon of the biceps muscle and its sheath within the bicipital groove, lesions of this structure must be kept in mind in the differential diagnosis of bursitis of the shoulder. The inflammatory process involving this bursa is usually not primary but is secondary to a fraying or a lesion of attrition of the tendon cuff beneath it. Depositions of calcium salts in this cuff are common, whereas deposition in the bursa is rare and occurs only when the mass of calcium is large enough to rupture out of the tendon and through the floor of the bursa. The inflammatory process binds the bursa and the cuff together and thus makes such a change in the position of the calcium possible. Further adherence between the bone, tendon, bursa and deltoid muscle leads to a gradually developing restriction resulting in the so-called frozen shoulder.

Open surgery is occasionally indicated to remove large calcific deposits after the usual conservative measures have failed. However the needling procedure and instillation of hydrocortone usually decompress the chemical "boil" and relieve the intense pain. The lower grade forms of bursitis in the shoulder will respond to heat, analgesics and stretching exercises designed to overcome the limited motion. X ray therapy has its enthusiasts when bursitis is in the acute stage, but most agree that in the chronic state with an associated fraying in the tendon cuff this type of treatment will not be effective. This is particularly true if night pain and restricted motion are featured.

In dealing with the painful shoulder it should be remembered that the mere presence of calcium in the rotator tendons does not necessarily mean that bursitis is present. Such calcium deposits are commonly seen in the routine chest films of patients without symptoms and so long as they remain deeply embedded, not involving the gliding surface of the tendon or bursa, they will be clinically silent.

No examination of the shoulder is complete without attention to the cervical spine. Tests to disclose the presence of reflex, motor sensory or trophic as well as al-

terations in the color temperature pulse and perspiration phenomena in the upper extremities, should always be performed. The shoulder pain, instead of being primarily about the joint may be referred from the cervical spine. Degenerative arthritic change protrusions of intervertebral disk material tumors, tuberculosis and congenital anomalies are conditions which may simulate shoulder inflammations.

READING REFERENCES

DePalma, A. F.: *Surgery of the Shoulder*. Philadelphia, J. B. Lippincott Company 1930.
 Garrett, E. L.: *The Syndrome of Rupture, Dislocation and Elongation of the Biceps Brachii*. An

Analysis of 100 Cases. *Surg. Gynec. & Obst.* 58:322, 1934.
 Jonsson, G.: *Malignant Tumors of Skeletal Muscle, Fascia, Joint Capsules, Tendon Sheaths and Bursae*. *Acta radiol. supp.*, 36.1 1938.
 Kendall, H. O., and Kendall, F. P.: *Muscles Testing and Function*. Baltimore, Williams & Wilkins Company 1919.
 Lewis, P.: *Brenneman's Practice of Pediatrics*, vol. 4 Chapter 37.
 McLaughlin, H. L., and Asherman, E. G.: *Lesions of the Musculotendinous Cuff of the Shoulder*. *J. Bone & Joint Surg.* 33-A:76, 1951.
 Meyerdier, H. W., and Chapman, J. P.: *Anserina Burniti*. *S. Clin. North America* 27:98 1947.
 Tillotson, J. F., McDonald, J. R., and Jones, J. M.: *Synovial Sarcomata*. *J. Bone & Joint Surg.*, 33-A:459 1951.

Acute and Chronic Infections of the Bones and Joints

By FRANCIS M. McKEEVER M.D. JOHN C. WILSON JR. M.D., and FRANCIS E. WEST M.D.

FRANCIS MICHAEL McKEEVER is a native Californian and received his education at the University of California. He was Chief of Orthopedic Surgery at the Army's large Percy Jones General Hospital during World War II. He serves as a senior orthopedic surgeon at the Children's and Los Angeles County Hospitals and teaches at the University of Southern California where he is Professor of Orthopedic Surgery.

JOHN CREEK WILSON JR., has followed in his father's surgical specialty. Also a native Californian, he was educated at Stanford University and Harvard Medical School. He is associated with Doctor McKeever in the Department of Orthopedic Surgery at the University of Southern California.

FRANCIS EDWIN WEST is a native California who impartially claims both the University of California and the University of Southern California as his alma mater. After a short sojourn at the University of Michigan, he returned to San Francisco to further his orthopedic surgical training. Following World War II in which he carried on his work as an orthopedic surgeon, he returned to San Diego where he practices his surgical specialty and has charge of the orthopedic section of a hospital for tuberculous patients.

Suppurative bacterial infection in bone is called osteomyelitis. Osteomyelitis in its strict interpretation signifies an inflammation of the marrow cavity only but by general usage

through the years this term has come to signify an inflammatory reaction in any or all parts of bone tissue. The term *periostitis* is used to designate specifically a pathologic

process which manifests itself by the production of increased bone immediately beneath the covering membrane and does not involve cancellous bone. It may be noninflammatory and is often the manifestation of neoplastic disease or a blood dyscrasia. It also results from a granulomatous infection such as syphilis.

Infection in bone occurs in two ways. The pathogenic organisms may be brought to the bone through the blood stream. When this is the method of infection, the bone disease is known as *acute hematogenous osteomyelitis*. Organisms may be introduced directly into a portion of the bone by a disruption of the soft tissues covering a part of the skeleton. This occurs with a compound fracture or perforating wound, when exposed bone becomes directly contaminated with bacteria. This type of bone infection is called *exogenous osteomyelitis*. A more descriptive designation is *osteomyelitis due to direct contamination*. Hematogenous osteomyelitis and osteomyelitis due to direct contamination have a different pathogenesis and are diseases with different manifestations, different prognosis and involve different surgical principles in their management.

Acute hematogenous osteomyelitis, promptly diagnosed and properly managed, has changed over the past twenty years from a malignant disease to a relatively benign illness. Since the advent of chemotherapy and antibiotics, the clinical course, sequelae and prognosis of bone infection from bacteremia have been greatly altered. Prior to 1937 acute hematogenous osteomyelitis was a disease with a high mortality and prolonged invalidism, often resulting in severe crippling and grotesque deformity. Since the development of powerful agents for bacteriostasis and a better understanding of fluid balance, the prognosis of blood borne osteomyelitis has been markedly improved. Antibacterial drugs, together with better appreciation of basic surgical principles, have also reduced the invalidism and loss of extremities from osteomyelitis due to direct contamination.

ACUTE HEMATOGENOUS OSTEOMYELITIS

Acute hematogenous osteomyelitis is predominantly a disease of infancy and childhood. The highest incidence is in children between the ages of one and ten years. It is seen less often in the first years of life but may occur even in the newborn. Acute hematogenous osteomyelitis is rare in the adult af-

ter closure of the epiphyses and the completion of growth.

Acute hematogenous osteomyelitis is a local manifestation in bone of bacteremia. The infecting organism most commonly recovered from acute hematogenous osteomyelitis is *Staphylococcus aureus* (*Micrococcus pyogenes var aureus*). Next in frequency is *Staph albus* (*St pyogenes var albus*) and then streptococcus, either alpha, beta or gamma. However any pathogenic bacteria may be the causative organism of osteomyelitis. Pneumococci, salmonellae, typhoid bacilli, enterococci and *Pseudomonas aeruginosa* have all been recovered from foci of osteomyelitis. The infecting organism is carried in the blood stream to the bone. The initial focus of infection very often cannot be found, as it is only a minute break in the skin or mucous membrane. At other times there is direct evidence of a portal of entry which may be a furuncle, an infected crusted abrasion, impetigo, omphalitis in a newborn infant or an abscessed tooth. On some occasions the onset of osteomyelitis is concurrent with or occurs during convalescence from acute tonsillitis, upper respiratory infection, pyelitis, or exanthematous disease such as scarlet fever.

The structure of the normal bone (Fig. 139) explains why hematogenous osteomyelitis is a disease of infancy and childhood. Although bacteremia to some degree is probably present as often in adults as in children, in adults organisms do not localize in bones early, therefore natural body defenses have an opportunity to bring about the death of the bacteria in the blood stream before localization results. During infancy and childhood, which are periods of rapid growth, the epiphyseal metaphyseal junction, where cartilage is being converted to bone requires a rich blood supply. The arrangement of the blood vessels in the metaphyseal area of growing bone is such that it predisposes to the focalization of any bacteria which may be free in the blood stream.

At the line of ossification in the bones of children there is a continuous proliferation of capillaries. The anastomoses between the vessels of the metaphysis and epiphysis result in many arterioles with a blind end. The sum total of this anatomic arrangement is a very slow movement of a very large supply of blood, which almost becomes pooled. This situation presents an ideal condition for bacteria to take tenancy and multiply.

With the lodgment of pathogenic bacteria in bone there is a cellular response with ex-

infection and the formation of an abscess. If the immunologic reaction is strong or when bacteriostatic agents are brought promptly to the site of infection, the bacteria may be rendered ineffectual and the abscess will be promptly localized. However, if the bacteria multiply because of weak bodily defenses or late diagnosis and the withholding of antibiotics, the abscesses which form may coalesce and extend, causing destruction and absorption of bone structures. The many microscopic canals in the haversian system are enlarged and eroded, and the products of inflammation extend through them beneath the periosteum. Periosteum in the bones of children is loosely attached to the cortex. The loosely attached periosteum is stripped from the cortex to an extent which is dependent on the pressure of exudate. When the abscess is permitted to persist and extend, the periosteum finally ruptures and the pus decompresses itself into the soft tissues covering the bone. Exudate in the cancellous bone of the metaphysis also forces its way to the epiphyseal junction and at this point soon extends beneath the periosteum. The bone destruction in the haversian canals and the elevation of periosteum deprive the denuded cortical bone of its blood supply so that metabolic processes cease in that portion of bone, which becomes inert, calcified tissue or dead bone. The dead bone which thus results is termed sequestrum. Various gradations of this process may result, from a promptly attenuated

microscopic abscess which may never be demonstrable by any x ray change to massive death and complete sequestration of the entire shaft of the bone. The outcome of the initial infection in bone depends on the virulence of the invading bacteria and on the resistance of the individual. The patient's resistance can be immeasurably improved and the virulence of the infection greatly diminished by early diagnosis and prompt institution of intelligent treatment.

The symptoms and signs of acute hematogenous osteomyelitis in infants under one year of age are different from those in children from one year of age to adolescence. Similarly in adults after epiphyseal closure and completion of bone growth, the symptoms show another pattern. These varying manifestations result from the anatomic differences in bone and the physiologic variations in individuals at progressive ages.

In the newborn full-term infant, the central nervous system is far from completely developed. Sensibility, though present, is not very acute. Deep muscle and joint sensibility is not highly developed until from eight to ten months after birth, when the infant first tries to stand. In the early months of life systemic reaction to infection may be sluggish. At birth and for several months following, there is some persistence in the extremities of the intrauterine position of flexion of the trunk with fixed flexion contractures of the hips. In addition, in the bones of infants

Diagram of Localization and Spread of Pyogenic Abscess in Bone

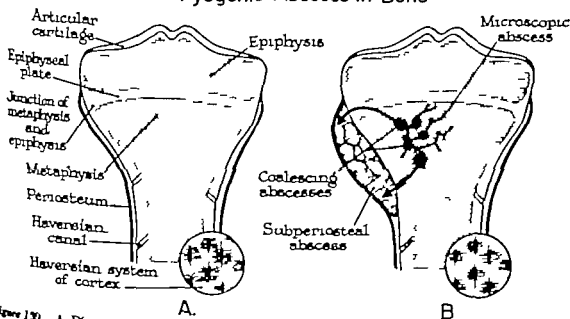


Figure 130 A, Diagrammatic sketch of structure of normal bone. B Diagrammatic illustration of pathologic changes which ensue in acute hematogenous osteomyelitis.

there is massive pooling of blood in blind capillaries at the zone of calcification. At the epiphyseal plate the adjacent cortical bone is very thin and like papier-mâché and the periosteum is very loosely attached about the metaphysis, an anatomic arrangement which permits the early escape of exudate at the metaphyseal-epiphyseal junction. These facts make it evident that in an infant it is easy to fail in making an early diagnosis of acute osteomyelitis. The first sign is usually elevation of temperature, which may be marked to 39 or 40 C. or need not be very pronounced. Very shortly after this there appears a swelling of the soft tissues over the part involved which is rapidly followed by a refusal on the part of the infant to move an extremity. Thus loss of function develops. Examination often reveals a flexion contracture of the joint adjacent to the infected bone. This deformity may be hard to determine definitely at the hip because of the persistence of the intrauterine position. Restriction of normal passive motion in the joint is also present and can be determined by comparison with the normal extremity. The evaluation of restricted joint motion in an infant is difficult because of a certain normal amount of rigidity and mass reflex action. The leukocyte count is generally elevated, strikingly so at times, at others only mildly. Roentgen ray examination in the first forty-eight to seventy-two hours of the disease gives little information. From five to seven days after onset of the illness in an infant, x-ray study often shows evidence of bone involvement, but this is very late in the disease and by the time x-ray examination reveals the lesion, the abscess has ruptured into soft tissues. The disease being usually juxtaepiphyseal the epiphysis has already been badly damaged and a growth disturbance can be anticipated. The adjacent joint is ordinarily distended with pus by the time a roentgenogram shows the lesion. The most frequent localization of osteomyelitis in infants is the upper end of the femur next most frequent is the lower end and upper end of the tibia, where the hip and knee joints are in jeopardy. The upper extremities, though less frequently involved, are not spared and any bone may be infected. Multiple foci are more common in infancy than at any other time of life.

In children from one year of age to adolescence, bodily reactions are more stabilized, and the central nervous system has reached physiologic maturity. The epiphyseal-metaphyseal function has become more resistant to the escape of exudate and the periosteum

more rigidly attached to the cortex in the progress to adulthood. The commonest sites of osteomyelitis in the child up to adolescence are the lower end of the femur the upper end of the tibia and the upper end of the femur. The earliest symptoms of acute hematogenous osteomyelitis in the child over one year of age are those of infection, which, of course, will vary with the virulence of the infecting organism and the patient's resistance. Although the initial temperature rise may be slight, the disease may at other times be ushered in with a chill and a very sharp febrile rise, which is probably the manifestation of the bacteremia. Vomiting with concomitant dehydration and alteration of fluid and electrolyte balance often occurs. Pain is an early symptom. It is usually constant, severe and aggravated by any motion or jar. The cause of this is the expanding exudate in the bone. When the periosteum ruptures and introsseous tension is released, the patient is more comfortable. Examination sometimes reveals an obvious portal of entry for bacteria, such as a furuncle, crusted abrasion or large succulent tonsils. There may be a history of proximate infectious disease, an upper respiratory infection or even an eczema. Early swelling over the painful area is absent. However there is an area of definite tenderness to pressure or percussion, which is more easily detected in a superficial bone like the tibia or ulna than in bones covered by bulky muscles. Careful examination will detect it even in the femur. If the focus is juxta-articular there may be spasm of the muscles about the joint, accompanied by limitation of motion, the adjacent joint being held in a position of comfort by this protective muscle spasm. The knee will be held flexed and the hip in a position of flexion and abduction. The joint even though not invaded by bacteria, may present an effusion due to adjacent inflammation. This often happens in the knee joint when the osteomyelitic focus is in the lower end of the femur.

The white blood cell count is generally markedly elevated, the count possibly ranging from 25 000 to 40 000 cells per cu. mm. of blood, with a marked preponderance of polymorphonuclear leukocytes. The blood culture may be positive, although in over half the patients with hematogenous osteomyelitis, because the bacteremia is most often transient, no organisms will be grown at the time the blood is taken for culture. In the early course of the disease roentgenograms are of no value except to rule out trauma. The earliest roentgen ray change is an area of vague de-

mineralization in the metaphysis or a punched-out defect at the epiphyseal junction. This change is rarely present at seven days and usually not until from ten to fourteen days after the onset of the infection. *For therapy to be effective the diagnosis must be established long before roentgen ray changes are evident.*

Acute hematogenous osteomyelitis in the adult after completion of growth is a rare occurrence. In such patients the symptom complex has no established pattern. It does not have the same proclivity for localization in the metaphyseal area as in the child. Not infrequently the infection is located quite a distance from the ends of the long bones. The disease is rarely fulminating, as in the infant or child. The infection may be ushered in by a sharp rise in temperature with general malaise, but more often the onset is subacute with intermittent bouts of mild fever and a feeling of fatigue and exhaustion. Pain may not be pronounced early but generally becomes established and asserts its character in from ten to fourteen days. It is dull and boring in nature, and often worse at night. Much less distressing than in a child it may be relieved by simple anodynes. It does not manifest itself strongly until the periosteal covering, which is tightly applied to the cortex, becomes irritated.

Examination most commonly reveals an area of tenderness to pressure or percussion, which can be detected even in those bones well covered by soft tissue. Late in the disease, after an abscess has emptied into the soft tissues, fluctuation may be palpable. However because of the great resistance of the mature calcified cortex, this may never occur and the abscess may be entirely confined within the bone, which shows enlargement with a very marked thickening of the periosteum. When periosteal reaction is pronounced, it is accompanied by muscular atrophy and the bone enlargement due to periosteal proliferation can be palpated. If the focus of osteomyelitis is juxta-articular there may be a contracture of the adjacent joint due to protective muscle spasm. This contracture is aggravated by activity and relieved by rest.

The number of white blood cells may be increased but usually to only a slight degree. The sedimentation rate is generally elevated. Early roentgen ray examination is unrevealing. It may show nothing even as late as the third week of illness. The earliest roentgen ray change is an area of radiolucency accompanied by slight periosteal thickening.

Even though changes are far advanced after the disease has been existent several weeks, there is often nothing pathognomonic about the roentgenograms. Roentgen ray changes resulting from infection in adult bone are frequently indistinguishable from those produced by malignant disease or blood dyscrasias. The finding most characteristic of infection is the presence of a definite sequestrum which is only seen very late in the course of the illness. Usually not until after changes are evident in the roentgenogram is acute hematogenous osteomyelitis in the adult diagnosed with certainty. By this time the disease has passed into the subacute or chronic stage. Biopsy of the bone will often be necessary to differentiate the process from neoplastic disease.

Although hematogenous osteomyelitis of the vertebral column and its appendages is rare its symptomatology is sufficiently different to warrant special mention. The initial signs of infection may be severe; however the signs localizing it to the vertebral column are so obscure that the first definite evidence of localization may be neurologic. If exudate drains into the epidural space causing pressure the neurologic signs will vary with its location in the spinal canal. When the epidural abscess originates from an osteomyelitic focus below the second lumbar vertebra, where the cauda equina and not the spinal cord occupies the canal, there will be nerve root signs and signs of meningeal irritation, but without paralysis. The earliest physical finding is a positive Lasègue sign or spasm of the hamstring muscles which restricts the range of flexion of the hip with the knee extended. If the abscess originates above the second lumbar vertebra and occupies the neural canal in the area of the spinal cord itself there may be rapidly progressive transverse myelitis with a sensory level and paralysis. Prompt and early decompression of the spinal canal by laminectomy and drainage is indicated if irreparable cord damage is to be avoided.

The general physical examination of a patient suffering from acute hematogenous osteomyelitis should be thorough and complete. A careful search of the skin may reveal infected cuts or abrasions, crusted papules or follicular infections, which may be the source of entry for the causative organisms. Mucous membranes must be carefully inspected with particular attention being given to the tonsils. Both middle ears should be examined for the presence of infection. The existence of an upper respiratory infection or the prodromal

phase of an exanthematous disease can often be detected by careful scrutiny of the mucous membranes. Auscultation and percussion of the lung fields and a careful evaluation of the heart are important. Abnormal murmurs may suggest endocarditis. The osteomyelitis may be secondary to the embolic seeding. Abdominal distention from alteration in fluid balance or from toxemia will give additional information as to the patient's general condition. An enlarged spleen or liver may indicate a blood dyscrasia. Tenderness in the costovertebral angle kidney infection. Stiffness of the spine may point to perinephric, spinal or meningeal infection. In an adult a history of typhoid fever may be suggestive since, in these patients, bone infection may occur many years following the initial systemic disease, as a result of liberation of typhoid bacilli in the blood stream from a chronic focus.

Local examination of the suspected site of infection should include observation for the presence or absence of swelling. The maintenance of an extremity in an abnormal posture from protective muscle spasm of an adjacent joint often helps to localize the site of infection. The range of motion present in the joints above and below the suspected site of infection and its departure from normal should be determined. Restriction of motion is especially difficult to determine accurately in infants but if the motion in the suspected joint is compared with that of the normal side, errors will be few. Palpation for sites of tenderness which are present early is very important. Percussion over a site of tenderness will help to verify its location. This part of the examination should be done last in the child, as it is very often so distressing that if done early the remainder of the examination is rendered difficult. The clinical examination yields the most information. Laboratory tests help in verifying clinical findings and in the estimate of the patient's total reaction to infection.

The patient should have an initial red blood cell count, white blood cell count, hemoglobin test and a differential count of the white blood cells. From this information the necessity for transfusion can be appraised and with repeated blood counts the course of the infection can be followed carefully. A blood culture should be made as soon as possible, although the blood may be sterile as the bacteremia is often transitory. Blood for the culture should be taken when the temperature is elevated and it is best to take two or three samples at different times rather than

only one. The initial sample should be obtained before antibiotic therapy is instituted. Organisms obtained in the blood culture should be tested for sensitivity to antibiotics. Moreover a culture should be made from material from any local lesion in the skin which is infected from an obviously infected throat or from a draining middle ear. Urine cultures should be obtained if the microscopic examination of the urine sediment indicates findings suggestive of an infection; stool cultures, if enteritis is suspected.

The urine should be analyzed at the initial examination and at least weekly during the acute illness. Acetone in the initial urine may indicate severe dehydration and acidosis. Occasionally an unsuspected diabetic will be detected. Such discovery alters the prognosis in any infection and gives definite therapeutic indications. The appearance of red cells and casts in subsequent urine specimens may forewarn of developing nephritis. Serologic studies on the blood should be made routinely during the initial laboratory survey. These latter findings are of more importance in the adult than in the child.

The roentgenogram records only change in density in bone. In acute osteomyelitis, time is required for the bony structure to be altered by decalcification to the extent that the x ray film will record these changes. As the bone becomes more mature a longer time is required for these changes to take place. Consequently in the initial stages of hematogenous osteomyelitis, the roentgenogram shows no abnormality as far as bony contour and structure are concerned, however soft tissue swelling and alterations in the soft tissue planes are sometimes of help in localizing the infection, if compared with roentgenograms of the normal side. In the child over one year of age, x ray study seldom shows bone changes prior to from ten to fourteen days. In an adult, longer periods are required for alterations in bony density. However it is of great importance to obtain an early x-ray picture of the suspected area to compare with later films. The roentgenograms should be obtained in at least two planes at right angles with each other and the joint above and below the suspected area should be visualized. A roentgenogram showing no abnormality does not exclude the possibility of acute hematogenous osteomyelitis if clinical findings warrant the diagnosis.

Any pain in an infant or child, accompanied by tenderness in the region of a bony metaphysis with concomitant signs of infection should be regarded as due to acute hematog-

eous osteomyelitis until proved otherwise. However pathologic conditions in the infant or young child which may be confused with acute hematogenous osteomyelitis are many. Scurvy may manifest itself by bone tenderness. Complete examination will usually reveal corroborating evidence, such as ecchymosis or petechiae or both. Bleeding gums are not seen before teeth have erupted but there may be stomatitis. A ray examination will show characteristic changes. The plasma ascorbic acid level will be low. Before the age of six months and after the age of eight months, scurvy rarely occurs.

Infantile cortical hyperostosis should offer very little difficulty in differential diagnosis being recognizable by its mild systemic manifestation, the frequent involvement of the mandible and the pronounced soft tissue thickening covering the involved bones. It most often involves the clavicle, scapula and humerus.

Hypervitaminosis A can be differentiated by systemic symptoms that precede it—anorexia, itching and fretfulness of weeks duration. There is always a history of more than adequate vitamin A intake and the systemic reaction is mild.

Cellulitis may be confused with acute osteomyelitis in an infant or child. At the onset of cellulitis, the signs of infection, pain and tenderness of the skin may be misleading. However osteomyelitis in its early stages is rarely accompanied by reddening or induration of the skin, which after forty-eight hours is usually pronounced in cellulitis.

Pyogenic infection of a joint may be secondary to osteomyelitis in the adjacent metaphysis. Joint infection however is most often primary. Swelling from intra-articular effusion is readily detectable in superficial joints, such as the knee, wrist and ankle. Deep-seated joints, such as the hip, manifest acute infection by marked restriction of motion, extreme pain on motion and pain on palpation, as well as the associated signs of infection. Aspiration of the joint with examination of the synovial fluid will determine whether it is infected.

Rheumatic fever should not offer great trouble in differential diagnosis. The manifestations of this disease are in the joint and not juxta-articular. Usually one or more joints are involved simultaneously or in rapid sequence. Moreover rheumatic fever is rare in children under four years of age.

Leukemia in an infant or child may manifest itself with acute bone pain and tenderness. The general physical examination will

probably reveal enlargement of the lymph glands or the spleen. The differential blood count should also give helpful information. The crisis of sickle cell anemia may cause acute bone pain and tenderness with subperiosteal hemorrhages and in a Negro patient it is to be considered as a possible cause of these symptoms. Careful blood study will reveal the abnormal erythrocytes and establish the diagnosis.

In the adult with complete growth and closure of all epiphyses it is often impossible to differentiate the roentgen ray findings of hematogenous osteomyelitis from those of a malignant bone tumor. The roentgenographic changes in the former so closely mimic those which result from malignant neoplastic disease that exploration and biopsy are the only methods of establishing a definite diagnosis.

The treatment of acute hematogenous osteomyelitis is twofold: prophylactic, or measures taken to prevent the disease, and therapeutic or treatment of an already established case.

From the prophylactic viewpoint a certain number of instances of blood borne osteomyelitis will be prevented if attention is given to superficial cutaneous infections and furuncles. Abrasions should be properly dressed so that they do not become crusted over and retain infected secretions. Furuncles should be drained when localized and not traumatized by squeezing. In the stage of diffuse cellulitis, they should be treated by warm applications and antibiotics if indicated. Persistent and recurrent acne should be properly treated and eliminated. Tonsillitis and upper respiratory infections should be respected and the child so afflicted made to avoid fatigue and receive proper therapy. Infected and decaying teeth with abscesses in the gums should be removed. Any other persistent haven of pyogenic bacteria, such as persistent pyelitis or an ingrowing toenail, should receive proper care. By elimination of the portal of entry for bacteria, bacteremia may be avoided.

The treatment of acute hematogenous osteomyelitis is both medical and surgical. With early diagnosis and promptly instituted medical treatment, many patients with acute hematogenous osteomyelitis will recover without surgical drainage of bone. However medical treatment should not be persisted in to the exclusion of surgical treatment when there is evidence of pus under pressure in the bone or soft tissues. These two types of treatment are not antagonistic; they are complementary and the appropriate application of each with

proper timing will produce the best results. The medical phase of the treatment consists of the following procedures: proper attention to the general condition of the patient, with restoration of fluid and electrolyte balance, maintenance of adequate caloric intake, prompt institution of high blood concentration of multiple antibiotics with the broadest possible antibacterial spectrum and maintenance of this concentration until it is possible to determine the specific antibiotic for the infecting organism, at which time this single antibiotic may be substituted, the use of antipyretic measures if fever is inordinately high in infants and children, drainage of any obvious purulent focus, such as a furuncle or crusted abrasion, which is not draining.

Many infants and children, following the onset of osteomyelitis, will be dehydrated and have acidosis. They may have severe anemia, with a low hemoglobin level and red cell count, as a result of destructive activity of the bacteremia on the blood cells. If severe anemia is present, transfusions may be indicated. To combat and rectify dehydration and also supply part of the caloric requirement, glucose in amounts suitable to the patient's weight should be administered intravenously. Potassium, as indicated may be added to the intravenous fluid. If the child cannot take medication by mouth, vitamin B and ascorbic acid in appropriate dosage may be added to the intravenous fluid. As improvement occurs and the patient resumes oral intake the intravenous administration of fluids may be decreased or discontinued. An adequate caloric intake with sufficient protein content should be striven for. Protein concentrates may be necessary to accomplish this.

Antibiotics must be administered promptly and continuously if a high blood concentration is to be maintained. Because the invading organism is usually not obtainable for typing as to sensitivity multi antibiotic therapy should be started as soon as possible to eliminate the possibility that resistant strains of bacteria may thrive. Even though the infecting organism may not be available for culture and typing, the probabilities are 93 to 98 per cent that the invading organism is either *Staphylococcus aureus* or a streptococcus. Unless there is clinical evidence such as enteritis, which might implicate salmonella, or some other clinical fact pointing suspiciously toward an unusual organism as the causative factor the antibiotics used should be those which are effective against staphylococci and streptococci.

In the light of the present effectiveness of antibiotics an excellent combination for broad-spectrum coverage against the staphylococcus and streptococcus is erythromycin, oleandomycin or tetracycline with chloramphenicol. Penicillin is no longer the drug of choice because many strains of staphylococci are insensitive to it and allergic reactions to it may occur. Erythromycin should be given in amounts of 30 to 40 mg. per kg. of body weight for twenty-four hours, oleandomycin in amounts of 75 to 100 mg. per kg. of body weight, tetracycline in amounts of 100 mg. per kg. of body weight. The dose of chloramphenicol is 75 to 100 mg. per kg. of body weight. If there is a history of previous therapy with chloramphenicol, this drug should not be used unless there is a failure of clinical response to a combination of the other antibiotics. In the infant and young child, whose cooperation is questionable, and even in the older severely ill child, antibiotics are best given in a continuous intravenous drip.

This intensive multi antibiotic therapy should be continued until there is definite general improvement and a drop in fever which will usually result after three or four days of treatment (Fig. 140). If the initial blood culture is positive, and an antibiotic specific for the infecting organism can be determined, other antibiotics may be discontinued and the specific antibiotic substituted in adequate dosage. When the patient will take nourishment and medication by mouth, intravenous therapy should be discontinued and oral therapy instituted. Antibiotic therapy should be continued for at least three weeks after the patient's temperature is normal to insure bacteriostasis in the body focus. If roentgenograms show any bone lesion, antibiotic therapy should be continued for a period of six weeks after the temperature has become normal. While membranous colitis is not a common complication of antibiotic therapy in children, if it becomes necessary to administer antibiotics for weeks it is well to administer an antifungal drug, either separately or in combination with the antibiotic, to avoid moniliasis.

When marked hyperpyrexia is a feature of the illness, alcohol sponges and an antipyretic drug, such as acetylsalicylic acid in adequate doses, should be used.

Surgical treatment of a possible primary focus of infection has already been mentioned. Any such focus should be insured prompt and adequate drainage. If during the course of the medical treatment of an acute osteomyelitis it is reasonably certain that there

is a collection of frank pus either subperiosteally or in the medullary canal of the bone drainage should be promptly instituted. Surgical drainage should be done with the patient under appropriate anesthesia and, when possible, with a tourniquet on the extremity so that a clear bloodless field may be obtained. The operation must furnish adequate drainage and the incision should be packed with petrolatum gauze to keep it open so that it can granulate from the depths to the surface. When surgical drainage is performed in acute hematogenous osteomyelitis the incision should not be closed. At the time of surgery the periosteum should be lifted from the cortex of the bone only to the degree that is necessary to permit removal of enough cortex to produce adequate drainage. The operative procedure should be planned so that there is not wanton stripping of periosteum and so that no unnecessary cortex is removed. Splinting of the joint above and below the operative site will give added comfort in the postoperative period. This can be accomplished by means of molded splints cast or traction. The stress of anesthesia and surgery should never be placed on a patient who is dehydrated, who has acidosis or who has severe anemia. These conditions can always

be rectified before the patient is subjected to surgery. At the time of the initial surgical drainage in the acute stage of the illness it is impossible to determine whether sequestration is going to result and, if it does to what extent. For these reasons, only enough bony cortex for adequate drainage should be removed. Even though pus is found in the soft tissue or immediately beneath the periosteum, a window should always be made through the cortex for drainage from the medullary canal.

The intelligent application of both medical and surgical treatment and the proper timing of well planned surgery, based on daily complete examinations of the patient, will result in the quickest recovery from acute hematogenous osteomyelitis. Recurrence of the infection and unfavorable sequelae (Fig. 141) will be reduced to a minimum by this program.

A discussion of the prognosis of disease due to bacterial infection should take into account that what is true today may be at variance with the past and may well not hold in the future. The era of chemotherapy and antibiotic therapy in which we now live has rather effectively combated the many pathogenic bacteria which were so devastating to

TEMPERATURE CHART

NAME R.L.

AGE 20 years

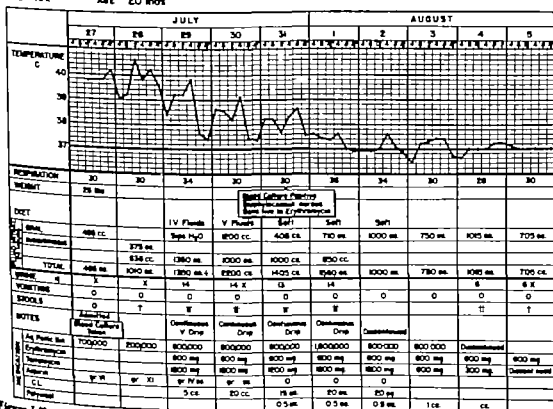


Figure 140 Clinical chart to illustrate the medical treatment of acute hematogenous osteomyelitis, with a common response when adequate treatment is instituted early. This patient recovered without surgical drainage; roentgenograms three months after illness revealed only minor bone changes.

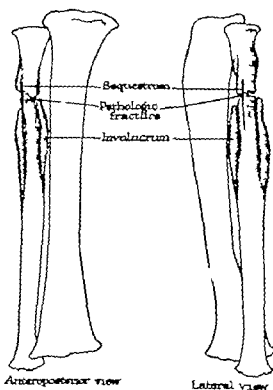


Figure 141 Drawing of roentgenogram of acute hematogenous osteomyelitis in the fibula of a two-year-old child. Prompt adequate medical treatment was instituted. However an obvious abscess was not drained surgically. Note the extensive destruction of bone, sequestration and pathologic fracture which might have been avoided by properly timed surgical drainage.

human tissues in the past. With the passage of years the balance of power between therapeutic agents and the invulnerability and toxicity of pathogens may shift. When this results, the prognosis in acute hematogenous osteomyelitis may be altered.

Today the prognosis in acute hematogenous osteomyelitis varies with several factors: the age of the patient at the time of onset of the disease, the promptness of diagnosis and the adequacy of treatment. In infants under one year of age acute hematogenous osteomyelitis has some mortality. The death rate is about 5 per cent. In those who survive there is a high incidence of crippling as a result of growth disturbances resulting from rapidity with which the epiphyses are destroyed by infection. Discrepancies in extremity length and aberrations in directional growth, such as genu valgum, genu varum and radial deviation of the hand, are common. Disruption of the hip joint, with pathologic dislocation, is also a relatively common condition resulting from osteomyelitis in infancy. The sooner pus is evacuated, the less frequently such distressing sequelae will occur. Regrettably di-

agnosis in the infant is often very late in being established. In one well-studied series of twenty-five patients, only seven escaped deformity from epiphyseal damage. In most of those free of deformity, diagnosis was made in the first seventy-two hours of their illness. The mortality rate in children over one year of age and in the adult is extraordinarily low. Death results in not more than 1 or 2 per cent of the patients. When the diagnosis is established in the first forty-eight hours of bone infection and treatment with antibiotics is adequate and continued long enough, 40 per cent of the patients can be expected to experience resolution of the inflammation and recover without surgical drainage of bone, with very little residual change from the normal bone demonstrable in subsequent roentgenograms. An additional group, perhaps 5 to 10 per cent, will recover but at a later date—from three to six months—and will present a small sequestrum which will require surgical removal. Complete recovery is usually prompt after sequestrectomy. About 50 per cent of children will have an abscess which requires surgical drainage even though diagnosis has been early and treatment adequate. A greater number of this group will require a second operation for sequestrectomy.

Although marked epiphyseal disturbance with aberration of growth is less frequent in patients above the age of one year than in those younger, it will occur in approximately 10 per cent of patients. Dislocation of the hip joint does not occur unless the child is deprived of medical observation and treatment is delayed. Massive sequestration of the entire shaft of a bone practically never occurs in patients of the younger age group when diagnosis is prompt and treatment intelligent and adequate. Multiple bone foci are uncommon also when treatment is prompt.

In the adult, after completion of growth and epiphyseal closure, hematogenous osteomyelitis is a rarity; consequently statistics regarding mortality are not available, but death is probably infrequent. Deaths which do occur are largely in the group of patients having rare cases of primary fulminating osteomyelitis of the spine, or of a bone of the skull, such as the mastoid process of the temporal bone. Death then results because of a spill of septic material into inaccessible areas. Growth disturbances do not occur in adults and joint destruction is not common, since the localization of osteomyelitis in the adult does not have the same predilection for the ends of bones as in the child.

CHRONIC HEMATOGENOUS OSTEOMYELITIS

Acute hematogenous osteomyelitis occurs in varying degrees of severity dependent on the relationship between virulence of the infection and host resistance. Today, although most patients with acute hematogenous osteomyelitis rarely enter the chronic stage when diagnosis is early and treatment adequate, nevertheless there are a few who, despite adequate early treatment, do not recover promptly but suffer a chronic phase of the disease, characterized by persistent sequestration of bone and continuous drainage from sinuses.

In the acute stage of hematogenous osteomyelitis, the purulent exudate of inflammation extends through the haversian system and out the epiphyseal junction beneath the periosteum to form an abscess and strip the periosteum from the bone. This is accompanied by thrombosis of blood vessels which causes a loss of blood supply to the calcified cortex and trabecular bone of the metaphysis and death of that portion of the bone deprived of blood. As the acuteness of the process subsides and the patient gains dominance over the invading bacteria, granulation tissue forms beneath the periosteum and in the medullary canal about the devitalized area of bone. This granulation tissue has many osteoclasts, or cells, which remove bone. When given enough time, this osteoclastic granulation tissue demarcates the dead bone from the live bone and at the junction of live and dead bone cuts a ridge. After a sufficient period, the dead bone is actually separated and cut off from the live bone and is completely surrounded by granulation tissue, or sequestered from the living bone. Hence the separated dead bone is called a sequestrum. The presence of a fragment of dead bone surrounded by granulation tissue in a cavity with rigid walls is conducive to continuous growth of bacteria and results in persistent drainage. A minute sequestrum may ultimately totally dissolve in the exudate or may extrude itself with cessation of drainage and spontaneous closure of a sinus. If the sequestrum is of appreciable size its dissolution or extrusion can take place only after years of drainage or it may never occur.

After sequestration has occurred, the elevated periosteum, because of its osteogenetic function, starts a reparative process (Fig 142). On the undersurface of the periosteum over and around the devitalized cortex, new bone is formed which has a blood supply and can survive. This new bone is formed to

replace that which has been lost by sequestration. It is separated from the sequestered bone by granulation tissue and is usually perforated with sinuses called cloacae to permit the escape of pus. The new bone formed over the dead bone is called involucrum. The involucrum may and often does form so that it encloses the sequestrum or entraps it. Removal of a sequestrum may require sacrifice of a portion of the involucrum. This sequence of sequestration involucrum formation naturally leads to cavities with rigid bony walls which cannot collapse even after the escape or removal of a sequestrum. Non collapsible cavities are a fertile ground for bacterial growth and of themselves are a cause of persistent drainage even though not housing a sequestrum.

The symptoms of the chronic phase of os

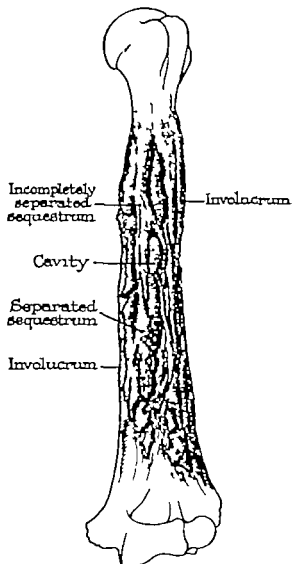


Figure 142 Drawing of roentgenogram of chronic stage of osteomyelitis of shaft of humerus, with chronic sinuses, large involucrum and multiple sequestra.

osteomyelitis are few provided drainage is constant and adequate, that is provided the sinuses are open and permit exudate to escape. When a sinus closes so that exudate is retained, pain will result and all the signs of inflammation, consisting of swelling, local heat and elevation of temperature may follow. Leukocytosis and anemia may occur if retained infection is not relieved.

A patient who has very extensive sequestration involving all or most of the shaft of a long bone, with drainage which is adequate to prevent an acute reaction but inadequate to drain the involved area completely may show signs of chronic infection. The large focus of infection may produce elevation of temperature, sweats, loss of weight, weakness and general malaise and the patient may have persistent microcytic anemia, mild leukocytosis and elevation of the blood sedimentation rate. Unrelieved chronic infection of this type leads to cloudy swelling of parenchymatous viscera and, if allowed to persist indefinitely may result in amyloid disease. The cloudy swelling of the parenchyma of the kidney may give rise to albumin and casts in the urine.

The patient presenting chronic draining sinuses following hematogenous osteomyelitis should be subjected to a general physical examination to determine his physical condition and to ascertain whether there is evidence of infection elsewhere. This individual, being a patient who at some time or other has had bacteremia, may have other sites in which there have been metastatic foci. He may have an infected genitourinary tract or an encapsulated pleural effusion. The initial source of bacteremia, such as an alveolar abscess or badly infected tonsil may still be present. The condition of the genitourinary tract, the cardiovascular system and the lungs should be evaluated. Chronic infection may produce severe depletion of the red cells, the hemoglobin and the plasma protein which would interfere with wound healing under the most favorable circumstances.

Complete and thorough x ray study of the involved bone should be made. X ray films should be exposed in at least two planes at right angles to each other. Oblique projections may be indicated when conventional anteroposterior and lateral views do not give the desired information. If there is very dense, extensive involucrum, it may be desirable to take films with an increased degree of exposure to penetrate the involucrum and identify sequestra.

Examination of the sinus by insertion of a

flexible steel probe or catheter may give valuable information. A small sequestrum which the x-ray study will not disclose may often be discovered by this means. Small sequestra of this type can be felt with a probe and occasionally extracted from the sinus. Such an examination also determines the direction in which a cavity or sequestrum may extend in relation to the mouth of the sinus. Roentgenograms made after the injection of a radiopaque material, such as Lipiodol, inserted into the sinus through a catheter will give information about the architecture of the problem which is not evident in conventional x ray films.

The object of treatment of the chronic stage of hematogenous osteomyelitis is to terminate drainage from sinuses by restoring the bone to as nearly a normal state as possible. This is accomplished by the following procedures: removal of sequestra, preservation of the involucrum, obliteration of all non-collapsible cavities with rigid walls in the bone in order that they may fill from the bottom with healthy tissue, and obtaining good soft tissue coverage for the involved bone. Antibiotics have their place in accomplishing this end but no antibiotics can remove sequestra or obliterate noncollapsible cavities. To use them to accomplish this not only wastes good drugs, but unnecessarily depletes the patient's resources and wastes his time. Antibiotics are nevertheless a valuable adjunct in the treatment of this stage of the disease. Their function is to work in conjunction with intelligent well-planned surgery. They should be used preoperatively for a short time to protect the patient against dissemination of bacteria incident to the surgery they should be used in the postoperative period to hold in check any increase in virulence the bacteria may gain at that time. After the operative site presents no evidence of infection and the patient shows no systemic signs of it, these drugs have served their purpose.

Operation in the chronic phase of hematogenous osteomyelitis should be deferred until a reasonable estimate of the amount of bone that may be sequestered can be made. Operation does not need to be deferred until sequestration is complete, as this may lead to undue invalidism. Good roentgenograms, properly interpreted, will outline the extent of devitalized bone long before the sequestrum is completely separated. At this time when the bone is exposed, a definite demarcation will be seen. The dead bone appears ivory like and the live bone pink and is an etched

line or groove between the two areas. At the time of surgery as little attached periosteum as is required to accomplish the proper exposure should be lifted. Unnecessary stripping of periosteum from the cortex may lead to further devitalization of bone and progressive sequestration. Involucrum may have to be sacrificed to free a sequestrum which is incarcerated in its meshes. This should be done with as little sacrifice of the new bone as possible. After the removal of all sequestra any cavity with rigid walls and undercut

shelving or roofing edges should be obliterated so that there is a gentle slope from the superficial edge of the bone into the bottom of the cavity. A well-sculptured cavity can be completely filled from the bottom by blood clot, which gradually turns to fibrous tissue, thus obliterating dead space. Cavities in bones which are well covered by muscle can be filled by pulling vascular muscle flaps directly into them. When the infected bone presents multiple-roofed cavities rather than a single cavity the removal of bone should

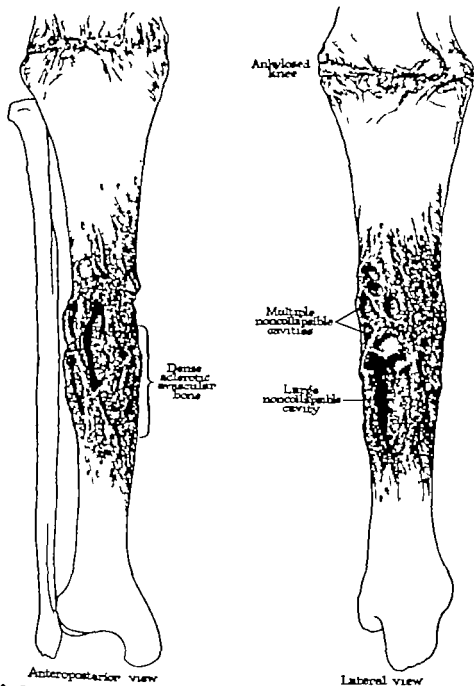


Figure 143. Drawing of roentgenogram of old chronic osteomyelitis of tibia with extensive sclerosis of entire bone, multiple bone cavities and ankylosis of knee joint. Because of extensive disease and avascularity of bone and soft tissues, surgical attempts to terminate drainage were unsuccessful and amputation was necessary.

be planned so that all or several small cavities are converted into the fewest possible large exenterated areas.

After removal of sequestra and creation of a bony cavity which has no overhanging roofing edges or noncollapsible tunnels the decision must be made whether to close the soft tissues over the bone or to place a petrolatum pack in the wide-open wound and permit the cavity to granulate from the bottom to the surface. This is a decision which requires experience and judgment. By and large, it is safer to leave the cavity open for drainage. However in certain selected cases more rapid convalescence may be effected by immediate closure of the wound. Certain principles to follow in making this decision may be suggested. An area extremely contaminated by much gross pus is safer when left open. The soft tissues must be adequate, well vascularized, flexible and must fall easily into the cavity. It is futile to obliterate a cavity with inflexible avascular scar. Consequently immediate closure should be restricted to those bones well covered by good muscle. It is rarely applicable to the subcutaneous area of the tibia, where chronic osteomyelitis so frequently occurs.

After completion of the operation the extremity should be immobilized in a splint or cast to insure physiologic rest. When the incision has been closed primarily this immobilization should be continued until it is soundly healed. When it has been left open, the use of the cast should be continued until healthy granulations approach the surface of the wound. The dressings and casts may be changed at intervals so that odors from drainage do not become offensive.

The prognosis for the termination of drainage and the restoration of good function depends on the extent of the pathologic process present and on the degree to which the involved bone has become sclerotic and avascular (Fig. 143). Generally the prognosis is good and most cases of chronic osteomyelitis can be terminated with cessation of drainage and restoration of a good extremity. When much ivory-like avascular bone is present and large segments of soft tissue have been replaced by avascular scar closure is sometimes impossible because of the diminished ability of the bone to resist bacterial invasion. At other times the architectural problem makes it impossible to eradicate noncollapsible rigid cavities without interfering with skeletal continuity. Occasionally under such circumstances, amputation of an extremity is indicated as being preferable to multiple ineffec-

tual operations foredoomed to failure. However amputation should not be resorted to until at least one attempt has been made to terminate drainage by surgery which will give an accurate knowledge of the status of the bone. With prompt, adequate and intelligent treatment of the acute phase of hematogenous osteomyelitis amputation is becoming less frequent than it was formerly.

OSTEOMYELITIS DUE TO DIRECT CONTAMINATION

Osteomyelitis due to direct contamination is a suppurative infection which results from the direct introduction of pathogenic bacteria into a localized area of bone. Its development necessitates a break in the soft tissues covering the bone. Unlike hematogenous osteomyelitis the bacteria are not brought to the bone through the blood stream and lodged in areas of sluggish circulation. True bacteremia may result, but when this does occur the bacteria gain access to the circulation and are fed to it from the localized osteomyelitis or concomitant infection of soft tissue enveloping the area of bone infection. The patient suffering from osteomyelitis due to direct contamination may and often does show the systemic signs of infection.

The commonest cause of osteomyelitis due to direct contamination is a compound fracture, in which the broken bone emerges through the skin or the soft tissue covering the fracture is perforated or crushed. There are other causes such as contamination at surgical operations, infection from metallic appliances used for skeletal traction and internal fixation, such as Kirschner wire, Steinmann pin, bone plates, screws and metallic rods for intramedullary fixation of bones and perforating wounds. A perforating wound seldom results in osteomyelitis unless the periosteum or the cortex of the bone or both are disrupted. Intact bone covered by periosteum, has great resistance to pathogenic bacteria, a normal bone may even be bathed in a large active soft tissue abscess for a long time without being invaded by bacteria. Bone surrounded by an abscess may become demineralized and lose radiographic density as a result of the inactivity of the patient and local increase in circulation. This radiographic change should not be misinterpreted as due to osteomyelitis. The exception to this occurs in the distal phalanges of the fingers. In the pulp of the finger, there exists an anatomic arrangement found nowhere else in the body. A small fragment of . . . is encased in

an unyielding closed space. The blood supply to the area is vulnerable to pressure so that swelling and edema will produce ischemia and necrosis of the bony phalanx. Subsequent to necrosis the phalanx is easily invaded by bacteria. This is the sequence of events in a felon of the distal closed space of the finger when drainage is delayed.

The pathologic anatomy of osteomyelitis due to direct contamination is different from that of hematogenous osteomyelitis. The pathology of the former varies with both the causative factor of the infection and with the bone in which it is located. The principal differences are, first, that the bacteria are not being fed to the infected area from the circulation, second, there is not an expanding area of infection within a rigid structure which must burrow its way to drainage by destruction of bone and elevation and rupture of the periosteum, since the exudate, because of the pathogenesis of the disease, has easier access to exterior drainage through the channel by which the infection was introduced. Third, there is not the same tendency to extensive thrombosis of blood vessels and devitalization of bone.

The pathologic anatomy varies in degree and extent with the factors and condition leading up to the implantation of the bacteria. In osteomyelitis due to direct contamination which results from a compound fracture, the most common cause, conditions may vary widely from a small perforation of the soft tissues to extensive laceration and bruising of the overlying muscles and skin. The bone may be broken into only two major fragments with little stripping of periosteum or it may be extensively comminuted into many fragments. The periosteum may be widely stripped and lacerated. In the latter circumstance, some bone may be deprived entirely of its vascular supply. More bone, although not totally deprived of blood, may have its vascular supply compromised. Bacteria introduced into such a locus may be brought to a state of submission if bodily resistance and therapeutic help (antibiotics) are promptly brought to the infected site. Or the bacteria may multiply and suppuration result, dependent to a greater or lesser degree on the relation of bodily defenses and virulence. Fortunately exudate travels along lines of least resistance. It can find its way out through the same channels by which the bacteria were introduced if wounds have not been closed too tightly. Even though drainage is promptly established and the exudate permitted to escape, devitalized bone with a

deficient blood supply cannot entirely rid itself of bacteria and in itself acts as a foreign body to perpetuate exudation. In the presence of devitalized bone even after the virulence of invading bacteria has been neutralized drainage tends to continue as granulation tissue carries on its efforts to dissolve dead bone. Consequently chronic draining sinuses result. New bone may form around the dead bone and union of the fracture may result or reparative osteogenesis may be interrupted and only fibrous union or even non union of the fracture may occur.

When osteomyelitis results from the entrance of bacteria with wires or pins used for transfixing the skeleton, the sequence of events is the same, but because there is little or no devitalized bone, the manifestations are milder. Pins for traction are placed through an area of cancellous or trabecular bone such as the lower end of the femur, upper tibia or os calcis (Fig. 144). The organisms multiply in the cancellous bone and produce necrosis of the trabecular bone to varying degrees, with or without the formation of a sequestrum. Fortunately spontaneous drainage results easily and early along the pin tract and is facilitated by removal of the pin. No great intraosseous pressure is established and thrombosis of vessels is not extensive. Little bone is destroyed. The end result is often a small noncollapsible cavity with rigid walls which houses granulation tissue and a small sequestrum. The small opening through the rigid cortex provides inadequate drainage for the abscess and a chronic sinus results. The abscess is analogous mechanically to a compression chamber. At other times a small ring-shaped piece of bony cortex will sequestrate about the pin where it perforates and grips the cortex. The ring sequestrum acts as a foreign body and drainage continues until it is removed.

When infection complicates the insertion of a large metallic rod into the medullary canal of a long bone, the indication for the intramedullary rod is practically always to immobilize a fracture. The area immediately adjacent to the fracture site is the most traumatized and, because of this, the most deficient in circulation. At this site, the least resistance is offered to the multiplication of bacteria. Fortunately this is the site at which exudate can most easily escape into the soft tissues. The inflammatory process generally localizes itself at the site of fracture. However the abscess or granuloma often extends a considerable distance up and down from the fracture site in the medullary canal and

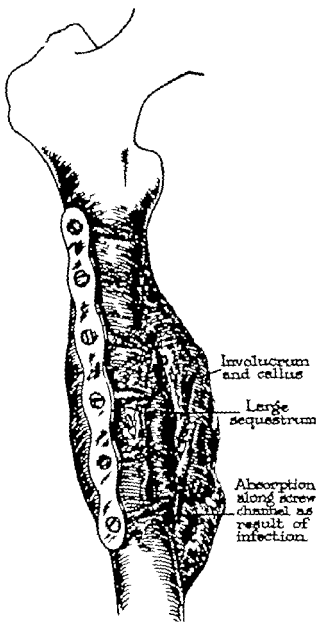


Figure 144 Drawing of roentgenogram of osteomyelitis of femur with large sequestrum, involucrum and infected screw channels resulting from direct contamination. This followed open reduction and internal fixation of a closed fracture.

infection may spread through the entire medullary canal. After the systemic aspect of the infection has subsided, the presence of the metallic foreign body tends to call forth an inflammatory reaction in the medullary canal with the formation of a granuloma and localized abscess. Sequestration is ordinarily confined to the area in proximity to the fracture. Drainage from sinuses continues until the metallic foreign body is removed. The same is true of metallic screws and plates. A granuloma will form about them and inflammation will not subside as long as the metallic objects remain in the tissues.

Treatment of osteomyelitis due to direct contamination involves two phases: prophylactic measures and treatment of established disease. The greatest opportunity to prevent infection of bone presents itself in the management of compound fractures. Such a fracture is an emergency of the highest degree and definitive treatment should be rendered as soon as a patient's general condition will permit the use of anesthesia. Débridement must be painstaking and thorough and internal fixation should be avoided if possible. Great judgment must be used in making the decision to convert a compound fracture to a simple fracture by primary closure at the time of initial débridement. Closure should not be done if there is any lack of good soft tissues which would be put under tension by suture. If the locale and extent of injury point to a high probability of infection, the fracture should be drained through the open wound and a delayed secondary closure should be substituted for primary closure. Broad-spectrum antibiotics should be administered promptly in adequate dosage.

In elective clean surgery the skin in the area of incision and in the line of lymphatic drainage must be absolutely free of any semblance of infection. Tissues must be handled with gentleness during operation to prevent unnecessary trauma. Good hemostasis should be effected and the soft tissues must never be approximated under any tension.

To avoid bacterial contamination of bone in the use of pins and appliances for skeletal transfixion, the same requirements for skin perfection are necessary as in clean elective surgery. Pins for skeletal fixation and traction which extend out of the skin should be so applied and the dressing so secured that in carrying out their function they are not permitted to slide back and forth through the bone. Dressings should be applied in the operating room, should be sealed and should not be changed until the pins are removed. Constant change of dressings results in contamination. The use of antibiotics should not be necessary for skeletal transfixion in closed fractures.

Most instances of established osteomyelitis due to direct contamination result from compound fractures. If a compound fracture has been closed primarily both the patient and the wound must be observed assiduously for any evidence of developing infection. At the first sign of infection, stitches should be removed to permit drainage of exudate. This simple procedure will often prevent the bacteria from gaining a foothold. The drainage

most be promptly cultured and the sensitivity of organisms to drugs determined so that the most effective antibiotics can be used. Once there is definite evidence of bone infection, adequate drainage must be maintained so that there is no retention of exudate. If the condition becomes chronic and a draining sinus continues, certain principles should be followed.

Adequate time should elapse between the infection and any definitive surgery so that the extent of bone involvement can be determined and, when a fracture is present to permit the formation of sufficient involucrum or callus to give some stability to the fracture site. As a rule, this requires ninety to 120 days and can be demonstrated in good x-ray pictures. Clinical examination will determine the solidity of the fracture.

Appropriate systemic therapy should be instituted so that humoral conditions are conducive to wound healing. Transfusions and high protein intake may be indicated prior to definitive surgery so that the wound healing will be at a high potential.

Appropriate antibiotic therapy should be instituted twenty-four hours prior to definitive surgery and continued as long postoperatively as is indicated by the condition of the patient and wound.

At the time of surgery all sequestered or potentially sequestering bone should be removed, leaving no shelving or roofing edges to trap infection.

Any and all foreign metal which may be in the draining area should be removed.

If the cavity thus created can be obliterated with flexible well-vascularized connective tissue and closure effected, this should be done.

If flexible vascular soft tissue is not available but only rigid ischemic scar tissue is available, the bone cavity should be left open, packed with petrolatum gauze and permitted to granulate from the depths to the surface.

Physiologic rest of the wound should be carried out for an adequate time postoperatively by immobilizing the joint above and below the wound in a cast or splint.

In many instances, the drainage from osteomyelitis by direct contamination will terminate spontaneously in a reasonable time if adequate drainage is maintained and the invading organism is sensitive to an antibiotic or chemotherapeutic agent. This, of course, does not hold when there are conditions of sequestration, noncollapsible cavities with rigid walls or deficiency in soft tissue coverage. Even when the above deleterious situa-

tions are present the drainage resulting from osteomyelitis due to direct contamination can be terminated if sound surgical principles are followed. It may at times be necessary to have prepared a good pedicle skin graft for use in the final closure to accomplish a satisfactory end result.

When osteomyelitis is associated with a compound fracture the fracture may fail to unite. Infection is the commonest cause of nonunion and bone defects. In patients with large bone defects and nonunion bone grafting may be required to produce union. Bone grafting procedures in a previously infected operative field are many times complicated by recurrence of the infection and failure of the bone graft. This is especially true if there has been extensive scarring so that bone grafts cannot be surrounded by well vascularized muscle and covered with soft flexible skin. Because of the failure of union of the bone with a large hiatus after osteomyelitis due to direct contamination, some extremities will require amputation despite the fact that the drainage from the initial osteomyelitis can be terminated.

PYOGENIC INFECTION OF JOINTS

The term "pyogenic infection of a joint" means the invasion of a joint by pathogenic bacteria which induce a suppurative reaction in the tissues of a joint. Suppuration in a joint is also termed "pyarthrosis" and is sometimes referred to as empyema of a joint. Most joint diseases are the result of nonspecific nonbacterial insults rather than bacterial invasion. However the effect of suppuration in a joint is so rapidly destructive to the tissues comprising the mechanism of the joint that it is imperative to recognize promptly and treat adequately a pyogenic joint if function is to be preserved. Treatment of most of the nonbacterial joint affections is not specific and the destruction of tissues is less precipitous than it is in a suppurative joint. Consequently the necessity for prompt and efficacious treatment is not so demanding.

Acute pyogenic infection of a joint is uncommon. Such infections represent less than 2 per cent of all joint disease. The condition is more frequent in infants and children than in adults. It is also more frequent in males than in females. The hip and knee are the joints most frequently involved. Bacterial invasion of joints may occur by three routes: hematogenous infection, direct extension and direct contamination.

The infection may be hematogenous, in which case the bacteria are carried to the

joint and implanted in the joint tissues by the blood stream from a site of infection elsewhere in the body. This mode of infection necessitates bacteremia.

In direct extension of infection a suppurative focus immediately adjacent to the joint ruptures into the joint cavity implanting pathogenic bacteria which activate an inflammatory reaction in the joint tissues. This cause of joint infection is most often seen as a complication of osteomyelitis. In certain locations, particularly in the upper femur the initial focus of osteomyelitis may be within the capsule of the joint. Consequently when the bone abscess escapes the confines of the cortex and periosteum, it empties bacteria directly into the joint.

A joint may become infected by direct contamination. This may result from an accidental wound with direct implantation of pathogenic organisms or foreign material into a joint cavity. An infection may also be caused by diagnostic or therapeutic paracentesis of a joint. With the increasing use of intra-articular injection of therapeutic agents in the treatment of nonspecific joint disease, this may more often be a cause of direct contamination of a joint. Strict asepsis must be observed in joint aspirations or injections to avoid contamination.

The bacteria most frequently found in a suppurative infection of a joint are the staphylococcus and streptococcus. Some type of these two organisms is the cause of 95 per cent of pyogenic joint infections. Gonococcus, pneumococcus, meningococcus, *Escherichia coli* or *Shigella dysenteriae* is found occasionally to be the etiologic organism in an infected joint.

A joint is made up of two or more bones, bound together at their cartilage-covered ends by a sheath of dense connective tissue. This sheath of dense connective tissue is attached to the bones and constructed in such a way that it produces stability of the opposed cartilage-covered ends of the bones and yet permits mobility. The inner surface of the intra-articular portion of the connective tissue capsule is lined by a specialized tissue termed "synovial membrane" creating a closed cavity (Fig. 145). The synovial membrane is connective tissue which is modified in structure and function. Through the synovial membrane, a fluid dialyzes and enters the joint to lubricate the articular cartilage on the ends of the bones. The specialized synovial membrane produces mucin which is added to the dialyzed synovial fluid. This increases the viscosity of the synovial

Diagram of Structure of Joint

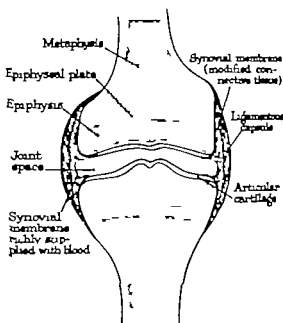


Figure 145 Diagram of joint, illustrating relationship of articular cartilage, which is avascular and highly vascular synovial membrane.

fluid within the joint. The synovial fluid, except for the addition of mucin, is a dialysate of plasma. The mucin is a product of the specialized function of the synovial membrane. The connective tissue capsule and its inner surface the synovial membrane, are richly supplied with capillaries and blood and have extensive powers of regeneration. The articular cartilage however is a tissue which is scanty in cells and has a very poor blood supply. As a consequence articular cartilage has little or no power of regeneration. Articular cartilage which is damaged or destroyed by any insult, is not regenerated, but is replaced by scar tissue. Since synovial fluid is a dialysate of plasma substances in the blood may enter a joint by crossing the synovial membrane in the synovial fluid. Bacteria, antibodies, chemicals and drugs all do this but are found in synovial fluid in a slightly lower concentration than in the plasma.

The synovial membrane is especially susceptible to the lodgment of organisms when bacteremia is present. When pathogenic bacteria are deposited in a synovial membrane, the outcome as in any infection, is dependent on the balance between the virulence and number of organisms and the local resistance of the tissue as well as the general reaction of the host. Vague joint pains of mild degree are common with many infections. These arthralgic pains are not

↓ by ob-

erable clinical changes in the painful joints but it is probable that they represent the lodgment of a small number of bacteria in the synovial membrane which are quickly rendered innocuous by host resistance. When the pathogenic organisms gain ascendancy, hyperemia and swelling of the synovial membrane are rapidly produced. Polymorphonuclear leukocytes congest the area of inflammation. The synovial lining thickens and becomes covered with exudate. There is increased production of synovial fluid which accumulates in the space which encloses the articular cartilage on the bone ends. In the normal joint, only enough synovial fluid is present to produce a film of viscose fluid for the smooth sliding of bone ends. Normal synovial fluid has few nucleated cells and the percentage of leukocytes is extremely low, being from 5 to 8 per cent of the total cells. The mucin content of normal synovial fluid is high. With the abnormal conditions which result from inflammation of the synovial membrane there is a marked outpouring of leukocytes and of bacteria into the synovial fluid. The increased intra-articular pressure and swelling of the synovial membrane decrease the normal resorption of synovial fluid. As a result of these changes considerable pressure may be built up in the joint cavity by the exudate. In advanced inflammation, the entire surface of the synovial membrane may be covered by a thick fibropurulent exudate and clots of fibrin may form in the accumulated fluid, which, because of its high concentration of leukocytes, may appear frankly purulent. The cell count of normal synovial fluid is below 100 cells per cc., of which from about 5 to 8 per cent are leukocytes, but in a pyogenic joint the abnormal synovial fluid may contain over 100,000 cells per cc. and 90 per cent of these cells may be polymorphonuclear leukocytes.

The articular cartilage which is a tissue with few cells and with a sparse blood supply undergoes pathologic changes as a result of the increase in abnormal joint fluid. As leukocytes in the pathologic fluid disintegrate, they liberate a proteolytic enzyme which digests the articular cartilage. The sites of greatest digestion of articular cartilage occur at areas of contact. If the inflammatory process subsides quickly the destruction of articular cartilage may be superficial and recovery may occur with negligible impairment of joint function. However if an exudate high in leukocyte content continues to occupy the joint space, the articular cartilage may be digested through to the underly-

ing bone and undermined so that pieces of varying size flake off.

As the virulence of the invading organism is overcome by the resistance of the host the accumulation of the high leukocytic synovial fluid decreases and the fluid is absorbed. Granulation tissue, which is formed in the areas of damaged and destroyed synovial membrane and articular cartilage is gradually transformed to fibrous tissue. The granulation tissue formed in a joint after damage to its normal tissues is called pannus. The degree of recovery of a joint depends on how severe the initial infection has been and how long the products of exudation have been permitted to act in the joint cavity. When only a small portion of the synovial membrane has been necrotized the joint may return almost to normal. If there has been extensive lysis of an articular cartilage, fibrous bands may grow between contacting surfaces form adhesions and markedly limit function of the joint. If articular cartilage is digested through to underlying bone, an osseous bridge may unite the bone ends making up the joint and produce bony ankylosis.

The symptoms associated with a pyogenic joint infection are the general symptoms of infection and the local symptoms in the joint involved. When a pyogenic joint is the prime manifestation of the bacteremia, the onset will vary with the virulence of the invading bacteria. Malaise is a general rule. The onset may be initiated with a mild febrile rise of from 1 to 2° C or it may be marked by severe hyperpyrexia and a chill, the temperature rising as high as 40 or 41° C. There may be concomitant dehydration and acidosis accompanied by vomiting. This is more common in the child than in the adult and persists until fluid and electrolyte requirements are satisfied. The infected joint itself causes pain which, in the first few hours or the first day may be mild and aggravated only by motion but soon becomes so severe that the patient does everything possible to maintain the joint immobile and resents the slightest jar of the bed. When a joint becomes infected during the course of a systemic disease associated with bacteremia, such as pneumonia, meningitis, meningococcemia or bacillary dysentery the general symptoms of the original illness may predominate. The only additional symptom will be pain in the involved joint. However if the joint becomes involved after improvement in the initial disease has occurred, there may be a sharp rise of a falling temperature and a recurrence of general malaise.

The earliest signs of a joint infection are, first, tenderness to palpation in the joint line and, second, restriction of the normal range of motion. Tenderness to pressure in the joint lines where they are palpable will often be present very early even before there is any evidence of an effusion or swelling about the joint. Restriction in the normal range of motion, particularly a loss of the last few degrees of extension of a joint, is also an early sign and precedes the appearance of swelling or joint effusion. As an infection progresses, restriction of motion increases rapidly and is accompanied by involuntary muscle spasm which is a protective mechanism to splint the joint in the most comfortable position. With an infection of the ankle joint, the foot will be held by muscle spasm in a position of plantar flexion the knee when infected will be held in a position of flexion. Muscle spasm about the hip in acute infections leads to a position of flexion, abduction and external rotation. In the upper extremity a finger will be held flexed, the wrist will be held in palmar flexion the elbow will be flexed, and with an acutely infected shoulder the arm will be held adducted against the thorax by muscle spasm. Any attempt to alter these positions of protective muscle spasm abruptly evokes increased muscle spasm and produces severe pain. Effusion and increase in synovial fluid, with distention and swelling of the joint, occur relatively early in pyogenic infections of joints. The effusion may be small in quantity if resistance to the invading organism is good. In superficial joints which are not covered by bulky muscles the effusion can be seen and palpated. However in the hip, shoulder and joints of the spine, which are heavily covered by large muscles, this is not possible. Local heat also accompanies the effusion into the joint, but may not be impressive when the patient has a general febrile reaction. Redness of the skin is not a common accompaniment of pyogenic joint infections.

Variations of intensity of the symptoms and signs of an acute suppurative infection of a joint may occur in an infant prior to the age of one year. Generally however the symptoms and signs are less pronounced, as the infants incompletely developed nervous system modifies pain and decreases muscle spasm.

With recent advances in the therapy of rheumatoid arthritis, there has developed another group of patients in whom the signs and symptoms of a pyogenic joint may be masked. Patients with multiple joints involved by rheumatoid disease who are receiving sys-

temically the antiphlogistic hormone, ACTH or cortisone or any of the modifications, over a long period, are in a nonreactive condition as far as the manifestations of an acutely infected joint are concerned. Because of the already well-established rheumatoid disease, any aggravations of symptoms in a joint or any increased swelling about a joint is likely to be dismissed casually by the patient and the doctor. Long-sustained hormone therapy abolishes or delays the systemic reaction to infection insofar as pain is concerned. In these patients the febrile response is diminished and leukocyte response to infection is altered. In addition, the ability to localize infection is abrogated and the additional stress of a superimposed infection is not tolerated. For these reasons any unnecessary insertion of a needle into a joint of a patient who is receiving cortisone systemically should be avoided. The ever present minimal contamination, which occurs in any surgical procedure under the most aseptic technique, is readily rendered innocuous by a patient not receiving antiphlogistic hormones, but with the loss of defense against infection which results from prolonged cortisone therapy empyema of a joint may result from the slightest contamination. Infections in closed cavities attain great virulence in patients in this state before systemic symptoms are manifested and a very large amount of exudate may accumulate in a joint under these circumstances before its significance is appreciated.

In the initial evaluation of the patient suspected of suffering from a pyogenic infection of a joint, detailed questioning should be carried out to determine whether there has been any recent illness such as an upper respiratory infection tonsillitis, urethritis or gastrointestinal disturbance. The possibility of a recent exanthematous disease should be inquired about if the patient is a child. All cutaneous surfaces and mucous membranes should be observed for a possible focus of bacteria. The throat and tonsils should be carefully inspected and in children the ears should be examined to determine the state of the middle ear. The condition of the lungs should be ascertained. If physical examination leaves any doubt, roentgenograms of the chest should be obtained. Pneumococcus is an organism which occasionally causes a pyogenic joint. Examination of the heart may give information suggesting rheumatic fever. Palpation of the abdomen is important, especially when the hip is the joint in question. Any irritation along the scale in its

retroperitoneal portion may cause the hip joint to be splinted in flexion. Examination of the abdomen may bring to light a tender appendix or enlarged retroperitoneal lymph glands. Enlargement of the spleen or liver may direct attention to a pathologic condition of importance. The presence or absence of abdominal distention is indicative of the extent of toxemia or disturbance in fluid balance or electrolyte metabolism. Urethral or vaginal discharge may point to a recent gonorrheal infection.

In the examination of the joint or joints involved, the condition of the skin and soft tissues overlying, surrounding and adjacent to the joint should be noted. Swelling, edema and redness of the skin with an obvious contiguous purulent area, may indicate that the infection is superficial rather than intra-articular. In the superficial joints which are covered only by skin and subcutaneous tissue, distention of the joint itself may be obvious. Distention is not easy to be certain of in the shoulder and is impossible to detect in the hip and joints of the spine. The position in which the patient maintains the joint and the protection which he extends to it will give information about involuntary muscle spasm. Palpation of the joint lines may reveal tenderness before an effusion is present. The musculature above and below the suspected joint should be palpated for areas of tenderness, fluctuation or thickening. A juxta-articular inflammation such as osteomyelitis may give joint signs when the joint itself is not involved. The presence or absence and the degree of effusion within the joint can be verified by palpation. Examination of the range of motion present in the joint should be carried out and compared with that of a similar uninvolved joint. Motion must be tested gently since rough or forceful handling of an infected joint will cause severe pain. In the examination of the hip and shoulder joints which are deeply buried under muscle, the range of motion is the most important fact to be determined. The presence or absence of muscle spasm and its degree can also be verified at this time. Measurements of the circumference of the joint and of the musculature above and below should be made at the initial examination and recorded, as they may be of value for comparison later in the course of the disease.

In the initial appraisal of the patient's condition, the hemoglobin value, red blood cell count and differential white blood cell count should be obtained. These examinations will shed light on the general condition of the pa-

tient and his reaction to infection. They may also bring to light an unsuspected hematologic condition of importance or present an indication for blood transfusion. Additional blood counts and hemoglobin determinations should be carried out through the course of the disease as needed, to obtain information concerning the condition of the patient and the efficacy of treatment. Initial laboratory studies should include urinalysis which may give evidence of a primary infection in the urinary tract or indicate severe parenchymatous damage and nephritis complicating the infection. The presence or absence of diabetes or acidosis due to dehydration may also be determined from urinalysis. Urinalysis should be repeated at least twice weekly during the febrile course and during the treatment. The initial laboratory investigation should include a blood culture. The blood culture should be made before antibiotic therapy is instituted and the blood sample is best taken when the fever is elevated. Organisms obtained by blood culture should be typed against antibiotics for sensitivity. Since bacteremia is often transitory the blood cultures may be negative. Special circumstances may indicate additional bacteriologic studies. An obvious purulent exudate from a focus of infection in the skin or mucous membranes or urine heavily loaded with pus cells should be cultured for organisms; any organisms found should be typed for sensitivity to antibiotics. A recent history of diarrhea or enteritis may be an indication for a stool culture. Serologic examination of the blood should be included in the initial laboratory survey.

Roentgenographic examination of the involved joint or joints should be made. This should include the bones and the joints above and below the suspected joint. X-ray examination is of value to exclude the possibility of a focus of osteomyelitis or an incomplete fracture. When the joint infection results from external contamination, a roentgenogram may disclose a foreign body in the joint or adjacent to it. Early in the course of pyogenic infection of a joint, in the absence of an osteomyelitic focus, the only roentgen ray findings are soft tissue swelling and evidence of effusion but, as the disease progresses, demineralization of the articular ends of bones will become evident. In those joints which progress unfavorably destruction of articular surfaces may be evident in a roentgenogram made late in the course of the disease. Small cartilaginous sequestra, which may cause continuous drainage will not show in roentgenograms, as cartilage is radiolucent. X-ray

examinations are of more aid later in the course of a pyogenic joint when they may then help to assess the degree of damage to the joint. They should be obtained as needed for the management of the patient.

Aspiration and study of the synovial fluid make up the most valuable procedure in the examination of a joint suspected of being infected with bacteria. Aspiration is a simple procedure which can be done with the patient under sedation and local infiltration an anesthesia. It must be carried out with strict aseptic precautions with adequate preparation of the skin and with sufficient sterile draping. Superficial joints such as the knee and elbow are very easy to aspirate. The shoulder and hip are more difficult, but still quite accessible to aspiration, and this is particularly true when these deep joints are abnormally distended with synovial fluid.

From the normal joint it is usually impossible to aspirate any fluid. At the most, not more than from 1 to 2 cc. may be obtained. Normal synovial fluid is clear pale yellow viscous and does not clot. It contains only a few nucleated cells. Generally there are less than 50 and rarely more than 150 nucleated cells per cu. mm. of normal synovial fluid. The polymorphonuclear leukocytes in normal synovial fluid comprise from 7 to 8 per cent of the nucleated cells present. Lymphocytes in the normal fluid comprise 25 per cent. The remaining nucleated cells are monocytes, clasmotocytes and unclassified phagocytes. The normal fluid is also sterile.

Synovial fluid is present in increased amount in a joint invaded by pathogenic bacteria and by aspiration it is usually easy to obtain it through a needle of adequate size. The fluid aspirated from a pyogenic joint is changed in appearance. It is turbid rather than clear. The degree of turbidity may vary from slight opacity to the consistency of frank pus, depending on the severity and duration of the joint infection. The fluid is decreased in viscosity and forms clots soon after withdrawal from the joint cavity. The total cell count of the synovial fluid from a pyogenic joint is increased astoundingly. Of ten from 60,000 to 120,000 cells per cu. mm. are present and if the fluid is frank pus, the cells may be too numerous to count. The polymorphonuclear leukocytes may be increased to as high as from 80 to 90 per cent of the total nucleated cells. Gram stain of a smear of the synovial fluid may indicate the presence of bacteria. Culture of the synovial fluid will often produce the organism which is the cause of the infection. The incidence

of positive cultures from synovial fluid is lower when gonococcus is the infecting organism than when a streptococcus or staphylococcus is responsible. Positive cultures are still less frequent in meningococcus infections. Thus with the valuable information to be obtained by joint aspiration, it is obvious that in any suspected pyogenic infection of a joint, aspiration and study of the synovial fluid are mandatory at the initial examination.

Because of the multiplicity of joint affections and of general systemic conditions causing acute reaction in joints, delay in the accurate diagnosis of acute pyogenic arthritis is likely to result unless its possibility is kept in mind. Any acute pain, accompanied by signs of infection, occurring in a joint or developing during or proximate to an acute infectious disease should call to mind the possibility of acute suppuration in the joint.

Acute pyogenic arthritis may be extremely difficult to differentiate from acute hematogenous osteomyelitis. When the osteomyelitic focus is in the neck of the femur the differentiation is impossible, as all the local manifestations are joint manifestations because of the fact that the area of bone inflammation is actually within the capsule of the hip joint. However in other sites and about the knee, which is the most frequent localization of osteomyelitis the area of bone tenderness is juxtaepiphyseal and not in the joint line. In the early phase of adjacent osteomyelitis, the knee joint does not present an effusion. These findings should permit differentiation. When there is an extensive focus of osteomyelitis in the upper tibia or lower femur the knee joint, even though not infected, may present an effusion, the result of contiguous inflammation. When there is an obvious increase of fluid in the joint, the issue can be quickly settled by aspiration and study of the synovial fluid. Pyogenic infection of the hip joint itself is common with osteomyelitis localized to the neck of the femur as a result of direct rupture of the bone abscess into the joint cavity. Other joints, because of different anatomic relationships, become contaminated from a juxtaepiphyseal bone abscess only after the osteomyelitis has existed for a long time or been inadequately treated. Extensive burrowing of exudate and necrosis of soft tissue barriers must occur before the adjacent joint becomes contaminated.

Cellulitis of the soft tissues adjacent to or surrounding a joint may lead to pain on motion and protection of the joint by the patient. However the redness, swelling and in-

duration of the skin should give a clue to the diagnosis. In the acute suppurative joint the skin itself about the joint is seldom altered in appearance.

Rheumatic fever may be difficult to differentiate from an acute joint infection. The joint manifestations of this disease although frequently presenting an effusion into the joint, as a rule show a greater periarticular reaction than is found with a pyogenic joint. Frequently multiple joints are involved and the manifestations are likely to be transitory and migratory rather than steadily progressive in the same joint. Other indications of rheumatic fever may be evident from the general examination.

Rheumatoid arthritis is seldom abrupt in its onset. There is usually a history of protracted mild discomfort in one or more joints prior to the acute swelling of a single one. The superimposition of a pyogenic infection in an already established rheumatoid joint may occur spontaneously or in the course of some infectious disease such as pneumonia or scarlet fever. In the rare instances when this happens, the diagnosis of acute sepsis is difficult to make but can be arrived at definitely by aspiration and study of the joint fluid. The undue, abrupt swelling of a rheumatoid joint, particularly with an elevation of temperature, calls for examination of the joint fluid. This is particularly indicated if the patient is receiving systemic hormone therapy.

Gout in its initial attack may suggest a purulent joint infection. The abruptness of onset and the severity of pain may appear to indicate a severe infection. This metabolic disorder usually involves a joint in the foot and has a predilection for the metatarsophalangeal joint of the great toe. It may, however, involve one of the larger joints or even one of the upper extremity. In gout, the swelling is predominantly periarticular joint effusion, if present at all, is usually small in amount. There is often redness of the tissues but the systemic signs of infection are absent. A febrile rise of more than 1 C. is uncommon. Determination of the serum uric acid level, which is always elevated in an acute attack of gout, will differentiate it from purulent arthritis.

Hemophilia with acute hemorrhage into a joint may be accompanied by great distress and elevation of temperature the result of blood in the joint, and may simulate an acute pyogenic infection. A history of previous epistaxes of bleeding is often obtainable and ecchymotic sites elsewhere in the soft tissues

may suggest abnormal bleeding. Determination of the bleeding and clotting time will establish the diagnosis. Aspiration of synovial fluid will reveal frank blood rather than cloudy fluid. It is imperative that incision and drainage of a hemophilic joint be avoided as an unnecessary incision may jeopardize the patient's life.

Scurvy can produce a joint effusion from intra-articular hemorrhage. When this occurs there will be other clinical evidence and roentgen ray findings to substantiate the diagnosis.

Bursae which become infected with pyogenic organisms should be easily differentiated from intra-articular infections. Those most commonly involved are the olecranon, prepatellar and pretibial bursae. Careful inspection and palpation will reveal the distended sac to be superficial to the joint, even though it is in proximity.

Tuberculous infections of joints are not sudden in onset. Pain is insignificant in the early stage of joint tuberculosis and the systemic manifestations of acute infection are absent. Aspiration and examination of synovial fluid will differentiate this type of infection from the acute purulent infection.

Erythema nodosum may be accompanied by joint effusion but history and inspection should bring to light the skin lesion and thus differentiate this condition from acute infection.

Traumatic effusion into a joint follows close on the injury therefore a definite history of some proximate trauma can be elicited. Systemic signs of infection are either mild or absent with traumatic joint effusions. Aspiration usually yields a blood-tinged fluid.

The hip joint deserves special consideration in the problem of differential diagnosis. It is motivated by the psoas muscle which extends in the retroperitoneal space from the second lumbar vertebra to the lesser trochanter of the femur (Fig. 146) and this muscle is a strong flexor of the hip joint. Any pathologic condition which irritates the psoas muscle may cause involuntary spasm and produce a flexion contracture of the hip joint. The commonest condition to cause this is retroperitoneal lymphadenitis. Retroperitoneal lymphadenitis is usually accompanied by systemic signs of infection. The enlarged glands may resolve spontaneously but they can suppurate and require surgical drainage. An inflamed retrocecal appendix or pelvic inflammation may lead to a flexion contracture of the hip joint by irritation of the psoas muscle.

Physical examination of the range of joint

Relation of Psoas Muscle to Hip Joint

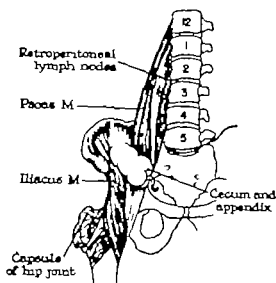


Figure 146 Diagram of psoas muscle and its relationship to the hip joint to illustrate how extra articular disease may produce abnormal signs in the hip joint.

motions will identify these conditions as being extra articular and not in the hip joint itself. No motion other than an attempt at extension of the joint against the flexion contracture, which puts stretch on the irritated psoas muscle, will cause pain. There is no loss of flexion or rotation of the hip joint. The patient will walk on the involved extremity with little discomfort but with a marked limp. In a thin child, the enlarged lymph glands may sometimes be palpated through the abdominal wall.

The treatment of a pyogenic infection of a joint requires the general treatment of the patient and the local treatment of the infected joint. General treatment of the patient strives to muster all body defenses against infection. Local treatment of the joint strives to minimize damage to the cartilaginous gliding surfaces, so that as nearly normal motion as possible will be preserved and deformities prevented. The patient affected with a pyogenic infection of a joint, because of the sepsis preceding or associated with its onset, may be severely dehydrated and in negative nitrogen balance. These conditions must be rectified as rapidly as possible and maintained as nearly normal as possible. Intravenous fluids or transfusions of blood may be indicated if there is severe depression of hemoglobin and red cells. Part of the caloric and vitamin requirement may be met by administration of appropriate substances with intravenous fluid.

A high blood concentration of multiple antibiotics with the broadest possible antibacterial spectrum, should be effected promptly after the diagnosis is made. In infants and children or in adults who will not take adequate oral dosage, these antibiotics may be added to intravenous fluid and administered as a continuous drip. This is the method of choice when the patient is profoundly toxic. Since the most probable organisms to infect a joint are the staphylococcus, streptococcus and gonococcus, the antibiotics administered should be effective against them. The broad coverage desired against the staphylococcus, streptococcus and gonococcus can be effected by a combination of erythromycin, oleandomycin or tetracycline with chloramphenicol. When there is a history of previous therapy with chloramphenicol, this drug should be used with the greatest reluctance and not unless the invading organism cannot be checked by any other antibiotic.

When there is clinical evidence or suspicion of an organism which may be insensitive to these drugs, other antibiotics or chemotherapeutic agents which seem appropriate should be added and administered in appropriate dosage. As soon as the invading organism has been identified, either from synovial fluid or blood culture and its sensitivity to antibacterial drugs determined, a single specific antibiotic may be substituted for the multiple-drug therapy. Under no condition should antibiotic therapy be delayed to determine the specific antibiotic, as this is a waste of valuable time. When the febrile reaction is severe, antipyretic drugs and alcohol sponges may be indicated. These are especially indicated in infants and children, as convulsions may result from inordinately high fever. Any obvious focus of infection, such as a crusted abrasion, a furuncle or infected middle ear which might feed bacteria into the blood stream should have adequate drainage.

Local treatment of the infected joint strives to rid the joint of all accumulation of pathologic synovial fluid with a high leukocyte count. The disintegration of polymorphonuclear leukocytes liberates a proteolytic enzyme which digests articular cartilage. The method chosen to prevent destruction of articular cartilage by leukocytic digestion will be influenced by two factors: the character of the synovial fluid obtained by aspiration and the individual joint infected. Certain joints, such as the knee, elbow and ankle joint, because of their superficial location and noncompartmented cavities, readily evac-

tated by aspiration. On the other hand the hip and shoulder because of their deep location, and the wrist because of its compartmentation, are difficult to aspirate. Thin synovial fluid with a cell count of 100 000 cells per cc. will come through a needle easily. Frank pus with clots and cells too numerous to count is difficult to aspirate and because of its high content of leukocytes is rapidly destructive to articular cartilage. Any joint which on initial aspiration yields frank thick pus should be drained by arthrotomy and drainage should be maintained until the patient's systemic reaction has subsided and the synovial fluid has returned to a serous state. When this eventuates, the incision may be permitted to close. The hip joint and shoulder joint, which are buried under bulky muscles, are difficult to repeatedly aspirate thoroughly even though the synovial fluid in these joints is not frank thick pus but is definitely purulent, they are best evacuated by surgical arthrotomy. The arthrotomy of each joint should be planned to give dependent drainage and not to damage supporting ligamentous structures. In superficial joints such as the knee, elbow and ankle which are easy to aspirate, if the initial fluid is not frankly purulent and the systemic response to therapy is prompt and effective, repeated aspiration of purulent fluid may be adequate to prevent destruction in the joint. If this treatment steadily decreases the effusion and the leukocyte count of the fluid drops, repeated aspiration is adequate. Transarticular lavage

of saline through trocars has been advocated for thick purulent synovial fluids. This method requires anesthesia and is not to be relied upon. When the synovial effusion is so thick that it will not come through a large needle open arthrotomy is the better course. An aqueous solution of antibiotics may be instilled directly into the joint cavity at the time of aspiration but if the blood concentration of antibiotics is kept at a high level, all antibiotics and chemotherapeutic agents cross the synovial membrane and are present in synovial exudate in concentrations only slightly below the blood concentration. In densely purulent exudate the effectiveness of even high concentrations of antibiotics is inhibited. Consequently a joint filled with frank pus should have the benefit of open drainage.

Local measures to relieve the pain of muscle spasm are indicated. Continuous skin traction to the extremity in infections of the shoulder hip and knee affords great comfort to the patient and prevents the development of contractures in positions of deformity which may develop very rapidly if muscle spasms go on unchecked. The elbow, wrist, ankle and foot should be splinted in the position of comfort and function. Immobilization and rest of themselves are therapeutic measures of value in treating infection.

When the course of the disease indicates that damage of articular cartilage will probably result with some loss of normal motion every effort should be made to have the re-

Arc of Motion of Best Function in Knee Joint

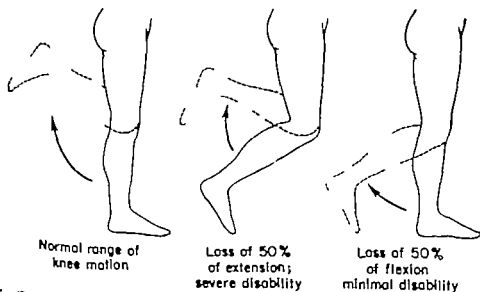


Figure 14—Diagram to illustrate the most useful arc of motion in the knee joint, which should be preserved when a loss of motion is anticipated

Functional Positions for Ankylosis

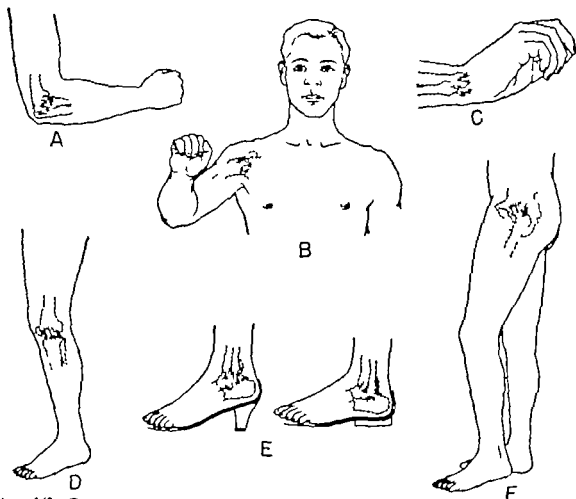


Figure 148 Diagram of functional position for bony ankylosis of joints. A, Elbow—90 degrees of flexion. If ankylosis of radiohumeral joint occurs, the forearm should be neutral in pronation and supination. B, Shoulder—humerus at 70 degrees to vertical border of scapula, 15 to 20 degrees forward of coronal plane, and 30 degrees of external rotation. C, Wrist—20 to 30 degrees of dorsiflexion and 10 degrees of ulnar deviation. D, Knee—10 degrees of flexion. E, Ankle—male, neutral in dorsiflexion and plantar flexion, in 90 degrees with leg. Female, 10 to 15 degrees plantar flexion to accommodate for slightly higher heel on shoe. F, Hip—25 to 35 degrees of flexion, neutral in abduction-adduction, and 5 to 10 degrees external rotation.

maintaining motion in the arc of greatest usefulness (Fig. 147). This can be accomplished by the use of traction and proper splints. For example, if as a result of purulent infection of the knee 50 per cent or about 70 degrees of the normal arc of motion is permanently lost, the extremity will still be very useful when the preserved 70 degrees of motion is possible from a position of complete extension. If the knee recovers with a severe flexion deformity and the remaining 50 per cent of motion is present only through the last half of the arc of normal motion, the individual will be severely handicapped and will require reconstructive surgery. When loss of motion is anticipated in the elbow joint, it is to be remembered that the completely extended elbow is useless. The forearm and hand function best

in the upper half of the normal arc of flexion. The extended stiff elbow cannot reach the head, face, shirt or belt, and is serviceable only to scratch the knee or foot. Each joint has its own angle of greatest usefulness when ankylosed and its arc of motion which is of greatest functional value. Preserved motion must be in the most useful arc and ankylosis must be in the position of greatest function (Fig. 148).

Infections of joints due to trauma, fortunately are rare but do occur occasionally from puncture of a joint by a sharp object or from lacerations extending into a joint. Any joint which has been punctured by a foreign body such as glass, a needle nail or piece of steel from the moment of puncture must be considered potentially infected. It is impera-

the to determine whether or not any portion of the object which punctured the joint has remained within the joint cavity. When there is certainty that a piece of foreign material is within the joint it should be promptly removed by surgical arthrotomy. A metallic foreign body or leaded glass will show in roentgenograms. A foreign body which is radiolucent will not be visible on x ray film and its presence in the joint must be deduced from the patient's history or from that portion of the foreign body which has been recovered. Radiolucent objects which cause great trouble in joints are palm thorns and cactus thorns. Even though the joint is not infected a fragment of such organic material in a joint causes a chemical irritation which will lead to disabling synovitis unless it is removed. The joint which has been punctured by a foreign body without deposit of foreign material in the joint cavity should be treated by rest and immediate administration of multiple broad spectrum antibiotics to provide a high concentration in the blood. This treatment should be continued until it is evident from the systemic response and the local condition of the joint that any contamination produced by the puncture has been rendered innocuous. If fulminating infection with a purulent synovial fluid ensues arthrotomy may be indicated.

A joint which has been opened traumatically to the outside should be treated as a compound fracture. As soon as possible with the patient under general anesthesia careful debridement, with removal of all foreign material and devitalized tissue should be done. This should be accompanied by copious lavage with many thousand cubic centimeters of warm saline solution. When possible, the joint should be closed primarily provided the lapse of time since the injury and the circumstances of the injury are not contraindications. It is often impossible to close synovial membrane or joint capsule but a covering of fascia or even skin will suffice for primary closure. If closure is contraindicated because of contamination, petrolatum gauze is placed in the soft tissues covering the joint and the open wound is allowed to granulate to the surface. Drains should not be inserted between the articular ends of bones.

Treatment of the joint which has become infected from the extension of an adjacent focus of osteomyelitis requires intelligent treatment of the osteomyelitic focus. A joint cannot be expected to recover without severe damage when an osteomyelitic focus continues to drain through it. The area of osteomy-

elitis must be diverted and given adequate extra-articular drainage. When this is done in the initial treatment of osteomyelitis joint destruction is rare except in the hip joint.

Pyogenic infection of a joint which occurs in a patient who is receiving protracted systemic treatment with the antiphlogistic hormones cortisone and ACTH occupies a special category. Such infection occurs in a joint which is already the site of rheumatoid disease and for that reason is difficult to recognize. The individual receiving prolonged cortisone therapy is unable to localize infection. Because of this lowered resistance to pathogenic organisms, drainage by arthrotomy should be promptly instituted. Any attempt at therapeutic relief by aspiration will be ineffective. Prompt open drainage helps to relieve the additional stress of the infection and does away with the closed focus which may be responsible for bacteremia and further metastatic abscesses. The hormone should not be withdrawn or decreased during the stress of infection; its dosage may need to be increased, especially prior to being subjected to an anesthetic or surgical procedure. It may be advisable after the infection has come under control to decrease or withdraw hormone therapy in order that the arthrotomized joint may close. While a patient receives constant high level antiphlogistic hormone therapy drainage may go on indefinitely from an open joint. When the patient's general condition has improved and drainage from the arthrotomy incision has decreased in quantity and changed in character to a serous type the antiphlogistic hormones can be slowly withdrawn and terminated. Their withdrawal is often associated with a great increase in discomfort from the rheumatoid disease, weakness and psychic depression and the patient may be difficult to manage.

The outcome of a pyogenic infection of a joint varies with the age of incidence, the mode of infection, the joint involved and the promptness of adequate treatment. In infants and very young children, in whom the infection most often follows bacteremia or complicates an infectious disease, mortality is highest. In older children and adults, death is rare.

The knee, shoulder, elbow and hip joints tolerate infection better than do the wrist, ankle and smaller joints of the foot and hand. In metastatic bacterial infections of the knee, shoulder, elbow and hip with prompt diagnosis and adequate intelligent treatment, it can be anticipated that a good range of mo-

tion will be salvaged. In the wrist, ankle and smaller joints of the hands and feet, even though treatment is prompt and intelligent and the infection mild, there is usually a great loss of normal motion.

In infants and young children who have not completed growth, because of the fact that the epiphyses are juxta-articular or even intra-articular as in the hip joint, growth disturbances may follow a joint infection. The aberrations of growth may manifest themselves as discrepancies in length or as directional deformities, for example, knock knee bowleg or increase of the carrying angle at the elbow the outcome depending on the portion of the epiphyseal line which is disturbed. Immediately following a joint infection, there may be an acceleration of growth, with length gain in an extremity of as much as 2.5 cm. This may occur during the first year but it is often followed by deceleration of growth so that, as adult life is approached and growth terminates the extremity is shorter than normal.

Infection of the hip joint may be complicated by spontaneous pathologic dislocation of the femoral head. This can be prevented by the use of traction with the leg abducted. Dislocation of the femoral head, if recognized early can usually be easily repositioned by simple traction. Sequestration of the capital femoral epiphyses occurs in infants and children. This is a serious complication and is followed by profound growth disturbance and severe deformity.

An infected joint which is secondary to adjacent osteomyelitis has a poorer prognosis for loss of motion, growth disturbance and deformity than the joint which becomes infected from bacteremia.

Fibrous or bony ankyloses will inevitably result in some infected joints. When ankylosis is in the proper functional position for the individual joint, the disability which results from the loss of motion is minimized and the necessity for subsequent corrective surgery obviated. With proper care, neither ankylosis of a joint in a nonfunctional position nor the loss of remaining motion in a useful arc should occur.

TUBERCULOSIS OF THE BONES AND JOINTS

Tuberculosis is one of the oldest diseases in medical history. The multiple clinical manifestations of this infection were well recognized long before Koch discovered the tubercle bacillus in 1882.

Tuberculosis of bones and joints is the

most common of the osseous infections. The spine is most frequently involved in osseous tuberculosis and the weight bearing joints of the lower extremities are more commonly affected than the joints of the upper extremities.

The infection occurs most often in childhood during the period of rapid bone growth, the greatest incidence falling between the first and sixth years of life.

There are variations in racial susceptibility to the tuberculous organism. The Negro, Mexican and Indian peoples show the lowest resistance, the Mongolian slightly more, while the Caucasian has the least susceptibility.

There are three types of tuberculosis bacilli which may produce the disease—the human, bovine and avian. In the United States the human type is the most common organism found, but in areas where cattle inspection and milk pasteurization are neglected, the bovine type of infection in bone is most often seen. The avian type rarely infects human beings.

The involvement of the bone or joint is secondary to tuberculous infection elsewhere in the body. It suggests the presence of pulmonary glandular or gastrointestinal foci. The most common route for bone infection from these foci is the blood stream. Minute bacterial emboli of tuberculous organisms circulate in the blood stream and lodge in the small end-arteries of the bones adjacent to the epiphyseal cartilage. The bacteria multiply and tubercles form which enlarge into small local abscesses. This process is typically without secondary bone reaction.

This local bone infection by gradual extension may erode through the articular cartilage into the joint or may progress along the epiphyseal cartilage extending into the adjacent capsule and synovium to involve secondarily the joint. In other instances, more commonly seen in adults than in children, the infection may start primarily in the synovial lining of the joint, producing primary synovial tuberculosis.

Characteristically tuberculous infection in bone is primarily a lytic process with very little tendency toward new bone formation.

When the infection enters a joint a fibrinous exudate covers the articular cartilage which gradually becomes organized into granulation tissue. At times the articular cartilage may sequester because of subchondral abscesses in the bone. The tuberculous pus which is formed may dissect into the adjacent tissues of the joint owing the

planes of least resistance develop abscesses in areas removed from the site of the immediate infection.

Tuberculous infection differs from acute pyogenic infection by the absence of local heat and redness. The abscess formation in the soft tissues is spoken of as the cold abscess or tumor albus. These cold abscesses may in the process of healing, go on to complete calcification. Occasionally the cold abscess may become secondarily infected following aspiration or drainage and then may behave the same as an acute pyogenic infection.

Tuberculosis of bone may occur as three distinct clinical types: chronic, acute and subacute. The chronic type has an insidious onset, with slow progress without tendency to form abscess. This is frequently spoken of as *cries sicca*. The acute type runs a rapid course, with high fever, more severe pain than in the chronic type and frequent abscess formation, at times it simulates an acute pyogenic infection. The subacute type, the most common type of osseous infection, follows a more moderate course characterized by low grade fever, subacute pain and progressive deformity. Frequently soft tissue abscess formation occurs.

The symptoms of bone and joint infection vary widely with the type of infection and rarely are the symptoms diagnostic. The general complaints of malaise, loss of appetite, low-grade fever and night sweats are frequently present. The local symptoms consist of pain, and muscle spasm in an attempt to protect the affected joint, producing a gradual progressive deformity. Boggy swelling of the joint is common with very little increase in local heat. Tuberculous joint disease is usually monoarticular. In children "night cries" are not uncommon.

A positive diagnosis of tuberculosis is made by the tubercle bacillus demonstrated by smear or culture from joint aspiration, positive identification of the tubercle bacillus in biopsy specimens of infected tissue and the production of active tuberculosis in guinea pigs inoculated with infected material.

A tuberculin skin test is of greatest value in a negative manner. It is rare to obtain a negative reaction to a tuberculin skin test in a patient with active tuberculosis. A positive reaction to the skin test is suggestive, but not conclusive, evidence of the disease in the young patient.

The roentgenogram in bone tuberculosis characteristically shows atrophy and destruction of bone with loss of joint space without

new bone formation. Because of frequent subchondral abscess formation, the x-ray film may reveal a scalloped appearance along the joint line which is peculiar to tuberculosis. There are frequent soft tissue abscesses which may be recognized on the roentgenogram.

The presence of pulmonary tuberculous lesions may be an aid in reaching the diagnosis of osseous tuberculosis.

Tuberculosis of the bone or joint must be considered as a local manifestation of a systemic disease and as such the treatment should be divided into two aspects: those measures which are directed to the control of the general systemic disease and the local measures which are employed in the management of the specific joint.

Until recent years there had been no specific treatment for tuberculosis.

At the present time, there are increasing favorable reports of the usefulness of streptomycin in combination with para-aminosalicylic acid for the control of tuberculous infection.

More recently the isonicotinic acid preparations have been used and seen to be of great value, especially in the treatment of severe chronic tuberculosis of the bones and joints with multiple chronic draining sinuses.

While it is still early to speak with complete assurance concerning the control of tuberculosis by the use of these medications, all indications have suggested that they are contributing a great deal in the control of the disease process and these drugs should be employed in the treatment of osseous and synovial tuberculosis. In addition to the specific medications it has for years been recognized that wholesome hygienic living conditions, fresh air and a well-balanced diet are of great value. Bed rest is of extreme importance during the acute phase of the disease. Adequate control of the anemia and hypoproteinemia is necessary. In osseous lesions heliotherapy as popularized by Rollier is indicated as an aid to local treatment but should be given in controlled dosage and its use not abused.

Local treatment of joint disease consists of proper immobilization of the involved joint in the functional position by continuous traction, casts or bracing.

When tuberculous disease has resulted in damage of the articular cartilage especially in weight bearing joints, return to activity may result in reactivation of the disease. As a result, operative fusion is frequently used to avoid recurrence.

Surgical treatment is employed when the

disease process is under control, both from the systemic and local standpoint. The disease is considered under control when there is relief of the pain and spasm, the absence of fever, a normal sedimentation rate and no progress in abscess formation.

Operative fusion may be intra-articular or extra-articular or a combination of both methods.

Abscesses. The cold abscess of tuberculosis is treated by aspiration or incision only with impending rupture or severe discomfort. The potential of secondary infection in these abscesses is always present and aspiration when undertaken is usually accomplished by introducing the needle through normal tissue to enter the abscess, rather than piercing the skin immediately over the infected tissue.

In rare instances when the abscess is completely isolated, total surgical excision of the abscess may be undertaken. When there are multiple draining sinuses great benefit has been observed with the use of chemotherapeutic agents such as streptomycin, para-aminosalicylic acid and isonicotinic acid. When these drugs are used, care must be taken in careful observation of the patient in order to avoid undesirable side reactions.

Tuberculosis of the Spine (Pott's Disease)
Tuberculosis of the spine is commonly spoken of as Pott's disease as this condition was first described by Sir Percivall Pott in 1779 and was considered then to be of tuberculous origin.

In tuberculosis of the bones, the spine is the most frequently involved structure. Although the highest incidence of Pott's disease is in children, 50 per cent of the cases occurring in children between three and five years of age the disease does occur frequently in adults. The area of the spine most commonly affected is that portion of greatest mobility the tenth dorsal to the first lumbar segments. The disease may occur in any portion of the cervical, dorsal or lumbar spine.

The anterior part of the vertebral body is the most commonly involved, but the disease may attack the posterior process, lamina or the transverse processes (Fig. 149). Often the intervertebral disk is the earliest site of the infection and narrowing of this space may be the first x-ray evidence of the disease. Several areas of involvement are not uncommon. With bony destruction of the vertebral body a progressive collapse and posterior angulation of the spine develops, resulting in the kyphosis which is frequently seen in Pott's disease.

Abscesses form along the anterior margin



Figure 149 Early tuberculous of the spine.

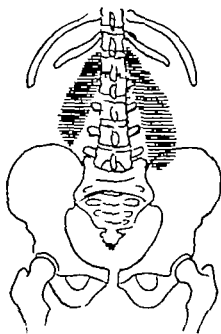


Figure 150 Psoas abscess (bilateral)

of the vertebral bodies and in the dorsal area, the abscess remains in the paravertebral space and is usually globular in shape. The abscess may readily be recognized in the anteroposterior roentgenogram as a paravertebral shadow adjacent to the area of the bony involvement. Abscesses arising in the lower dorsal and lumbar region may extend along one or both psoas sheaths into the pelvis (psoas abscess Fig. 150). These at times may present themselves in either the femoral or inguinal area under Poupart's ligament. Less often, the abscess may point posteriorly in Petit's triangle and, on rare occasions, may point in the gluteal region.

The general symptoms of tuberculous infection are frequently present. The local symptoms of spine involvement depend on the area involved. Pain and weakness in the

back associated with stiffness and muscle spasm are common. Local tenderness to percussion may be present.

Night cries are frequent in children during the early stage. A change in gait may be noticed due to protective mechanisms to avoid jarring.

In the cervical lesions motions of the head and neck are limited and the head may be supported by the hands.

Dorsal lesions are more silent and give less evidence of limited mobility because of the natural rigidity of this portion of the back.

Lumbar spine involvement shows a loss of flexion and extension with marked protective splinting of the muscles. Any of the signs and symptoms of chronic low back conditions may be present.

With irritation of the spinal nerve roots, there may be radicular pain and, in the lower lumbar region, symptoms simulating ruptured intervertebral disk syndrome are not unusual.

With irritation of the psoas muscle, hip flexion contractures may develop suggesting hip disease.

Occasionally the first sign of Pott's disease may be a bulging in the inguinal or femoral region from a psoas abscess which may be mistaken for a hernia.

The kyphosis of Pott's disease is a late finding resulting from vertebral body collapse and posterior angulation (Fig. 151).

Involvement of the spinal cord is not uncommon with tuberculosis of the vertebral bodies and the associated abscess formation.

It may appear in about 10 per cent of the cases.

The area most frequently involved is the upper and mid-dorsal region. The spinal cord involvement is usually secondary to extension of the granulomatous tissue with encroachment on the spinal cord rather than collapse and angulation of the spine. The lesion may progress to complete paraplegia. If regression of the symptoms is not rapidly obtained with rest, medication and splinting drainage of the tuberculous abscess by costotransversectomy is indicated. Laminectomy is usually not advisable.

In tuberculosis of the spine the roentgenogram is of great aid in establishing the diagnosis. The demonstration of the narrowing of the intervertebral disk and the lytic destruction of the adjacent vertebral bodies accompanied by paravertebral abscess is extremely suggestive. A positive reaction to the tuberculin test is corroborative evidence, especially in patients of the younger age group. A negative reaction to the tuberculin test, as mentioned before is of greater value in eliminating the possibility of tuberculosis. Confirmation of the diagnosis occasionally may be obtained by aspiration of the abscess and recovery of the organism. A presumptive diagnosis is aided by the presence of tuberculosis elsewhere in the body usually of the pulmonary or gastrointestinal type.

Conditions which stimulate tuberculosis of the spine especially in the adult, are low grade pyogenic osteomyelitis—the proliferative bony changes seen in pyogenic osteomyelitis are an aid in differentiation malignant disease, chronic arthritis and fractures of the vertebral bodies other granulomatous infections such as actinomycosis.

As in all forms of osseous tuberculosis, the treatment of tuberculosis of the spine should be divided into the general and local measures.

The general supportive measures are those mentioned for the treatment of systemic tuberculosis including the use of the beneficial drugs, associated with supportive measures of rest, well balanced diet and heliotherapy.

The spine should be given complete rest and, in the earlier phase of treatment of this disease, the patient should have bed rest either in a plaster shell, a Whitman type frame or Stryker bed (Figs. 152 and 153). By means of these devices the patient can be turned for his bathing and general body care without putting torsional strain upon the infected area of the spine.



Figure 151 Tuberculosis of the spine with collapse and kyphosis.

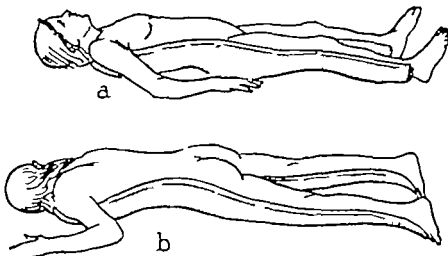


Figure 152 Plaster shell. a, Supine b. prone.

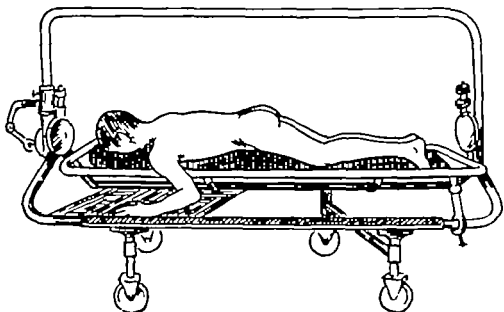


Figure 153. Stryker bed with patient in prone position. Position may be reversed to supine.

In the past, many attempts have been made to hyperextend the spine gradually to prevent vertebral body collapse; however it is generally felt at the present time that hyperextension of the spine is to be avoided, as the healing process will be benefited by the gradual contact of the adjacent infected vertebral bodies and that treatment in a flat or neutral position is more conducive to healing than the position of hyperextension used in an attempt to prevent deformity.

Recumbency with protection of the spine is maintained for prolonged periods until the disease process shows clinical and x-ray evidence of becoming arrested. The average period of recumbency is about eighteen months.

Operative fusion of the posterior processes of the spine throughout the diseased area,

incorporating one or two vertebrae above and below the involved area, is used after the active infection has become controlled. Operative fusion is a further means of fixation of the affected spine using the bony fusion as an internal means of support in addition to the external support of braces or casts.

Prolonged immobilization of the spine after healing, either following conservative or operative treatment, is indicated. A brace or cast (Fig. 154) is employed during the period of beginning ambulation and is continued over a period of eighteen to twenty four months.

Tuberculous abscesses, as a rule, are not opened unless there is pain or threatening rupture. Most abscesses will retrogress under conservative treatment and frequently go on to complete healing by

lon.

Tuberculosis of the Sacroiliac Joint. Tuberculosis of the sacroiliac joint is quite rare in children but is occasionally seen in adults and sometimes may be bilateral.

Abscess formation is frequent in this infection and the tendency is to point anteriorly more often than posteriorly. The abscess may point into the groin into the iliac fossa, into the buttocks, passing through the sacrospinous notch, and into the ischioanal fossa.

The onset of tuberculosis of the sacroiliac joint as a rule is slow and the abscess may be the first symptom. Pain in the back may be increased with sitting or walking and is accompanied by signs of chronic low-back pain with limited back motions. Pain is elicited on iliac compression, being felt on the affected side.

The x-ray changes seen in tuberculosis of the sacroiliac joint are atrophy of the bone on the adjacent margins of the sacroiliac joint and scalloping of the articular margins of the joint due to subchondral abscesses. The differential diagnosis must exclude the complaints of lumbosacral and sacroiliac strain and the intervertebral syndrome. Sciatica is frequently complained of by patients with this disease. Other types of infectious processes must be ruled out.

The usual supportive measures should be used and the patient should be treated in the recumbent position. The back and hips are immobilized by a bilateral hip spica cast and, later operative fusion of the affected joint is undertaken either by the intra-articu-

lar or extra-articular routes when the disease process has shown evidence of subsiding.

Tuberculosis of the Hip. This type of tuberculous infection also is seen most commonly in children. It has been stated that 85 per cent of the cases occur in children younger than ten years of age. The remainder have approximately equal distribution in persons up to sixty years of age. In tuberculosis of the bones the frequency of tuberculosis of the hip is second only to that of the spine.

The process may start as a primary synovial involvement or the original infection may lodge in the terminal vessels of the capital epiphysis of the femur or the marginal epiphysis of the acetabulum.

It is felt that trauma may be a localizing agent for the infection.

There may be three distinct types of the disease—acute, subacute and chronic. The symptoms vary accordingly.

The general symptoms of tuberculous infection are low-grade fever, malaise, loss of weight, anorexia and night sweats.

The local symptoms are limp and pain in the affected hip or pain referred to the knee. In children, night cries are occasionally noted. Deformity is frequently insidious. As a rule abscess formation becomes apparent later in the disease. With the acute type, all symptoms are more pronounced, and with the rapid lysis of the bone, absorption of the femoral head or development of a "wandering acetabulum" may result.

X-ray examination frequently reveals capsular swelling with early decreased density of the adjacent portions of the ilium and acetabulum. Later loss of joint space and gradual destruction of the femoral head or acetabulum, or both, are observed. The reaction to the tuberculin skin test may frequently be positive to the dilute strength of tuberculin. The diagnosis may be confirmed by aspiration and recovery of the bacilli either on smear culture or guinea pig inoculation. Occasionally biopsy of the hip joint may be necessary for positive diagnosis.

The differential diagnosis of tuberculosis of the hip in children is frequently difficult. The more common hip afflictions which must be considered are Perthes disease, traumatic epiphysitis, low-grade osteomyelitis of the upper end of the femur, infectious arthritis, irritative lesions of the psoas muscle, seen occasionally in association with lymphadenitis in the iliac fossa, and arthrofibrosis (Luck).

Supportive measures previously described



Figure 154. Taylor type of back brace for support of spine.

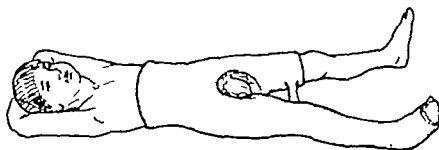


Figure 155 Hip spica cast for immobilization of hip or knee

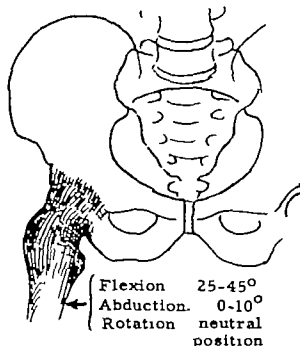


Figure 156. Extra-articular arthrodesis of the hip (healed)

should be used in conjunction with the chemotherapeutic drugs.

Bed rest with immobilization of the affected hip either by traction or hip spica cast (Fig. 155) in the functional position should be instituted. This conservative treatment is reinforced by drug therapy until the patient shows evidence of general improvement with x-ray evidence that the disease process is arrested and improving. Efforts are made to maintain the red blood count, hemoglobin and serum protein at normal levels. When the sedimentation rate approximates normal, surgical treatment of the lesion may be considered.

Surgical fusion of the hip joint by intra-articular extra-articular or combined methods may be considered at the point when the disease is under control. This is particularly warranted when there has been serious destruction of the articular cartilage. Operative fusion is undertaken maintaining the hip in a

functional position, which consists of approximately 25 to 45 degrees of flexion neutral in regard to rotation and 0 to 10 degrees of abduction (Fig. 156)

Following the operative procedure, the patient should continue to be kept in a protective cast for the period necessary to obtain solid bony union and the hip further protected for an additional six months to a year until solid healing has been completely obtained

Tuberculosis of the Knee. Tuberculosis of the knee is the third most common type of osseous tuberculosis. It is predominantly a disease of children. Infection is secondary to pulmonary or gastrointestinal tuberculosis and the infection is considered to be blood borne. Bacterial emboli lodge in the epiphyseal cartilage and form tubercles. The infection enters the knee joint by extension along the capsular structures or directly through the articular cartilage. There are instances in which the infection may be primarily in the synovium and, when the infection enters the joint, the synovium becomes thickened and granulation tissue covers and destroys the articular cartilage. This goes on to a granulomatous process associated with tuberculous pus and produces a thick, boggy swollen knee unaccompanied by heat or redness. Abscesses frequently develop

The onset is usually marked by a slight limp with limitation of knee motion. This is followed by swelling of the joint associated with moderate pain and tenderness. The patient gradually develops a flexion deformity: the knee becomes progressively enlarged, boggy and painful. These symptoms are usually associated with the other general systemic manifestations.

The diagnosis of tuberculosis of the knee is aided by the knowledge of existing systemic tuberculosis. A positive reaction to the skin tuberculin test in early childhood also is suggestive. X-ray changes may or may not be present. If the disease is primarily synovial tuberculosis the evidence will

be mild bone atrophy. In the more advanced disease of osseous tuberculosis there is narrowing of the joint space, lytic destruction of the subchondral bone and fragmentation of the articular cartilage.

Positive identification of the tubercle bacillus in cultures from the aspirated material of the knee joint or from a guinea pig inoculated with the material confirms the diagnosis. When there is doubt as to the proper diagnosis, a tissue biopsy may be the only means of obtaining a positive diagnosis.

Tuberculosis of the knee may occasionally be difficult to differentiate from chronic synovitis, atrophic arthritis, rheumatic fever in children, low-grade pyogenic arthritis and hemophilia, with repeated bloody synovial effusions.

The usual supportive measures as previously described for other osseous forms of tuberculosis are essential.

Complete rest of the affected joint in the position of function is desirable. Immobilization of the knee may be obtained by either traction or a hip spica cast. If deformity has developed before the treatment is undertaken, it is necessary to correct the flexion deformity.

In the great majority of patients bony ankylosis for a permanent cure is desirable. After the acute disease process has been brought under control with the supportive measures of proper drugs, heliotherapy, rest and immobilization, operative fusion of the knee is desirable, especially in adults.

Surgical arthrodesis is usually of the intra-articular type with removal of the synovium and articular and semilunar cartilages. The freshened bone ends are shaped and brought into contact. The patella is frequently used as a bridge graft from the femur to the tibia. The knee is fixed in a position of function somewhere between 145 and 170 degrees of extension, depending upon the type of occupation and the age of the individual. In children, it is advisable to fix the knee in more extension as further growth of the lower femoral epiphysis will tend to cause the knee to grow into flexion.

Occasionally in patients with synovial tuberculosis who are seen quite early in the course of the disease, arrest and cure of the process may be effected without surgical management, however this is extremely rare and one should watch these patients carefully for exacerbation of the disease process on resumption of activity.

Operative fusion may take from four to nine months to solidify and should be pro-

tected during this period with a long leg hip spica cast subsequently supported by a caliper brace to avoid weight bearing until complete bony union is obtained.

Tuberculosis of the Ankle and Foot. Tuberculosis of the ankle and foot is more common in males than in females and is not common in children under ten years of age.

The primary lesion in tuberculosis of the ankle and foot is most frequently osseous when it occurs in children and synovial when it occurs in adults. Occasionally tuberculosis in the foot starts as tenosynovitis and subsequently enters the tarsal joints because of the intimacy of the tendons to these joints. There is a tendency for the infection to spread and it may involve the entire tarsus. Abscess formation in tarsal tuberculosis is extremely common.

When the infection remains restricted to the ankle or to one tarsus the prognosis is good. When there is evidence of multiple infection and sinus formation, the prognosis becomes extremely poor and amputation may be the treatment of choice to prevent general spread of the infection.

In the child conservative treatment with immobilization for at least twelve months is indicated, with the cast extending from the toes to above the knee with the foot in a functional position. When a single joint is involved, arthrodesis of the involved joint may be accomplished when the disease process shows evidence of being under control.

Extra-articular arthrodesis in tuberculosis of the ankle may be accomplished satisfactorily with a good end result provided there has not been too extensive destruction of the joint.

In patients having fulminating tarsal tuberculosis early amputation may be necessary because of the great tendency for the disease to metastasize to other parts of the body.

Tuberculosis of the Shoulder. Tuberculous infection of the shoulder is most often of the chronic type (*caries sicca*) and involves the bone rather than the synovium of the joint. There is frequently no enlargement of the joint. This type of tuberculosis does not have the tendency to abscess formation.

The history is that of gradual onset of pain in the shoulder with progressive loss of motion. If an abscess forms it usually points anteriorly. The disease is usually associated with active pulmonary tuberculosis. The clinical picture of pain in the shoulder with

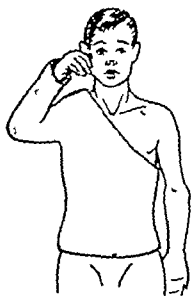


Figure 157 Shoulder spica cast

gradual loss of motion associated with tuberculous processes in other portions of the body accompanied with x ray changes, aids in the diagnosis.

Differential diagnosis consists in ruling out the possibility of chronic subdeltoid bursitis, adhesive capsulitis and rheumatoid arthritis.

The frequent occurrence of nontuberculous adhesive capsulitis in patients with pulmonary tuberculosis under treatment with bed care is well known and it is a constant source of disturbance to the clinician to exclude the possibility of active tuberculosis of the shoulder in these patients.

Tuberculosis of the shoulder is treated by immobilization of the shoulder in a plaster spica cast with the shoulder held in a position of function of approximately 65 degrees of abduction, 35 degrees of forward flexion and 35 degrees of external rotation (Fig. 157). The patient is treated, as in other forms of osseous tuberculosis, in the position of rest until the disease process has come under control and evidence of other foci of tuberculosis does not contraindicate surgical management. Then operative fusion of the shoulder is indicated. Combinations of intra-articular and extra-articular types of arthrodesis are performed and, following the operative procedure, immobilization in a shoulder spica cast is continued until solid bony fusion is obtained. Occasionally metallic internal fixation may be used, transfixing the head of the humerus to the glenoid process of the scapula, to provide stability while bony union is occurring. The functional result of arthrodesis of the shoulder is quite satisfactory when the shoulder has

been fused in the proper position. The mobility of the scapula on the thoracic cage allows for adequate movement of the arm up to a point of approximately 90 degrees of abduction and will enable the patient to get the hand to the head and mouth.

Tuberculosis of the Elbow As in other types of osseous tuberculosis, this infection usually starts in the bone. The infection then extends to gradually involve the joint proper. Tuberculous infection of the elbow is more common than that of the shoulder and carries the best prognosis of any joint which may be involved by tuberculosis with regard to healing with some residual useful motion.

The usual symptoms are gradual swelling, pain and stiffness about the elbow with doughy thickening of the elbow joint which may go on to abscess formation. Motion is painful.

If treatment is instituted early with immobilization and general supportive measures, the prognosis is good for healing and, if the disease occurs in childhood, useful motion may be obtained after healing is complete. In adults, it may be necessary to undertake operative fusion as a final definitive course of treatment. In some instances, extra-articular fusion may be accomplished with bone grafts extending from the humerus to the olecranon.

With surgical arthrodesis of the elbow the arm should be maintained in a functional position with the elbow at a right angle and the forearm in midposition in regard to pronation and supination.

Tuberculosis of the Wrist Tuberculous infection of the wrist is not common. It is usually secondary to infection of the carpus or metacarpal bones with extension into the wrist joint. The disease is frequently quite disseminated throughout all the carpal bones and, because of the superficial nature of the wrist joint, the swelling becomes quite marked. The symptoms of the disease are manifested by pain, swelling and restricted motion of the wrist. Frequently there is a flexion contracture of the wrist and there are large fusiform swellings extending in the tendon sheaths above and below the carpal ligament.

Carpal tuberculosis occurs in adults rather than in children and is associated with other tuberculous infections. Positive diagnosis can be rather readily obtained by aspiration of the joint and demonstration of the organism.

The prognosis in tuberculosis of the wrist must be somewhat guarded. General systemic treatment is indicated along with drug

therapy. The wrist should be immobilized in a long arm cast and held in a functional position of 20 to 30 degrees of dorsiflexion with the forearm in midposition. The cast should incorporate the elbow to eliminate motion at the distal radioulnar articulation.

After the disease has become quiescent, arthrodesis of the wrist may be achieved by operative procedure. A cancellous bone graft should extend from the radius to the meta carpal bones. It is preferable to delay operative treatment until the local process has become quiescent.

Bursal Tuberculosis. A bursal type of tuberculosis may be encountered in which there is a primary infection of the gluteal bursa lying over the greater trochanter of the hip. This occurs as primary tuberculosis and is manifested by pain and discomfort about the hip with local swelling. In the earlier stages, the bone is not involved but secondarily the trochanter may become involved.

Positive diagnosis is made by aspiration or biopsy.

Treatment of this condition consists of the

general supportive measures as previously outlined and early surgical excision of the trochanteric bursa. If the bone of the trochanter is involved partial excision of the greater trochanter may be undertaken. If the soft tissue or early bone infection is not treated the disease may progress ultimately to involve the hip joint with much more serious results. This type of tuberculosis is one which is seen in adults.

There are rare occasions of tuberculosis of the metaphysal portion of the bone without involvement of the joints.

READING REFERENCES

- Bennett, G., in Anderson, W. A. D. Pathology, 2nd ed. St. Louis, C. V. Mosby Company 1933, Chap. 43.
- Blanche, D. W., Osteomyelitis in Infants. *J. Bone & Joint Surg.* 31-A:71 1952.
- Forbus, W. D.: Reaction to Injury. Baltimore, Williams & Wilkins Company vol. I, 1943.
- Ropes, M. W. and Bauer, W. Synovial Fluid Changes in Joint Diseases. Cambridge Mass., Harvard University Press, 1953.
- Speed, J. S., and Knight, R. A., eds. Campbell's Operative Orthopaedics, St. Louis, C. V. Mosby Company 1949 vol. I.

Tumors of the Bones, Joints and Soft Parts of the Extremities

By MURRAY M COPELAND M D
and CHARLES F GESCHICKTER M D

MURRAY MARCUS COPELAND was educated at Oglethorpe University in his native state of Georgia. Graduating in medicine from Johns Hopkins University he became a fellow in surgical pathology. This interest led him logically into the field of tumor surgery in which he developed his talents further at Memorial Hospital, New York, and Union Memorial Hospital, Baltimore.

CHARLES FAREBORN GESCHICKTER graduated from George Washington University and Johns Hopkins University. He is a product of the well-known school of surgical pathology developed in the surgical laboratory of Johns Hopkins made famous by Joseph Bloodgood. He is one of the few pathologists who have combined the discipline of pathology with the clinical application of its principles.

In appraising any method of therapy it is necessary to know the essential pathology, the natural history of the disease and what influence specific therapy may have upon it. The classification of bone tumors based upon the relation of bone development and subsequent tumor formation separates the tumors into specific types each of which follows a

definite course. A survey of these groups reveals the effect of treatment more accurately than would be possible otherwise and prognosis is thus placed upon a sound basis.

A brief survey will be given of each group, the treatment used and where irradiation seems of benefit, the physical agent used. No comprehensive rule can be laid down as to

Classification of Bone Tumors*

TUMORS OF OSSIFEROUS ORIGIN

Cartilaginous
Osteochondroma,
solitary and multiple
Chondroma
Chondroxytoid fibroma
Chondroblastoma,
benign and malignant
Chondrosarcoma,
primary or secondary

Osteous
Osteomas and ossifying fibromas
of skull and jaws
Osteodystrophia
Osteogenic sarcoma, sclerosing
and osteolytic
Parosteal osteoma and myositis
osificans

Resorptive
Bone cyst
Diffuse osteitis fibrosa (para-
thyroidism)
Fibrous dysplasia, polyostotic
or monostotic
Giant cell tumor

TUMORS OF NON-OSSEOUS ORIGIN

Marrow and Hemopoietic Systems
Ewing's sarcoma
Primary reticulum sarcoma
Multiple myeloma
Chloroma and leukemia of bone
Reticuloendotheliosis
Xanthomas and granulomas
of bone

Metastatic Deposits
Carcinoma of prostate, breast,
kidneys, etc.
Metastatic lymphomas and
sarcomas

By Inclusion or Direct Inoculation
Chordoma
Angioma, angiosarcoma
Fibroma and fibrosarcoma,
fascial or nerve sheath
Myosarcoma
Liposarcoma

After Geschickter, C. F. and Copeland, M. M.: Tumors of Bone. J. B. Lippincott Co.

whether a tumor process is radiosensitive. Experience has been our best teacher as to the behavior of tumors under radiation by radium, x-rays or radioisotopes.

They are not radiosensitive. Isolated tumors of this origin have been affected by irradiation or have been treated successfully using irradiation as an adjunct to surgery.

TUMORS OF OSSEOUS ORIGIN

In general, surgery offers the best method of treatment in tumors of osseous origin.

CARTILAGINOUS TUMORS

Osteochondroma (Exostosis) This tumor presents a surgical problem when treatment

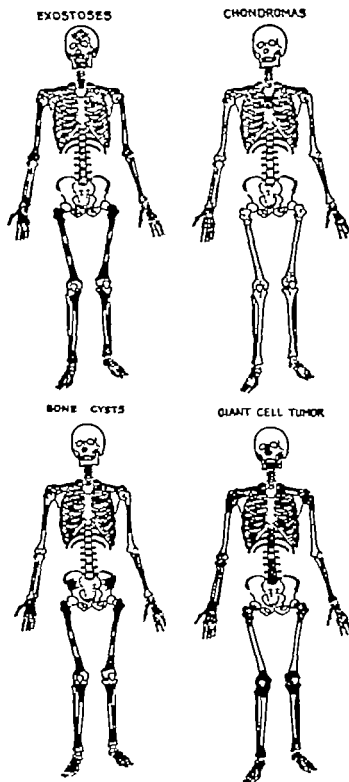


Figure 153 Charts showing the skeletal sites involved in benign tumors of bones. The solid black areas indicate the most frequent sites, the checked areas indicate the common sites and the diagonal lines indicate the occasional sites.

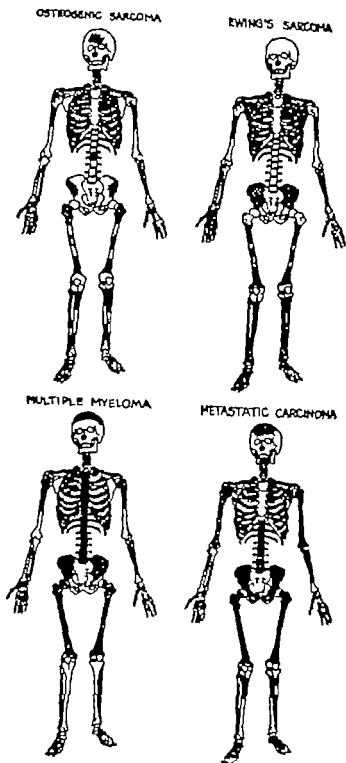


Figure 159 Charts showing the skeletal sites involved by malignant tumors of bones. The solid black areas indicate the most frequent sites, the checked areas indicate the common sites and the diagonal lines indicate the occasional sites.

is indicated. It forms the largest group of benign tumors arising from precartilaginous connective tissue in the skeleton and is found most frequently near the ends of the long bones of patients between the ages of ten and twenty-five years. The other sites of importance are the bones of the thoracic cage

and the small bones of the foot and hand. The distinctive diagnostic features include the pedicle of normal bone protruding from the bone cortex and the rim of cartilage overlying it. A similar structure may be seen microscopically. The roentgenogram demonstrates the compact differentiated outgrowth



Figure 160. Roentgenogram showing osteochondroma of the sessile type. Note the widened metaphyseal region near the outgrowth of the tumor. The features of the growth depicted are typical.

of bone and an overlying cartilaginous cap flecked with calcium (Fig. 160). Instances of single osteochondromas without symptoms frequently go unnoticed. The tumor may be left untreated but should be watched by interval roentgenographic examination, since it may undergo secondary malignant change, particularly after the patient reaches the age of thirty years. Simple excision usually suffices to cure the osteochondroma which produce pain or dysfunction. Secondary malignant change occurs in about 8 per cent of these neoplasms.

In *hereditary or multiple osteochondromas* (Fig. 161) the regions most frequently and severely affected are those of the forearm and leg, the bones of which may be fused at one point. The prognosis as far as life is concerned is good, but there is no adequate form of treatment, except operation, for correction of deformities and removal of osteochondromas causing pain or dysfunction. In a few of the patients with multiple skeletal involvement, secondary malignant change is observed in one or more cartilaginous growths microscopically showing chondromyxosarcoma. Resection or amputation when necessary is the treatment of choice.

Chondroma Chondroma, a common type of central cartilaginous tumor is often classi-



Figure 161. Roentgenogram of hereditary deforming chondrodysplasia or multiple exostoses. Note the widening of the metaphyseal regions of the long bones and tendency to develop outgrowths of osteochondromas.



Figure 162. Photomicrograph taken from the margin of the central chondroma of the phalanx seen in Figure 164. The tumor is composed of adult hyaline cartilage.



Figure 163. Roentgenogram of a large central chondroma of congenital origin occurring in a long bone. The defect shows longitudinal streaking, destruction of cortex and calcareous stippling, typical of chondroma. See Figure 164 for benign osteochondroma of the phalanx.

Bed with benign exostoses or osteochondromas. It occurs in the small bones of the hands and feet, also in the long bones, spine, ribs and sternum. In the small acral bones

the tumor is composed of radiolucent cartilage and appears as a central lesion with rarefaction. It is visible in the roentgenogram as a cystic area, flecked with circumscribed, dense areas of calcification, within a shell of cortical bone. Histologically a central area of cartilage with orderly arrangement of cells is usually seen (Fig. 162). In some chondromas fetal cartilage may be present in varying amounts. The lesions are rarely multiple and occasionally affect long bones (Fig. 163). In deciding whether a particular chondroma is to be treated from the benign or malignant standpoint, the location rather than the pathologic changes, is given primary consideration. Lesions in the small bones of the hands and feet may be looked on as benign and curable by thorough extirpation by curettage and chemical cauterization (Fig. 164). Only rarely has a central chondroma of the phalanges, metacarpals or metatarsal bones shown malignant propensity. True chondromas of large size occurring about the sternum or in the long bones must be looked upon as potentially malignant. They should be removed if possible. Roentgen ray or telerradium therapy has been of benefit in retarding the growth in a few cases.

Maffucci's syndrome or dyschondroplasia with hemangiomatosis is a nonhereditary mesodermal dysplasia. Skeletal development may be retarded on one side or in individual bones, principally the bones of the extremi-

ties, thoracic cage pelvis and vertebrae. Multiple central chondromas made up of fetal cartilage, associated with soft part hemangiomas, are observed in the affected bones. The soft-part angiomatous tumors are cavernous to cellular in morphology and appearance. The deformities may appear in early life, becoming stationary in the second decade. Trivial injuries may cause fractures through the chondromatous areas. The patients present marked bone and soft part deformities, occasionally requiring amputation of an extremity for comfort because of loss of function.

Chondromyxoid Fibroma This is a recently described benign tumor which appears to be a variant of central chondroma which arises from blastemal tissue (cartilage-forming connective tissue). The lesion is usually situated in the metaphysial region of a long bone, with the tibia and femur contributing more than half of the chondromyxoid fibromas seen. The small bones of the hands and feet occasionally reveal a similar process. The majority of patients affected fall within the first three decades of life. Pain is the chief symptom and, occasionally palpable swelling is noted. The roentgenogram characteristically reveals an eccentric, sharply cir-

cumscribed rarefied defect. The cortex may be completely destroyed, with or without a faint periosteal reaction (Fig. 165). Trabeculae may be seen traversing the defect. Calcific deposits are usually not visualized, in contrast to those seen in roentgenograms of central chondromas. Histopathologic study reveals fibrous, myxomatous and early forms of cartilaginous tissue (Fig. 166). The cells in some cartilaginous portions of the tumor may show bizarre nuclei of irregular shape and size. The periphery of the tumor may have a concentration of nuclei. Collagen changes of varying degrees are seen.

Where possible, the treatment of choice consists of en bloc resection of the affected portion of bone. In some lesions curettage, followed by chemical cauterization with or without insertion of bone chips, is carried out with good results.

Chondroblastoma—Benign and Malignant. Chondroblastoma arises from a proliferation of cartilage at the epiphysial line and usually occurs in persons between the ages of ten and twenty years. It is most frequently found about the upper end of the tibia, the lower end of the femur and the upper end of the humerus. Symptoms of pain, tumor and

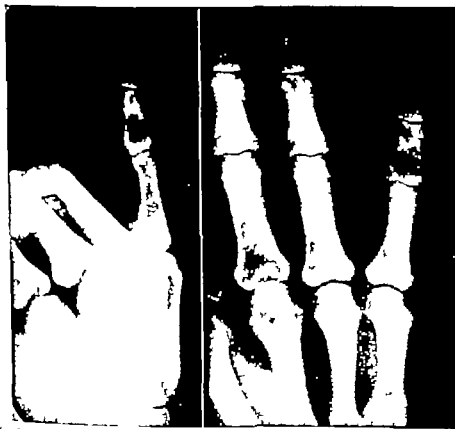


Figure 164. Roentgenogram of central chondroma occupying the middle phalanx of the index finger. Note the thinning of the cortex and faint calcific deposits within the tumor area, successfully treated by curettage and chemical cauterization.



Figure 165 Roentgenogram of tibia showing chondromyxoid fibroma in upper metaphysis. Note the sharp margin of radiolucence.



Figure 167 Roentgenogram of a benign chondroblastoma arising at the epiphyseal line involving the epiphysis and the proximal metaphyseal area. Note the periosteal reaction in the medial side of the tibia. Trabeculation is minimal.

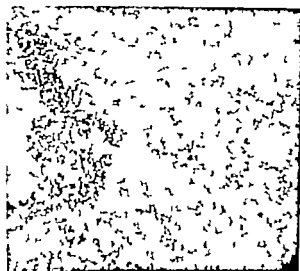


Figure 168 Microphotograph showing myxomatous tissue with areas of condensed nuclei and chondroid changes in chondromyxoid fibroma. No evidence of fibroid reaction present.

dysfunction average five months in duration prior to operation.

In the roentgenogram, there is a characteristic mottled cystic area of bone destruction, with expansion of the bone shell and usually some periosteal reaction (Fig. 167). Not infrequently the tumor bears a marked similarity to giant cell tumor. Microscopically

one sees masses of young cartilage cells of chondroblastic type with areas of uncalcified matrix. Zones of chondroid material are present with characteristic lace-like calcification about residual lacunae. Some areas resemble adult hyaline cartilage. The tumor has areas of marked vascularity. About the vascular areas and the periphery of the cartilage, numerous giant cell osteoclasts are seen (Fig. 168).

Some of these tumors show malignant degeneration, differentiated from the benign lesions only by histopathologic study. Many investigators separate the benign tumors from the malignant variety, placing the latter with the chondrosarcoma group.

Radium or roentgen ray therapy is recommended in the benign lesions following exploration and curettage unless the tumor occurs in a bone which can be resected without sacrifice of function. The malignant variety of chondroblastoma has been uniformly fatal despite primary amputation.

Chondrosarcoma and Osteogenic Sarcoma. These include several subvarieties of malignant tumors which may arise either subperiosteally or centrally in the skeleton from the bone-forming tissues and produce bone, cartilage, osteoid material or preosseous connective tissue. These malignant tumors dis-

place and disrupt the pre-existing bone. They are characterized in the roentgenogram by varying degrees of bone destruction, new bone formation, and periosteal reaction. When bone formation predominates the tumors are referred to as sclerosing or osteoblastic osteogenic sarcoma. When bone destruction predominates, the tumors are highly vascular and are referred to as osteolytic or telangiectatic osteogenic sarcoma. When cartilage rather than bone formation or destruction predominates the tumors are called chondrosarcomas. Primary chondrosarcoma refers to a malignant tumor which affects pre-existing normal osseous structures. Secondary chondrosarcoma refers to similar tumors which arise in pre-existing osteochondromas or chondromas.

All forms of chondrosarcoma and osteogenic sarcoma are more frequent in the long bones, especially in the end of the femur tibia and fibula about the knee joint. They tend to occur in the adolescent or postadolescent period. Sclerosing osteogenic sarcoma and primary chondrosarcoma are largely limited to members of this age group. Osteolytic and secondary chondrosarcomas tend to have a wider age distribution. Regardless of the subvariety or the point of origin, all forms of osteogenic sarcoma tend to affect

the cancellous cortical and periosteal structures of the affected bone. The roentgenogram shows increased density, rarefaction or mottling, and the periosteal zone near the tumor infiltration shows lipping and elevation due to the density of reactive bone formation and the disease beneath it.

The patient usually suffers pain, swelling and lameness in the affected part. The symptoms are of increasing severity and vary from six weeks to six months duration. Trauma is usually coincidental with exacerbation of symptoms rather than being of etiologic significance. There may be a mild degree of fever and leukocytosis. Tenderness, swelling and limitation of motion are often apparent on physical examination. Skin changes and regional adenopathy are not present in patients with early disease. The tumors tend to metastasize to the lungs more often than to the lymphatics.

Adequate roentgenologic examination and surgical biopsy under tourniquet, are the only definitive diagnostic procedures. None of the subvarieties of chondrosarcoma or osteogenic sarcoma are cured by radiation. Radical surgery (amputation, occasionally resection) is the treatment of choice. The five year survivals vary from 10 per cent to 30 per cent.

Primary chondromyxosarcoma arises chiefly in young patients (aged fourteen to twenty one years) and at the sites where tendons insert directly into the bone. The favorite sites are about the knee, the lower femur or upper tibia and also at the shoulder and pelvic girdles. Symptoms of pain average five months in duration before examination. Examination reveals swelling, usually rubbery in consistency. Pathologic fracture is extremely rare. The neoplasm is composed of tumor cartilage, some ossification and areas of cystic destruction.

In the x-ray film, the growth appears as a subperiosteal shadow streaked with calcium spicules. It does not involve the cortex until late in the disease, causing bone destruction (Fig. 169). Five year cures in this group average 15 per cent. Most surviving patients have had amputation performed. Irradiation, however, has relieved pain in many instances and reduced soft-part swelling.

Secondary chondrosarcoma arises from a pre-existing chondroma or osteochondroma, occasionally complicating Paget's disease and hereditary chondrodysplasia. Most of the patients are between the ages of thirty five and fifty-five years. The tumor shows a tendency to occur about the upper humerus,

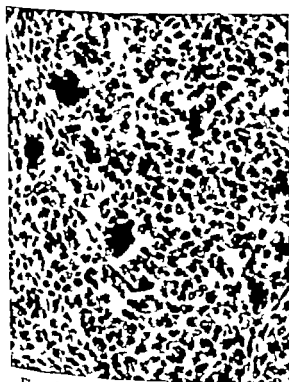


Figure 168. Photomicrograph of benign chondroblastoma. Note the proliferation of chondroblasts and osteoclasts about the area. These lesions are sometimes confused with giant cell tumors. Malignant place.



Figure 165 Roentgenogram of tibia showing chondromyxoid fibroma in upper metaphysis. Note the sharp margin of radiolucence.



Figure 167 Roentgenogram of a benign chondroblastoma arising at the epiphyseal line involving the epiphysis and the proximal metaphyseal area. Note the periosteal reaction in the medial side of the tibia. Trabeculation is minimal.

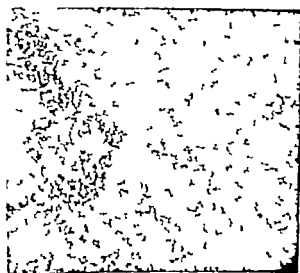


Figure 166 Microphotograph showing myxomatous tissue with areas of condensed nuclei and chondroid changes in chondromyxoid fibroma. No evidence of fibroid reaction present.

dysfunction average five months in duration prior to operation.

In the roentgenogram, there is a characteristic mottled cystic area of bone destruction, with expansion of the bone shell and usually some periosteal reaction (Fig. 167). Not infrequently the tumor bears a marked similarity to giant cell tumor. Microscopically

one sees masses of young cartilage cells of chondroblastic type with areas of uncalcified matrix. Zones of chondroid material are present with characteristic lace like calcification about residual lacunae. Some areas resemble adult hyaline cartilage. The tumor has areas of marked vascularity. About the vascular areas and the periphery of the cartilage, numerous giant cell osteoclasts are seen (Fig. 168).

Some of these tumors show malignant degeneration, differentiated from the benign lesions only by histopathologic study. Many investigators separate the benign tumors from the malignant variety, placing the latter with the chondrosarcoma group.

Radium or roentgen ray therapy is recommended in the benign lesions following exploration and curettage unless the tumor occurs in a bone which can be resected without sacrifice of function. The malignant variety of chondroblastoma has been uniformly fatal despite primary amputation.

Chondrosarcoma and Osteogenic Sarcoma. These include several subvarieties of malignant tumors which may arise either subperiosteally or centrally in the skeleton from the bone forming tissues and produce bone cartilage, osteoid material or preosseous connective tissue. These malignant tumors dis-

shaft regions of the long bones. It seems to be related to the formation of cancellous bone which normally follows in the wake of calcified cartilage.

Clinically the tumor is usually observed about one year after the beginning of symptoms, which are not characteristic. Many of the patients show a sequence of symptoms

including pain, tumor limp and fracture. The tumor has a wide age distribution being most frequent in young adults. It has an unusual tendency to involve the shaft of bones and to produce a pathologic fracture.

The roentgenograms may be hard to interpret and the findings are often confused with those due to benign bone cyst, giant cell tumor, metastatic carcinoma and Ewing's tumor. The distinguishing features in the x-ray film are the melting away and perforation of the bone shell at an early stage when the lesion is asymmetrically located, the presence of a periosteal reaction and a slight degree of cortical expansion. In advanced cases, osseous destruction leaves little doubt of malignant change in the bone (Fig. 171).

External irradiation is an effective form of palliative therapy but amputation only has permitted five year survivals without disease (10 per cent).

BENIGN LESIONS OF FIBRO-OSSEOUS ORIGIN

Osteoma and Ossifying Fibromas. These growths usually occur in children or young adults in the frontal or parietal bones of the skull in the bony walls of the frontal or maxillary sinuses and in the mandible. These tumors may be quite deforming and produce pain or pressure symptoms when they grow to large proportions.

The more rapidly growing osteomas are composed of cellular fibrous tissue in which small, round osteoid bodies are formed. The



Figure 170 Roentgenogram of sclerosing osteogenic sarcoma arising on the lateral aspect of the lower femur. The tumor mass is seen arising subperiosteally causing a dense extracortical shadow.



Figure 171 Roentgenogram of osteolytic osteogenic sarcoma involving the lower end of the femur with marked destruction of the medial cortex, rarefaction of regional bone shaft and pathologic fracture. There is moderate periosteal reaction.

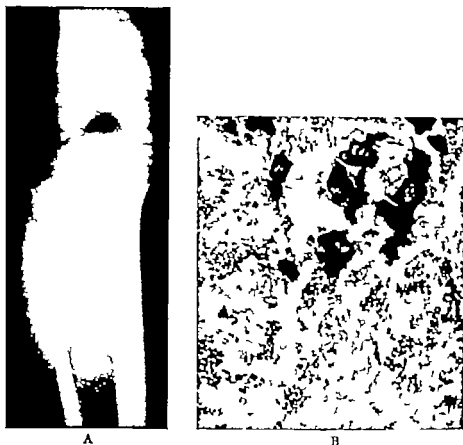


Figure 169 A, Roentgenogram of advanced primary chondrosarcoma of the upper tibia. Note the translucent character of the periosteal growth flecked with calcium and the destruction of the adjacent cortex. B, Photomicrograph showing clusters of small spindle cells (precartilaginous connective tissue) fetal cartilage cells and irregular calcification in a hyaline matrix typical of primary chondrosarcoma.

ribs, femur and heel. There may be a history of rheumatic pain for years with subsequent evidence of growth potential followed by pain swelling or pressure phenomena.

In the x-ray picture this tumor is most easily diagnosed when a portion of the primary lesion still remains and when the superimposed malignant change appears as a fuzzy infiltrating periosteal shadow flecked with calcium. Destruction of the cortical bone follows occasionally with pathologic fracture.

The relative benignity of the lesion plays an important role in its cure, which is by means of resection or amputation. Irradiation may be of benefit as an adjunct to surgery. Thirty per cent of patients treated by radical surgery have passed the five year survival period free of disease.

OSSEOUS TUMORS

Osteoblastic Osteogenic Sarcoma. This is the most highly differentiated type of osteogenic sarcoma. Fibrous elements may predominate microscopically but usually proliferation of malignant osteoblasts and new

bone formation are seen, with rare islands of cartilage. This tumor is most frequent in persons between the ages of fifteen and twenty five years and is usually situated in either the lower end of the femur or the upper end of the tibia. Other locations are the upper humerus, ribs, vertebrae and pelvis. The disease runs a fairly acute course with pain, tumor and dysfunction, perhaps preceded by trauma. The average duration of symptoms is ten months.

The x ray picture is characterized by dense, radiating, new bone in the periosteal zone, giving a "sun-ray" appearance. Later the medullary cavity is obliterated by tumor bone with some secondary destruction of the cortex (Fig. 170).

Cell differentiation of the tumor process plays an important role in the prognosis.

Radical extirpation by amputation or resection is the procedure of choice. The lesion is not radiosensitive. Cures have been achieved in 21 per cent of the patients.

Osteolytic Osteogenic Sarcoma. This is a destructive tumor arising in the region of the marrow cavity near the ends or in the mid-

Parosteal Osteoma and Myositis Ossificans.

Parosteal osteoma is a rare lesion composed of ossifying fibrous tissue and sometimes cartilage or myxomatous tissue. The tumor grows at a slow rate, giving rise to symptoms of tenderness, pain and ultimate dysfunction. A palpable lump is felt when the symptoms are first manifest or shortly thereafter. The roentgenogram reveals an ossifying mass usually of considerable size, extending out into the soft parts with or without intimate connection to the neighboring bone (Fig. 174). A common site of this tumor is the region of the lower femur. Microscopically bony trabeculae embedded in fibrous tissue are seen. The fibroblasts, osteoblasts and giant cells may be intermingled in areas which suggest osteolytic sarcoma. Close observation of the model, however is necessary to determine whether the lesion is benign. In a number of instances, malignant changes in the tumor may supervene and require amputation to eradicate the disease. In those tumors which appear to be benign, radical excision should be carried out, followed by intensive x ray therapy. If renewed growth is seen amputation must be performed because of eventual malignant change in these tumors.

Circumscribed *myositis ossificans* may be divided into the traumatic and atraumatic forms arising about the thigh, arm, elbow neck and lumbar muscles. Occupation plays little or no role in the case histories studied. Injury is definitely an important factor in

60 per cent of the lesions. The age incidence varies between ten and seventy two years. The earliest physical sign after injury is the development of a soft, compressible mass which soon becomes indurated. Bone may be detected in the roentgenogram within three weeks after the onset of the tumor formation. Small dense areas of calcification soon assume large proportions, forming a large spicule of bone, usually separated from the shaft of the neighboring bone (Fig. 176). Many lesions gain their maximum growth in six weeks or more and then spontaneously regress or become quiescent.

Histologically degeneration of muscle, hyperplasia of connective tissue and organization of hemorrhage are early findings while osteoid tissue with marrow spaces, osteoblasts about spicules of bone, occasional islands of cartilage (30 per cent of cases) and benign myxomatous tissue represent fully developed *myositis ossificans*. Malignant changes may complicate *myositis ossificans*, but these are exceedingly rare. Occasionally the disease is confused with parosteal osteoma.

Myositis ossificans progressiva a special form of the malady usually begins in childhood. It affects the spinal muscles primarily. As the name implies, it eventually involves the muscles of the entire skeletal system and is ultimately fatal.



Figure 174. Roentgenogram of a parosteal osteoma which reveals an ossifying mass extending out into the soft parts. Note the thickened cortex of the femur in the area.



Figure 175. Roentgenogram of a bone cyst in the shaft of the humerus. Note the central location with slight expansion of cortex. Trabeculations present suggest giant cell tissue activity.



Figure 172. Roentgenogram of an osteoid osteoma in the neck of the femur situated in the inferior intracortical zone. Note the discrete rarefied area with extensive sclerosis about it.

more slowly growing tumors form spongy bone and sometimes ultimately give rise to compact bone, frequently referred to as an eburnated osteoma. Roentgenographically they show a dense mass of new bone, smoothly outlined, with a sharply demarcated base formed by the thickened, slightly depressed inner table of the skull. When the tumor is present in the mandible, the roentgenogram reveals a radiolucence of regular contour projecting from the normal bony structure. The new bone is less dense than the normal bone. In the region of the antrum, the affected side shows increased density or cloudiness. In most instances, the tumor arises

outside the antrum and encroaches on the antral cavity or pushes downward toward the alveolar border displacing the teeth downward. These lesions are often complicated by trauma or infection.

Osteoma and ossifying fibroma are benign tumors. Simple excision usually suffices to eradicate the disease.

Osteoid osteoma is a small, rarefying lesion, situated in a dense overgrowth of osteoblastic activity. The lesions are frequently found in the bones of the hands, feet, vertebrae and shafts of the long bones. The patients are usually young adults or adolescents. Localized pain is often the primary complaint and may be associated with tenderness without fever or leukocytosis. Individual lesions may persist for a period of months to one or more years before remedial action is taken. In the roentgenogram, a dense, sclerosing, opaque zone about a small oval or rounded area of rarefaction is seen (Figs 172 and 173). The tumor may occur either as an intracortical lesion or in the cancellous portions of the bone.

The two principal theories on the nature of osteoid osteoma at this time are that the lesion is a primary neoplastic process of bone and that the process is inflammatory in origin. Another unverified interpretation holds that osteoid osteoma represents a healing area about a bone infarction of minute size. Local excision is adequate treatment for these lesions.



Figure 173. Low-power photomicrograph of an osteoid osteoma showing the central lesion composed of vascular fibrous tissue with proliferating fibroblasts in which trabeculae of new bone are found. The bone trabeculae about the lesion show increased thickening and proliferation.

A typical bone cyst discovered in the shaft of a long bone of a child as a result of pathologic fracture may be treated by simple splinting or surgical curettage and use of bone chips. Recurrence may take place following irradiation or curettage. If the lesion is explored, the lining should be stripped, the shell of bone crushed and the cavity obliterated with bone chips. The giant cell variant of the bone cyst responds well to deep x-ray therapy in moderate dosage. Latent bone cysts in adults usually require no treatment.

Diffuse Osteitis Fibrosa. While the term "osteitis fibrosa" is used loosely to refer to all types of cysts of the bone, there is now a tendency to restrict this term to a diffuse disease of the skeleton accompanied by adenoma or hyperfunction of the parathyroid glands. In such cases the fundamental disturbance is a supersaturation of the blood serum with calcium, increased calcium excretion and a generalized demineralization of the bones. The blood calcium level may be elevated to from 13 to 20 mg. and the phosphorus lowered to 1 to 1.5 mg. per 100 cc. In isolated areas, the rarefied bones show localized cyst formation and, occasionally, giant cell tumors. In the x-ray examination, the diagnosis depends on evidences of diffuse osteoporosis, bending deformities and glob-

ular areas of cysts or tumor formation (Fig. 178).

In the diagnosis of this diffuse skeletal disease, determinations of the blood calcium and phosphorus values and also of the blood alkaline phosphatase value, which is elevated, estimations of the urinary excretion of calcium and demonstration of parathormone in the blood stream by the Hamilton rabbit test are important. If proper laboratory tests are carried out confusion of this condition with metastatic carcinoma or multiple myeloma should not occur.

Treatment is directed at reduction of the parathyroid hormone output and recalcification of the skeleton. The parathyroids should be explored and the adenoma surgically removed. The patient should be placed on a high calcium and phosphorus diet, plus vitamin D.

Fibrous Dysplasia. There are two forms of fibrous dysplasia, the diffuse or polyostotic form, and the monostotic form.

In the diffuse or multiple form of fibrous dysplasia, the disease involves multiple bones in young individuals on one or both sides of the body. Albright and his coworkers have emphasized the extraskeletal features of the disease, which include pigmentation of the skin and endocrine dysfunction in girls in the form of sexual precocity and hyperthy-



Figure 178. Roentgenograms of bone lesions in the lower femur, lower humerus, radius, ulna and bones of the hand associated with hyperparathyroidism. Note the rarefaction of the bones due to depletion of calcium and phosphorus. The metacarpal bones and phalanges show cystic destruction. There is evidence of expansion of the bone shell in some of the metacarpal bones.



Figure 176 Roentgenogram depicting myositis ossificans. Large calcified shadow noted paralleling cortex and separated from it when viewed stereoscopically

A conservative form of treatment is desirable in the majority of patients with circumscribed myositis ossificans. Postoperative recurrences are frequent if surgical removal is employed too early (under six months) or if excision has been inadequate.

There are isolated patients who have had successful regression of recurrence following therapeutic dosages of x-rays. Insufficient experience with this agent in myositis ossificans prevents any final evaluation of the results.

RESORPTIVE TUMORS

Benign Bone Cyst. In children under fifteen years of age, a benign bone cyst occurring in the upper metaphysis of the humerus, femur or tibia is one of the commonest tumors of bone. Frequently pathologic fracture is the first indication of the presence of the disease. Mild pain or dysfunction, running a chronic course over a period of two or more years, may be elicited in taking a careful history. When roentgenograms are made, a central defect appears, surrounded by an intact and symmetrically expanded cortex. The cavity in the bone is traversed by irregular trabeculae of dense fibrous tissue (Fig. 175). When a pathologic fracture is



Figure 177 Roentgenogram of a bone cyst on the shaft side of the epiphyseal line in the upper end of the humerus. Note the trabeculated cystic area with expansion of bone shell and fracture through the thinned cortex.

present, signs of reossification are seen. Exploration discloses a cyst containing fluid, enclosed by a wall of osseous and fibrous tissue. Under the microscope, the proliferation of connective tissue and new bone formation suggest a healing reaction. Smaller cavities, when present, contain the remains of old hemorrhage and a sprinkling of large giant cells. Cysts with such multilocular cavities are termed "polycystic osteitis fibrosa," a rare subvariety.

Cysts immediately adjacent to the epiphyseal line on the shaft side (Fig. 177) which have been present for less than a year show an even greater amount of vascularity and more numerous giant cells than are seen in polycystic osteitis fibrosa. These giant cell variants of bone cysts are to be distinguished from true giant cell tumor by their occurrence on the shaft side of the epiphysis in patients under twenty years of age. The prognosis is far better than in a case of typical giant cell tumor and the patient will respond more readily to curettage or deep x-ray therapy.

Arrest in the growth of a bone cyst without complete healing and obliteration of the cavity is accountable for the latent bone cyst seen in adults. These latent cysts are symptomless and are usually found accidentally in a routine x-ray examination.



Figure 180. Roentgenogram of a benign giant cell tumor of the lower end of the radius. Note the asymmetrical position of the lesion in the epiphyseal zone. Trabeculae are seen within the cystic area and the bone shell is perforated.

irradiation and the occasional contractures about the joints, have brought back into favor treatment by curettage, advocated by Bloodgood. Recurrent giant cell tumors in the ulna, fibula or radius should be resected.

Extraskeletal Giant Cell Tumors. A lesion similar pathologically to the benign giant cell tumor which occurs about deciduous teeth on the gums is known as giant cell epulis. Tumors of this type are seen in children or in young adults and are related to the normal loosening of the roots of the milk teeth by odontoclasts. Their treatment is similar to that just described for giant cell tumor namely curettage and cauterization.

TUMORS OF NONOSSEOUS ORIGIN TUMORS OF MARROW AND HAVERSIAN SYSTEMS

Ewing's Sarcoma (Endothelial Myeloma)
This malignant tumor which arises intracortically or subperiosteally occurs in the first two decades of life: it involves the metaphysis primarily most often affecting the long pipe bones, especially the tibia and femur. The disease never involves the epiphysis primarily. It shows the usual symptoms of pain and tumor followed by dysfunction.

In the roentgenogram a widening of the shaft, with increase in cortical structure and onion-peel periosteal reaction, is an early finding followed later by varying degrees of

bone destruction (Fig. 181). Microscopically the tumor is composed of small round cells with dense nuclei and scanty cytoplasm simulating a lymphosarcoma (Fig. 182).

Irradiation provides a good therapeutic test and the best available palliative therapy. Irradiation alone, however is not sufficient to control the disease in the majority of cases.

A review of our patients the Bone Sarcoma Registry cases and the cases of Coley leads to the conclusion that combined preoperative irradiation (Coutard method) in full therapeutic dosage, followed by amputation or radical resection gives the best results (9 per cent five-year survivals).

Primary Reticulum Sarcoma of Bone. In our opinion, tumors described under this category are controversial as to origin. For the sake of completeness however a description of the process is included as distinct from Ewing's sarcoma, although we believe it to be an atypical form of the tumor first described by Ewing. Parker and Jackson first described primary reticulum sarcoma of bone as a separate entity.

Reticulum cell sarcoma of bone arises in both the long and flat bones but is commonly found involving the metaphyseal portion of a long bone. Fifty per cent of the patients are under the age of forty years though the disease is found in patients up to the eighth



Figure 179 Roentgenogram of monostotic fibrous dysplasia showing extensive bowing of the tibia and thinning and expansion of the cortex. Note the polycystic rarefaction traversed by many trabeculae.

roidism. These manifestations are not present in all patients. On roentgenographic examination, the bones are seen to be bowed and rarefied, with thinning and expansion of the cortex (Fig. 179). Pathologic fractures occur. The pathologic changes show the interior of the bone to be replaced by fibrous tissue of a gray or yellowish color which may contain islands of calcification, cartilage, ossified bone or small cysts.

In monostotic fibrous dysplasia, the first evidence of the disease is usually a local swelling with occasional tenderness or pain of an arthritic character. Pathologic fracture may be the first sign of the disease. The lesion appears either to grow slowly or to remain stationary. Cutaneous and endocrine abnormalities are lacking. The flat as well as the long bones may be affected. The roentgenogram shows areas of radiolucency and/or expansion of bone shaft, sometimes traversed by delicate trabeculae of bone in the long bones. These changes may be centrally or eccentrically located, especially in the metaphyseal area of the long bones.

Fibrous dysplasia is frequently recognized at the beginning of the second decade, though most often it comes under observation in the third decade. It appears to be a chronic disease, persistent throughout life.

All fibrous dysplasias are apparently true

dystrophies in which the tendency to normal ossification is prevented by a fundamental local disturbance yet to be demonstrated.

Surgical excision is justified in lesions which give rise to symptoms or interfere with functional activity. Resection or thorough curettage should be performed, incomplete removal leads to recrudescence of the process. Irradiation is given without benefit.

Giant Cell Tumor Adults, usually between twenty and thirty years of age, may have a tumor of bone which produces a globular area of rarefaction in the epiphysis of the lower femur upper tibia or lower radius. There is a history of trauma, pain, swelling and occasionally pathologic fracture—the typical clinical picture of benign giant cell tumor of the long bones. In the x-ray picture, the tumor produces a more or less circular defect, asymmetrically situated in the epiphysis, overlaid by a thin shell of bone, which is usually perforated at one or more points (Fig. 180). If the tumor persists without treatment for a period of more than fourteen months after the onset of symptoms, extension into the soft parts is the rule. Under a microscope many large multinucleated cells are seen embedded in highly vascular stroma with small round and spindle cells. Histogenetically the tumor is related to the resorption of calcified cartilage by giant cells, which occurs as a step in normal growth of bone in the region of the epiphysis until late in life. Healed giant cell tumors, following irradiation, show the formation of a typical bone cyst. This and other pathologic considerations indicate that the bone cyst and giant cell tumor are closely related lesions; the two may coexist in von Recklinghausen's disease of the bones.

In the first two decades of the present century the standard treatment for giant cell tumor was curettage followed by chemical (50 per cent zinc chloride) or thermal cauterization. Satisfactory results were obtained in about 70 per cent of the patients. About a decade ago irradiation was introduced as a method of treatment. Fractionated deep x ray therapy results in satisfactory reossification in many instances. This treatment does not afford microscopic confirmation of the diagnosis and sarcoma may masquerade under the diagnosis of giant cell tumor. Moreover repeated courses of irradiation over a period of one or more years may result in malignant transformation with the development of osteogenic sarcoma at the tumor site. Such errors in diagnosis, coupled with the long period required to

results with—

cat is from 65 to 70 per cent of the subjects. The plasma proteins may be markedly increased.

Roentgen ray examination of the tumors reveals areas of bone destruction which appear as multiple punched-out defects varying in size from that of a pea to that of an orange (Figs. 183 and 184). Pathologic fracture occurs in 62 per cent of the patients, a rib being most often affected. Microscopic study shows that the tumors are composed of plasma-like cells each with an eccentric nucleus containing a spoke-like arrangement

of chromatin. There is a very scanty amount of stroma.

Prognosis is uniformly unfavorable, though some patients have lived as long as seven years after the diagnosis was made. The prognosis seems to be little influenced by treatment although roentgen ray or radium therapy may bring about remissions. The average duration of life is three years after the patient comes under observation. Among palliative measures other than irradiation is the administration of stilbamidine and Pentamidine in conjunction with a diet low in animal protein. More recently urethane given in protracted dosages has shown promise in inhibiting the progress of the disease. In a few patients radioactive phosphorus has relieved pain and retarded the disease for a limited time. Deep roentgen ray therapy is the most valuable form of treatment in bringing about symptomatic improvement.

Chloroma and the Leukemias. Chloroma is a rare form of bone disease usually involving the skull vertebrae, spine, sternum, pelvis and long bones and associated with leukemia. There is a special tendency to infiltration of muscles and tendinous attachments near the affected bones. The disease usually occurs about the time of puberty. It frequently involves the orbital structures causing exophthalmos. Enlargement of the lymph nodes, liver and spleen is also found. Roentgen ray examination reveals areas of bone destruction or areas of rarefaction. Occasionally a periosteal reaction may be seen. The tumor tissue is definitely green in color and on section shows an increase in fibrous stroma with a crowding of large atypical monocytes everywhere. The disease seems to be a form of myeloid leukemia in which bone manifestations are a prominent feature. The process is radiosensitive and roentgen ray therapy is recommended for palliation.

Bone changes associated with *lymphatic* or *myeloid leukemia* are relatively rare. They frequently appear late in the disease. In *lymphoid leukemia* bone changes are found more often than in the myelogenous variety. Apparently there is no correlation between the early development of changes in the bone and the duration of life after such changes appear. When bone manifestations are prominent and appear early, the diagnosis is apt to be confused with that of multiple myeloma or chloroma. The total white count usually remains low in these patients, varying from 5000 to 12,000 cells per cc. of blood. In lymphoid leukemia, the long bones are more frequently affected than



Figure 183. Roentgenogram showing multiple punched-out areas in skull typical of multiple myeloma, at times these defects may reach several centimeters in size.



Figure 184. Anteroposterior view of skull involved by multiple myeloma seen in Figure 183.

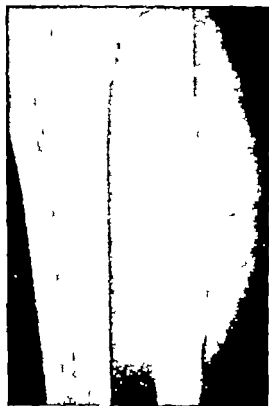


Figure 181. Roentgenogram of Ewing's tumor located in the fibula. There is widening of the shaft with increase in the cortical structure and onion-skin periosteal reaction.



Figure 182. Photomicrograph showing the fibrous stroma and small round cells resembling lymphocytes seen in typical Ewing's sarcoma.

decade. Pain, swelling and subsequent disability are the cardinal symptoms.

Roentgenographically the lesions appear to be extensive but without characteristic appearance. Bone destruction is the predominant feature, giving the bone a mottled and worm-eaten appearance with occasional new bone reaction. Periosteal reaction is variable. The cortex may be thickened in about 25 per cent of the lesions seen. Grossly extraosseous extension is often found in the metaphyseal region.

Microscopically the tumor is often difficult to differentiate from Ewing's sarcoma. Reticulum cell proliferation, demonstrated in the section, is considered crucial for making the diagnosis. Typical Ewing's tumor has proliferation of small round cells with no fibrillar intercellular material present. A difficult point in the diagnosis is posed by the morphologic similarity between reticulum cell sarcoma which begins in bone and reticulum cell sarcoma which begins elsewhere in the body but spreads to bone.

Primary reticulum cell sarcoma, appearing as single lesions in bone, has a five-year survival rate of 40 per cent when adequately treated by surgical resection or amputation. Irradiation has also been effective in

ablating the disease in a large number of cases. A clear-cut indication for the use of either radiation or surgery cannot be defined at this time, except in those instances in which resection or amputation cannot be performed.

Multiple Myeloma. This rare and fatal condition, as the name implies, produces extensive involvement of the skeleton. The systemic manifestations are equally widespread. In adults multiple foci of destruction are usually found limited in distribution to the red bone marrow. Rheumatic pains of increasing severity usually in the back or lower limbs discovery of a tumor by the patient sharp pains following exertion, fracture after injury and general weakness with anemia are the cardinal features marking the onset of the malady. As the disease progresses gradual collapse of the spine permits the lower ribs to sink to the pelvic brim, giving a characteristic habitus. The patient stands with protruding abdomen, flaring ribs, shoulders braced back and feet widely apart to assist in standing. The chin is thrust forward and may sink to the chest. Involvement of the spine and ribs is accompanied by radiculitis, paraplegia and emphysema of the lungs. Nephrosis with nonprotein nitrogen retention and low blood pressure, associated with an albuminous substance in the urine known as Bence Jones, is pres-



Figure 187 Roentgenogram of anterior rib involved by eosinophilic granuloma. Note the expanded and thinned cortex with bone resorption extending along the rib.

frequently observed. Mild leukocytosis and mild eosinophilia are usually present. Radiologically the lesions in the bones are eccentric. Osteolytic and irregular defects are observed, quite well circumscribed or punched out in character. The cortex may be expanded or perforated (Fig. 187). Varying degrees of periosteal reaction may be observed. Histologically the lesions show sheets of eosinophils with varying degrees of fibrosis and histiocytes interspersed through the lesion. Surgical excision or curettage promotes healing. Mild roentgen ray therapy also will control the disease. Spontaneous healing is rare.

METASTATIC DEPOSITS

Metastatic Carcinoma. Metastatic carcinoma of bone is usually seen in late adult life. It affects the bones in an extremely variable manner. About half of the lesions may occur as a single focus in the ends of the long bones, producing a central area of bone destruction, while the other half may involve the skeleton diffusely producing either osteolytic or osteoplastic changes. The source of the tumor varies but the most frequent sites of the primary growths are cancer of the breast, hypernephroma, cancer of the prostate, cancer of the thyroid and some location in the female genitals or gastrointestinal tract. Solitary areas of metastatic carcinoma to bone occur most frequently in the spine

and in the upper end of the humerus or femur and are extremely rare below the knee or elbow. They may also involve the trunk or the skull. A central area of bone destruction, as seen in the x ray film, rapidly increases and destroys the cortex outwardly from within, with little bone expansion. Multiple areas of skeletal involvement by metastatic tumors may also be osteolytic in character and central in location. Such lesions have a tendency to involve the pelvis and the spine, in addition to the upper ends of the humerus and femur (Fig. 188). Pathologic fracture is common. In the roentgenogram, metastases are visible as confluent and mottled areas of bone destruction in medullary locations. Osteoplastic involvement of the bones by skeletal metastases is most frequently a diffuse affair and, in addition to the absorption of bone always present, there is a laying down of irregular new bone of moderate density.

Roentgen ray therapy offers varying degrees of palliation in bone metastases. The disappointing results have stimulated investigators to seek other therapeutic measures. Testosterone and castration have proved beneficial in bone metastases from carcinoma of the female breast. Estrogenic therapy and orchietomy have given effective palliation to patients suffering with bone metastases from prostatic cancer and male breast cancer. Radioactive phosphorus, combined with



Figure 165. Roentgenogram of tibia and fibula showing rarefaction with slight periosteal reaction, associated with chronic lymphoid leukemia. Such changes are less common with myeloid leukemia.



Figure 166. Roentgenogram of upper tibia in a child with punched-out areas associated with Hand-Schüller-Christian disease.

are the pelvis, skull or vertebrae. The roentgenogram reveals rarefaction in the long bones with a slight periosteal reaction (Fig. 185). Occasionally the changes resemble those due to an early Ewing's sarcoma. In the skull, osteoporotic changes, diffusely scattered, with neighboring periosteal elevation or thinning, form the characteristic picture. Microscopically the disease resembles lymphosarcoma or Ewing's sarcoma of bone. The involvement of other organs however is diagnostic when considered with findings in the blood. Fractionated doses of roentgen rays over the affected bones and lymph glands are excellent palliative therapy.

Granulomatous Lesions of Bone. This group of bone lesions is often associated with disturbances in lipid metabolism. Organs connected with the reticuloendothelial system reveal a variety of changes.

There appears to be a close relationship between Letterer-Siwe's disease, Hand-Schüller-Christian disease and eosinophilic granuloma. Further it seems logical to assume that these diseases represent gradations of disturbance in lipid metabolism and in the reticuloendothelial system.

Letterer-Siwe's disease, a nonlipoid histiocytosis, is a rapidly fatal disease of infancy seldom seen in individuals older than the age of two years. The clinical features are

fever, skin rash, purpura, and rapidly developing and severe anemia. There is marked proliferation of cells in the reticuloendothelial system, namely the spleen, lymph nodes and also lymphoid tissue in the skin. Bone lesions are encountered, identical with those of Hand-Schüller-Christian disease. Patients seen at this age invariably die.

Hand-Schüller-Christian disease usually makes its appearance in late infancy and is rarely seen after childhood. The clinical manifestations are variable. Exophthalmos, diabetes insipidus, pigmentation of the skin and splenomegaly may be observed. Bone changes are a constant finding and roentgenographically appear as large, rarefied defects, clearly demarcated in the skull and in other flat bones. Diffuse cystic areas may also be seen in the long bones, with thinning of the cortex (Fig. 166). There is no periosteal reaction. The blood picture may show severe anemia. Transfusions are frequently indicated during the period of active roentgen ray therapy. In the x-ray treatments, small divided doses are best tolerated. Many of these patients survive for an indefinite time with relative quiescence of the disease as they grow older.

Eosinophilic granuloma occurs in children and young adults. It is a relatively benign lesion localized in the skeleton. It frequently appears first as a single lesion arising in the medullary cavity though often it may appear in multiple bones. A complete skeletal survey in all patients will probably reveal that it is a multiple lesion of the bones in most of the cases. The symptoms are unreliable though pain, swelling, and

rounded by varying degrees of increased density representing healing bone. Biopsies of the bone lesions show varying stages of disease activity. While irradiation is still the treatment of choice, other forms of palliation include the nitrogen mustard like compounds and radioactive phosphorus therapy. The disease has been found to be uniformly fatal following bone involvement.

Lymphosarcoma. Bone involvement in lymphosarcoma is rare. In a large series of patients having lymphosarcoma only 10 per cent were ultimately found to have involvement of the bone. Pain is an early symptom following its onset there is a variable interval before definite roentgenographic changes can be seen. Most of the patients die before the third year of the disease. Demonstrable changes in the bone are noted from twenty days to one year prior to death. As in Hodgkin's granuloma, the systemic manifestations of the disease are superimposed on changes brought about by osseous involvement such as localized pain, girdle pain and symptoms of cord compression. The bones involved, as in Hodgkin's granuloma, are those containing red marrow. Roentgenographically the lesions are either predominantly osteoplastic or osteolytic. As the disease progresses the entire bone is frequently involved. The osteolytic changes are seen more frequently than are the osteoplastic. The microscopic picture is characteristic of lymphosarcoma—a diffuse growth of lymphoid cells lying in reticular tissue. The treatment of lymphosarcoma consists in irradiation and other palliative forms of therapy which are used for Hodgkin's granuloma.

BONE INVOLVEMENT BY TUMORS OF MESODERMAL ORIGIN DUE TO INCLUSION OR DIRECT INVASION

Fibrosarcoma. Fibrosarcoma occasionally arises as a central medullary lesion but more often may be of periosteal (fascial sheath) or neurogenic origin. It may extend in two directions, both outside and beneath the periosteum. This form of sarcoma represents only about 1.5 per cent of all neoplasms of bones. The majority of central and periosteal fibrosarcomas are of a low degree of malignancy. The age of the patient is usually well over thirty years. Only two of the patients studied by us were children. The average duration of symptoms is about one year. The disease most frequently involves the femur and tibia, but other bones may be affected.

In the x-ray picture there is usually a large, soft-part shadow. The bone shows ero-

sion from without. The reaction of the bone is variable. The periosteum may show varying degrees of distorted ossification or no response at all. Bone destruction may be extensive. The ratio of soft part tumor to bone destruction is important (Figs. 189 and 190).

Microscopically the spindle cell varies from



Figure 189 Roentgenogram of the upper portion of the tibia showing marked destruction of the inner upper tibia with extensive soft part tumor (fibrosarcoma).



Figure 190 Microphotograph of fibrosarcoma involving the tibia, depicted in Figure 189. Marked proliferation of malignant fibroblasts with pleomorphism is noted.



Figure 168 Roentgenogram of metastatic carcinoma involving the upper femur and the fourth lumbar vertebra. Note the central destruction in the upper metaphyseal region of the femur and destruction and compression of the body of the fourth lumbar vertebra.

testosterone therapy also has been valuable in relieving bone pain in breast metastases.

Hodgkin's Granuloma. The bone marrow is more frequently involved in Hodgkin's granuloma than is usually supposed, the reported incidence ranging up to 40 per cent in patients with Hodgkin's granuloma having postmortem examinations. The duration of Hodgkin's granuloma before definite invasion of bone is quite variable, as is the duration of life following bone involvement in this disease. Some patients are living with bone changes which have existed from three to four years. In the patients who died, skeletal

changes were noted from two months to two years prior to death. Superimposed on the systemic manifestations of Hodgkin's granuloma may be the changes brought about by bone destruction, such as pain, rigidity of the spine and localized tenderness of the affected bones. The blood picture is quite variable ranging from leukopenia to marked leukocytosis. Marked secondary anemia is present in some patients. The bone changes as seen in roentgenograms are either osteoclastic or osteoplastic. Roentgenograms of the skull usually show a predominant osteolytic reaction. Rarefied areas in the bone are sur-

Surgical excision is the treatment of choice but recurrence is the rule.

Liposarcoma of Bone. Primary sarcomas thought to arise from fat in bone structure have been reported by Stewart, Geschickter and Copeland and also by Dawson. No unequivocal liposarcoma of bone however has been described. The tumors reported have their primary origin based on circumstantial evidence, such as the peculiar gross appearance with alveolar characteristics. They follow an unusual clinical course having a tendency to appear as multiple primary tumors and to metastasize to other bones. In treating liposarcoma of bone amputation rarely may prove curative. Irradiation of metastases is an effective palliative procedure.

Mysarcoma. This tumor is occasionally found invading bone and must be considered in the differential diagnosis of a variety of conditions under which neoplastic invasion of bone may occur.

TUMORS OF THE JOINTS AND BURSAE

Tumors of the joints correspond pathologically to those found in the tendon sheaths but are more rare. The following classification shows the variety of lesions which may be found.

- Osteochondroma or chondromatosis
- Cartilaginous cysts
- Synovial cysts and ganglia
- Giant cell tumors
- Xanthomatous giant cell tumors
- Lipomas
- Fibroma and fibrosarcoma
- Synovial sarcoma
- Hemangioma and lymphangioma

Chondromatosis of the joint synovium is usually found in adults between the ages of twenty and fifty years and is more frequently seen in males than females. The joints involved, in the order of frequency are the knee, elbow, ankle, hip and shoulder. The disease arises in the capsule and synovial membrane; it sometimes involves the neighboring bursae and tendon sheaths. The symptoms are usually mild and progressive and nonarticular in occurrence. Pearly bodies varying in number from a few to more than a hundred may be found free or attached to the synovial membrane. Various stages in the development of cartilage and bone are noted in the free bodies.

In the roentgenogram, the structures enclosed by the joint capsule show numerous spotted calcareous deposits while stippling may extend along the surface of the bones or along the neighboring tendons for some dis-

tance. The condition can be eradicated by removal of the loose bodies and excision of the synovial membrane or bursae containing the cartilaginous masses. Recurrences are seen but are infrequent.

Chondromatosis of the joint synovium occasionally gives rise to chondrosarcoma.

Cysts are occasionally seen in the articular cartilages of the knee. The patients are usually between fifteen and thirty years of age. The cysts are from 2 to 4 cm. in diameter and usually involve the external meniscus. A few have been found on the internal side, some involving both menisci. Rarely is the lesion bilateral. Limitation of motion, pain and atrophy of the thigh may occur. Microscopic examination shows that these cysts are due to mucoid regression in the joint cartilage. Treatment consists in excising the cyst and the involved meniscus.

Cysts in the synovial membrane within the joint are similar to those found in tendon sheaths. They may recur after excision and some are true neoplasms. One cystic tumor within the synovial membrane proved to be a lymphangioma and was connected with a bursa surrounding the joint. Baker's cyst is a posterior herniation of the synovium of the knee joint and often connects with the knee joint through a fibrous channel.

Synovial cysts are treated by surgical excision. Baker's cyst should be isolated by careful dissection and excised. The pedicle should be ligated if it connects with the joint cavity.

Excepting benign cartilaginous tumors of the synovium, the majority of benign synovial tumors are *giant cell xanthomas* and are characterized histopathologically by giant cell proliferation, accumulation of foam cells (xanthoma cells) and connective tissue derived from the synovial membrane. Some of these synovial tumors characteristically of the benign giant-cell xanthoma type, show a tendency to involve neighboring bone. Of nine patients studied with giant cell xanthoma and fibroxanthoma with giant cells about the joints, five patients had associated bone involvement.

Radical excision may be resorted to in the treatment of these tumors, but, if recurrence supervenes, a resection of the joint and affected soft parts usually suffices to eradicate the disease. When synovial tissue predominates in a tumor of the joint, the lesion is usually malignant.

Hypertrophic out pouchings of synovial tissue distended by fat are frequently observed in association with arthritic changes. True

the more aggressive oat cell type to the large fusiform spindle cell, with transition cell forms. Usually no bone or cartilage is seen in the fibrosarcomas. A definite percentage of the fascial sheath tumors are not sarcomas but cellular fibromas. This accounts in part for the unusual number of cures of so-called fibrosarcoma.

These tumors are not highly radiosensitive. The cell differentiation is all-important in this group. There is some evidence that irradiation inhibits tumor growth for a time, producing a slow sclerosing reaction in the fibrospindle-cell type of lesion. Patients with the fibrospindle-cell variety of tumor will survive five years or longer irrespective of the inadequacy of the treatment. Amputation or radical resection cures the majority of patients.

The undifferentiated oat cell type of fibrosarcoma is extremely aggressive and neither x-ray treatment, radium therapy nor local excision offers a permanent cure. Primary amputation when possible is the treatment of choice.

Malignant Neurilemmoma (Neurogenic Sarcoma) The nerve sheath sarcomas, invading bone, are differentiated microscopically by myxomatous structure intermingled with fibrous tissue. The nuclei are longer and deeper staining than are those of other sarcomas invading bone. Many tumor giant cells are seen.

Clinically and roentgenographically these neurogenic tumors bear a close resemblance to the fibrospindle-cell tumors, although more pronounced destruction of bone is noted in the neurogenic lesions. As in the fibrospindle-cell tumors there is a gradual transition among the neural tumors from the benign neurofibromas to the most malignant form of neurogenic sarcoma. The more malignant and cellular forms predominate the picture when bones are involved.

Neurogenic sarcomas as a group are highly radioresistant tumors. Interstitial irradiation by buried radon implants has been almost uniformly disappointing. A certain number of cases have shown some regression following external irradiation alone.

It may be stated, however, that clinical cures in neurogenic sarcoma are obtained only by surgical intervention. The best results are obtained by early amputation and, even with this radical form of treatment, permanent cures are not numerous.

Angioma of Bone. Angioma of bone is considered rare. The ages of the patients vary between four and forty years. The tumors observed have been located in the humerus,

ulna, radius, femur or calcia, skull and vertebrae. The vascular tissue obtained is characteristic of angiomatous tissue elsewhere.

On reviewing the literature, one gains the impression that the majority of the angiomas of bone are of the cavernous type. Many however show the capillary type of angioma and there are lesions with proliferative changes suggesting malignant disease.

Angiomas affecting bone are essentially benign. They show varying degrees of radiosensitivity and the younger the patient the more effective the irradiation. This is also characteristic of angiomas found in other parts of the body.

Isolated patients may be pointed out who have been cured by x-ray therapy alone or by surgical intervention.

Lymphangioma. Occasionally lymphangioma involving one or more bones with changes much like those seen in angioma is noted. In one patient, marked resorption of bone structure was seen in the ischium and ilium. The surrounding soft parts showed invasion by the lymphangiomatous process. Microscopically lymphangiomatous channels were noted, some of which were quite dilated. The lymphangiomatous tissue was associated with accumulations of lymphocytes and lymphoid tissue. Bone surrounding resorption areas revealed little evidence of osteoblastic activity. The treatment is largely limited to roentgen ray therapy.

Chordoma. Chordoma is a rare malignant neoplasm, found near the spheno-occipital or sacrococcygeal regions, thought to arise from remnants of the notochord. Rarely these tumors have been found at various levels of the spinal column. Young adults and children are usually the persons affected by the disease. The symptoms vary with the location. Intracranial symptoms or symptoms from spinal cord and nerve involvement may be observed. A tumor in the cervical region may produce pharyngeal symptoms. The chordoma grows slowly but kills by invasion of vital structures. Twenty seven per cent of the growths are said to metastasize. The roentgenogram usually shows destruction of bone associated with a soft part shadow. The microscopic picture is variable. Solid cords of polyhedral and globular cells, in some instances becoming more vacuolated and having a homogeneous intercellular mucinous matrix resembling hyaline cartilage are seen. Great difficulty is experienced in differentiating these tumors from atypical chondromas and mucoid carcinoma arising in the gastrointestinal tract.

Irradiation has little

an outer fibrous layer and an inner layer of synovial cells which often form pseudo villi. Fibrous tissue is constantly present throughout the tumor manifested either as dense sheets of malignant spindle cells (Fig. 193) fibrosarcoma-like in appearance or merely leading support to pseudoglandular tufts of synovial tissue forming the central core of the papillary projections. In instances where the tumor has a fibrosarcoma-like appearance, careful search is often needed to find the pseudoglandular clefts typical of synovial sarcoma. The more characteristic tumor consists of epithelial like cells upon supporting stroma about spaces resembling slits.

The tumor has a propensity for metastasizing to regional lymph nodes in about 15 per cent of patients. Vascular metastases occur in the majority of patients not successfully treated (pulmonary metastases 65 per cent bone metastases, 10 per cent). Simple excision followed by irradiation has been the treatment of choice until recently the prognostic outlook is very poor. Adequate surgical resection is the method of choice in the treatment of early synovial sarcoma. If the tumor is deep seated or extensive, amputation, where possible should be performed. The clinical stage of the disease determines the form of ablation. When regional lymph nodes are involved a radical lymph node dissection is recommended, with en bloc dissection of the primary lesion or with a regional amputation. Postoperative irradiation

is advisable when local resection has been carried out. Therapeutic survival rates in large series of studied cases vary but average between 22 per cent and 25 per cent.

SOMATIC SOFT PART TUMORS OF THE EXTREMITIES

The soft part tumors of the extremities may be divided into a broad classification of benign and malignant fibromatous lipomatous angiomatous myomatous neuromatous and synovial tumors.

The majority of soft part sarcomas originate as such. The benign neoplasms rarely become malignant.

Malignant soft part tumors in the order of their frequency include sarcomas of undetermined origin (36 per cent) liposarcoma (15 per cent) rhabdomyosarcoma (14 per cent) synovial sarcoma (9 per cent) Kaposi's sarcoma (7 per cent) neurogenic sarcoma (6 per cent) fibrosarcoma (5 per cent) and others (8 per cent).

Sarcomas of undetermined origin account for approximately 36 per cent of all malignant tumors of soft somatic tissues. The rapid and abundant growth of fibrous tissue frequently obscures the primary origin of a tumor. Poor differentiation of anaplastic tumors organoid origin, with numerous elements of a given organ present, origin from primitive mesenchyme—these among other factors, prevent accurate histologic classification.

Liposarcomas constitute about 15 per cent of all primary malignant tumors of the soft somatic tissues in which exact histogenic definition is available. They may occur wherever fat is present. The thighs buttocks and shoulder girdles are sites of greatest frequency. About 13 per cent of cases involve the retroperitoneum and trunk. Rarely is liposarcoma found arising from a pre-existing lipoma. The pseudoencapsulation of liposarcoma, when found, usually reflects a relatively benign course for the tumor. The primary disease is often located deep in the intermuscular zones or in the areas about the joints. Recurrent liposarcomas, following incomplete removal, are more likely to show invasive characteristics. Histopathologically the tumors may show (1) well-differentiated fat cells associated with varying numbers of embryonal stellate cells (2) embryonal stellate cells or spindle-shaped lipoblasts with many bizarre nuclei, or (3) a round cell type of morphology the cells having a central nucleus with abundant foamy cytoplasm.

Liposarcoma in the peripheral tissues of



Figure 193. Microphotograph depicting synovial sarcoma with marked spindle cell proliferation but with typical epithelial-like cells producing pseudo-glandular formations.



Figure 191 Gross specimen showing synovial sarcoma involving knee joint and invading the femoral condyles and metaphysis.



Figure 192 Roentgenogram of same amputated specimen seen in Figure 191 showing invasion of the femur with marked soft part tumor proliferation along the bone shaft.

articular lipomas are uncommon however. Isolated instances of the occurrence of lipomas about the knee joint in adipose women have been noted. The tumors are accompanied by fluid in the joint, which is relieved by excision of the growth.

Hemangiomas and *lymphangiomas* are of rare occurrence and are usually benign. They are composed of a diffuse mass of blood vessels held together by loose connective tissue or there may be a mixture of vessels of both vascular and lymphatic type. Cystic changes may occur within the synovium. Such tumors create a considerable hazard during surgical extirpation. Surgical excision has proved successful in the majority of cases but irradiation may be utilized if surgical resection fails.

Fibroma and *fibrosarcoma* are rarely found about the joint, in our experience, only one instance of fibroma has been noted and in one patient fibrosarcoma developed about a joint. Individual case reports may be found in the literature. Fibrosarcomas which involve the synovial membrane also have invaded the neighboring periarticular structure and may well be regarded as cellular synovial sarcomas.

Synovial sarcomas (*synoviomas*) arise from synovial tissue and occur with the greatest frequency in the lower extremities (80 per cent). The joint synovia are rarely involved (Fig. 191). The tumors often occur

in bursae about the knee and ankle joints in addition to arising from synovial tissues about the foot and hand. Synovial sarcoma is essentially a disease of younger adults. No specific etiologic factor is known. There is no characteristic clinical syndrome. A mass with or without pain or pain without a palpable tumor may be the patient's chief complaint.

The tumor grossly may be encapsulated or may show varying degrees of aggressive infiltration into surrounding structures. The tumor often produces distortion of bones from pressure or there may be marked invasion of the osseous structure (Fig. 192). Pseudoencapsulation does not represent localization of the disease. More obvious tumor infiltration is apparent in the majority of cases. The cut surface of the tumor appears grossly firm, grayish pink, with areas of hemorrhage. Small or large cystic cavities may be present filled with gelatinous fluid of variable color. Occasionally one may find foci of yellow xanthomatous like tissue and also small areas of calcification.

Histopathologic study is influenced by the two layers observed in the tissue of origin,

presence of fibrils with cross striations (Fig. 195) (2) the presence of giant cells (3) the presence of spindle cells and (4) pleomorphism of the tumor cells. Special stains are often helpful in distinguishing either the cross-striations or the longitudinal myofibrils which are important from the diagnostic viewpoint.

As with liposarcoma the choice between radical surgical dissection and amputation depends on the regional location, fixation of tumor to surrounding tissues, whether the lesion is primary or recrudescence the presence of regional or distant metastases and the experience of the surgeon. Wide radical excision is indicated in localized lesions. It is strongly recommended that a complete muscle group be resected when the tumor involves one or more of the components, such as the adductor muscles of the thigh. While prognosis is poor recrudescence of the disease is amenable to operative cure as are primary lesions. Five-year survival rates without disease vary widely. Pack has a remarkably successful five year "cure" rate of 33.8 per cent (twenty-two of sixty five cases). A great number of the patients living five years had had previous inadequate excision of the tumors, followed by adequate surgical treatment. Operable untreated lesions were relatively rare in this series. An increasing percentage of five-year survivals should follow adequate treatment of early lesions. Postoperative irradiation is not ef-

fective in altering the course of the disease although in isolated instances preoperative irradiation has reduced a primary tumor to the status of successful operability.

Synovial sarcomas represent about 9 per cent of definable soft part sarcomas. Since they have already been discussed they are included here only to complete the classification.

Kaposi's sarcomas are atypical sarcomas manifest in the skin of extremities of patients over age forty years (70 per cent). The disease may be found in the viscera and lymph nodes before classic skin lesions are discovered. The initial dermal lesion is a red dened well-demarcated nodule. Progression of disease includes change in color (bluish red) multiplicity of nodules involving symmetrical areas of both extremities edema and ulceration ultimately with bleeding. Death generally results from intercurrent infection, progressive weakness or hemorrhage from the cutaneous gastrointestinal or lung lesions. Kaposi's sarcoma appears to be a dysplasia of the reticuloendothelial system.

Wide radical excision of the early lesion is indicated and results in a few five year survivals. If the disease is extensive conservative irradiation yields reasonable palliation. Overreatment with x ray should be avoided to prevent massive necrosis and gangrene.

Neurogenic sarcomas (malignant neurilemmomas) have for their site of predilection the extremities. The nerve trunks anatomically demonstrated to be associated with malignant neurilemmomas are, in their order of frequency median, peroneal, sciatic (Fig. 196) ulnar and femoral. There are no clinical symptoms characteristic of the disease. The histologic features of the tumor influence prognosis. Tumors arising in major nerve trunks are clinically more malignant than peripherally situated tumors. The best results in treating malignant neurilemmomas are obtained by early amputation and, occasionally by hemipelvectomy. Five-year survivals without disease are few and do not necessarily reflect the complete control of the disease. Recrudescence has been noted as late as eight years after apparent control of the primary lesion.

Fibrosarcomas comprise about 5 per cent of malignant tumors of the soft somatic tissues. They occur most frequently in the extremities (80 per cent). The peak of incidence is noted in patients about the third and fourth decades of life.

Fibrosarcomas develop without obvious relation to any etiologic factor save, occasionally previously damaged tissue. Satellite le-

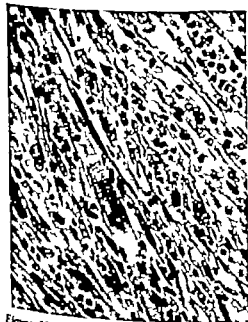


Figure 195. Microphotograph showing morphologic characteristics of rhabdomyosarcoma. Note the striations with cross-striations, the presence of giant cells (microphotograph courtesy of Dr. Albert Cannon, Assistant Professor of Pathology Georgetown University School of Medicine, Washington, D. C.)



Figure 194 Specimen of inner right thigh and pelvis following hemipelvectomy for rhabdomyosarcoma of adductor muscles. Note the dense infiltrating tumor mass in the region of the adductor muscles

the body usually begins as an inconspicuous swelling, growing gradually larger. Pressure symptoms ultimately supervene. Pain is usually not an early symptom. Early recognition is made difficult by the similarity between the aggressive tumor and its benign counterpart.

The best prognosis is found in patients with superficial tumors which are radically excised or in those whose tumors are amputated. Patients having deep-seated or incompletely excised tumors have a poor outlook.

The choice between radical surgical excision and amputation depends on a variety of factors such as (1) the surgeon's experience, (2) the degree of malignant change, (3) location of tumor, (4) whether the tumor is primary or recrudescant and (5) the presence of regional or distant metastases. Irradiation is considered an important postoperative adjunct in treating recrudescant tumors and in the case of distant metastases. Pack and Ariel report 35 per cent of patients with liposarcoma are living five years without disease following adequate surgical removal and with postoperative x-ray therapy in selected cases.

Rhabdomyosarcomas are infrequent when the relative amount of skeletal muscle tissue making up the body soft parts is considered. About 14 per cent of histogenically definable soft-part sarcomas are from striated muscles. They occur at all ages but have a peak

incidence between forty and seventy years of age. They are most often located about the lower extremity including the buttocks and groin (54 per cent) and the upper extremity including the shoulder (30 per cent). The individual muscles most often affected are the quadriceps femoris, the adductor muscles of the thigh, the muscles of the lower leg, the deltoid, triceps, biceps and brachialis muscles of the arm. Other muscle sites are less common (Fig. 194).

Pain is an infrequent complaint. The tumor is usually first noticed as a painless localized swelling which often reaches a considerable size before medical advice is sought. Interference with function is a late manifestation. On examination the mobility of the tumor disappears when the involved muscle contracts. The tumor may occasionally infiltrate the skin and produce necrosis.

The gross appearance of rhabdomyosarcoma is variable. The tumor may appear multilobulated or multicentric; it may appear as a discrete nodule or as a diffuse widespread process. The tumor is also variable as to its palpable density. A highly vascular tumor may be soft and compressible whereas an increase in the connective tissue components gives a sense of firm induration. Hemorrhagic necrosis may be massive.

Many of these tumors go undiagnosed because of the anaplastic or atypical morphology which they display. The diagnostic morphologic characteristics (1) the

Amputations and Artificial Limbs

By CHARLES O BECHTOL M D

CHARLES ORVILLE BECHTOL born in Chicago was educated at Stanford University and its medical school. Trained in orthopedic surgery he served on the faculty of the University of California and then became chief of the Division of Orthopedic Surgery at the Yale Medical School. He has now returned to the University of California, Los Angeles, as Professor of Orthopedic Surgery and chairman of that division in the medical school.

The reason for amputation is commonly stated as being to save life or to improve function. With improved types of prostheses developed in recent years the great improvement in function which can be offered to the amputee places an added burden on the surgeon to perform amputation with the greatest possible skill. This means that the surgeon must know the present concept of the sites of election for amputation, the amputation technique to be used under varying conditions and the great importance of proper postoperative care. The amputee clinic team must be utilized in prescribing the proper prosthesis to fit the amputee's particular requirement and to institute and guide his training in the use of the prosthesis.

AMPUTATION LEVELS

Amputation levels previously unacceptable are now considered to be desirable because of improvements in prostheses which allow their use. In the upper extremity (Fig. 197) all possible lengths should be saved down to and including the level of wrist disarticulation. The carpal bones should be saved only if the amputee will be doing some sort of labor and may not be wearing a prosthesis a good part of the time. Beyond this level, through the hand, all possible lengths should be saved which are compatible with good amputation technique. Because of the great importance of sensation, prosthetic replacement of portions of the hand is usually not successful except for cosmetic purposes.

In the lower extremity (Fig. 198) less

change in the classical sites of election has occurred. Disarticulations through the knee joint, although leaving a satisfactory stump still do not have an entirely satisfactory prosthesis available. Very short below-knee stumps give trouble in fitting and below knee stumps longer than 7 or 8 inches may produce circulatory difficulties or difficulties in the alignment of the prosthesis. The area of partial foot amputations has been greatly improved by the addition of fusion of the ankle in 5 or 10 degrees of dorsiflexion, thus avoiding the fixed equinus position so frequently seen with this type of amputation.

SURGICAL PRINCIPLES OF AMPUTATIONS

The surgical principles of amputation are similar at any level. Varying conditions indicate three general types of amputation.

Open or Guilloine Amputation. This amputation is recommended in the presence of already established severe infection or in a wound so severely contaminated that infection seems inevitable particularly if adequate hospital care will not immediately be available for the patient.

The various layers of the extremity are divided by successive circular incisions each layer being retracted so that the next incision is made at a slightly higher level. The entire end of the stump is in the shape of an inverted cone with the apex pointed proximally.

There are two essentials in the postoperative care of this type of amputation. First is a dressing over the open end of the wound

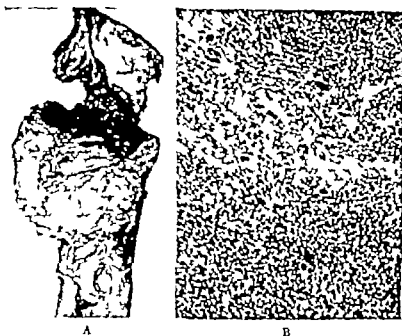


Figure 196 A, Gross specimen following hemipelvectomy for malignant neurilemoma of the sciatic nerve sheath. Note the bulky tumor in the thigh below the sciatic notch. B Microphotograph of malignant neurilemoma showing anaplastic tumor proliferation with concentration of nuclei. Malignant giant cells are frequent.

sions ultimately occur with growth of the tumor in many instances. There are no symptoms characteristic of the disease. The diagnosis must depend upon surgical biopsy though soft part roentgenograms may direct the surgeon to a more exact location for biopsy. Pseudoencapsulation is often noted at surgery lending false security to the operator. Neighboring bone muscle, skin and occasionally the synovium of joints may be invaded. On cut section, the tumors are firm and grayish pink. Areas of hemorrhage with cystic changes and bone and cartilage may be present occasionally.

The histopathologic findings vary from well-differentiated, relatively benign forms of fibrosarcoma to a high degree of anaplastic malignant transformation. In the latter case stroma becomes less evident with packing of the cells. There is irregularity in shape and size of the cells with evidence of hyperchromatism. Mitoses and giant cell formation may be observed.

Proper treatment of primary fibrosarcoma consists of radical extirpation of the disease with a wide normal tissue margin, or amputation of the extremity as the logical alternative. The prognosis in fibrosarcoma is better than for other sarcomas of the soft somatic tissues. They are notorious however for recrudescence following initial surgical procedures. This high recurrence rate reflects the folly of incomplete removal. Indeed, the lesions may develop in-

creasingly malignant potential through surgical insults. Over 50 per cent of fibrosarcomas seen in our clinic are recrudescence ones. The relative five-year survival rate is approximately 60 per cent.

READING REFERENCES

- Ackerman, L. V. and del Regato, J. A.: *Cancer: Diagnosis, Treatment and Prognosis*, 2nd ed. St. Louis, C. V. Mosby Company 1954, pp. 1025-1065.
- Adams, R. D., Denny Brown, D., and Pearson, C. M.: *Diseases of Muscle*. New York, Paul B. Hoeber 1953, pp. 854-860.
- Albright, F. and Reifenstein, E. C., Jr.: *The Parathyroid Glands and Metabolic Bone Diseases*. Baltimore, Williams & Wilkins Company 1948.
- Coley, B. L.: *Neoplasms of Bone*. New York, Paul B. Hoeber 1949.
- Dahlin, D. C.: *Bone Tumors*. Springfield, Ill., Charles C. Thomas, Publisher 1957.
- Geschickter, C. F., and Copeland, M. M.: *Tumors of Bone*. Philadelphia, J. B. Lippincott Company 1949.
- Lichtenstein, L.: *Bone Tumors*. St. Louis, C. V. Mosby Company 1952.
- Lock, J. V.: *Bone and Joint Diseases*. Springfield, Ill., Charles C. Thomas, Publisher 1950.
- Pack, G. T., and Arlet, I. M.: *Tumors of the Soft Somatic Tissues*. New York, Paul B. Hoeber 1953.
- Pugh, D. G.: *Roentgenologic Diagnosis of Diseases of Bones*. New York, Thomas Nelson & Sons, 1950, 1951.
- Stout, A. P.: *Tumors of the Soft Tissues*, Atlas of Tumor Pathology Sec. II Fascicle 5. Washington, Armed Forces Institute of Pathology 1952.
- Weinmann, J. P., and Sicher, H.: *Bone and Bones*, 2nd ed. St. Louis, C. V. Mosby Company 1955.

Amputations and Artificial Limbs

By CHARLES O. BECHTOL, M.D.

CHARLES ORVILLE BECHTOL, born in Chicago was educated at Stanford University and its medical school. Trained in orthopedic surgery he served on the faculty of the University of California and then became chief of the Division of Orthopedic Surgery at the Yale Medical School. He has now returned to the University of California, Los Angeles as Professor of Orthopedic Surgery and chairman of that division in the medical school.

The reason for amputation is commonly stated as being to save life or to improve function. With improved types of prostheses developed in recent years the great improvement in function which can be offered to the amputee places an added burden on the surgeon to perform amputation with the greatest possible skill. This means that the surgeon must know the present concept of the sites of election for amputation, the amputation technique to be used under varying conditions and the great importance of proper postoperative care. The amputee clinic team must be utilized in prescribing the proper prosthesis to fit the amputee's particular requirement and to institute and guide his training in the use of the prosthesis.

AMPUTATION LEVELS

Amputation levels previously unacceptable are now considered to be desirable because of improvements in prostheses which allow their use. In the upper extremity (Fig. 197) all possible lengths should be saved down to and including the level of wrist disarticulation. The carpal bones should be saved only if the amputee will be doing some sort of labor and may not be wearing a prosthesis a good part of the time. Beyond this level, however, all possible lengths should be saved which are compatible with good amputation technique. Because of the great importance of sensation, prosthetic replacement of portions of the hand is usually not successful except for cosmetic purposes. In the lower extremity (Fig. 198) less

change in the classical sites of election has occurred. Disarticulations through the knee joint, although leaving a satisfactory stump still do not have an entirely satisfactory prosthesis available. Very short below-knee stumps give trouble in fitting and below-knee stumps longer than 7 or 8 inches may produce circulatory difficulties or difficulties in the alignment of the prosthesis. The area of partial foot amputations has been greatly improved by the addition of fusion of the ankle in 5 or 10 degrees of dorsiflexion, thus avoiding the fixed equinus position so frequently seen with this type of amputation.

SURGICAL PRINCIPLES OF AMPUTATIONS

The surgical principles of amputation are similar at any level. Varying conditions indicate three general types of amputation.

Open or Guillotine Amputation. This amputation is recommended in the presence of already established severe infection or in a wound so severely contaminated that infection seems inevitable particularly if adequate hospital care will not immediately be available for the patient.

The various layers of the extremity are divided by successive circular incisions, each layer being retracted so that the next incision is made at a slightly higher level. The entire end of the stump is in the shape of an inverted cone with the apex pointed proximally.

There are two essentials in the postoperative care of this type of amputation. First is a dressing over the open end of the wound

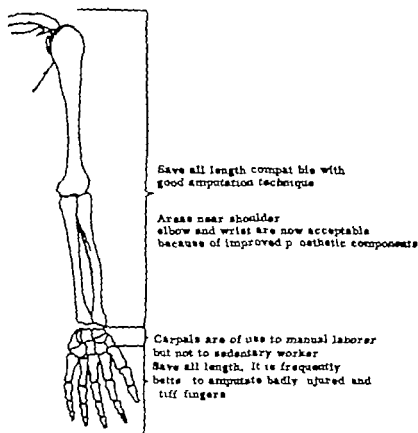


Figure 197. Amputation levels in the upper extremity

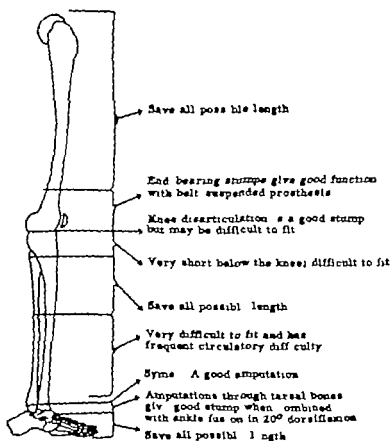


Figure 198. Amputation levels in the lower extremity

using petrolatum gauze or some similar substance with copious dressings to absorb the drainage. The second is the application of skin traction to prevent the elasticity of the skin from retracting the edges of the wound. After a granulating surface is established on the end of the stump this may be covered with split-thickness skin graft and when the danger of infection is considered to be passed the stump may be revised by fashioning flaps and achieving a proper closure.

There are two great pitfalls in this revision. The first is the attempt to save too much bone length, thus leading to inadequate covering of this bone with skin and soft tissue. The second is the failure to dissect out meticulously all of the scar tissue which has been laid down in the process of granulation of the wound, and particularly the failure to identify and dissect free from scar tissue all of the nerves of the stump. Neglect of the first precaution leads to a stump with a prominent bone at its end covered only by skin, subcutaneous tissue and muscles of the stump, which have not obtained attachment to this bone and therefore may move up and down a considerable distance with each step. This leads to abrasion of the skin inside the socket and a most uncomfortable stump. Failure to carry out adequate removal of the scar tissue at the end of the stump with dissection free of all nerves may lead to extremely painful stumps with a great amount of phantom pain.

Elective Flap Type of Amputation. This amputation can be carried out if the danger of infection is not great and if adequate hospital care is immediately available for the patient. Flaps are so fashioned as to leave the scar over the end of the stump in almost all cases. Advantages of bringing the scar anteriorly or posteriorly are usually outweighed by the extensive undermining of the skin necessary and may lead to subsequent breakdown of the skin because of poor circulation. In such an amputation the bone should be divided at such a level that muscle tissue can be closed over the bone end, using sutures in the fascia of the muscles since sutures in the muscle itself usually will not hold. Exceptions to this are cases in which no muscle tissue is present, such as disarticulations of the wrist, or the anterior flap in a tibial amputation where no muscle tissue is present. Redundant skin should not be left expecting traction at a later date. The skin should be sutured under moderate tension and this tension relieved in the early

postoperative period by the application of skin traction.

Amputations for Vascular Gangrene. Amputations in this category require an entirely different technique because here the aim is dictated by the desperate need to obtain healing in skin which has at least moderately impaired circulation and by the knowledge that this is merely a palliative procedure in a disease process which will progress. Flaps can be fashioned in the same manner as in elective amputations. Great care should be taken not to dissect the planes between the various layers of the soft tissue. Catching of bleeding points should include the minimum amount of tissue in the hemostat and the use of forceps or retractors on the skin should be avoided. Closure of the skin edges should be so loose that no traction will be necessary and no circumferential bandages should be used. In placement of the skin sutures the skin should be held in the fingers rather than in a forceps while the needle is driven through. During postoperative care, prevention of flexion contractures of the joints is of the greatest importance. This can be accomplished in the upper extremity by having the patient carry his joints through a full range of motion as soon as possible, and in the lower extremity by having the patient lie on his abdomen.

In vascular amputations the patient will frequently be found to have developed flexion contractures during the course of the gangrene and it is therefore important that such an amputee lie on his face during the preoperative treatment as well as in the postoperative period. Sutures should generally be removed in ten to twelve days and the stump wrapped with an elastic bandage to encourage shrinking except in cases in which circulation is precarious.

In nonvascular amputations fitting can usually be carried out somewhere between eight and sixteen weeks postoperatively and should not be delayed too long. During this period the patient should have active rehabilitation treatment with supervised exercises to the stump muscles stretching of contractures and active joint motion three times a week. Prescription of the prosthesis should follow consideration of the individual problems of the amputee and is best carried out by a team consisting of the physician, therapist and prosthetist, each of whom contributes his specialized knowledge. This same team should determine what type of therapy may be necessary before fitting and

the extent of training when the prosthesis is received. The team also carries out a check out procedure to insure that the prosthesis conforms to the prescription and fulfills satisfactory functional standards.

UPPER EXTREMITY FUNCTION AND PROSTHETIC REPLACEMENT

Man has been described as the animal who uses tools. The function of the upper extremity is largely the manipulation and transportation of tools. The function of the hand is to grasp these tools. This is accomplished in most cases between the tips of the thumb, index and middle fingers. The remainder of the arm acts as a crane and moves the hand about to its various positions. In the prosthetic replacement a terminal device is substituted for the hand. The most functional terminal device is the so-called split hook to allow the amputee to carry out all the ordinary activities of daily living and almost every form of employment. In addition to this, various types of artificial hands are available. Although these are cosmetically superior none of them approach the split hook in function. The functions of the remaining joints of the arm are reproduced to a limited degree. The most important function is that of the elbow in which a lock is supplied to maintain it in any desired degree of flexion. The arm prosthesis is controlled through a shoulder harness which is so designed that movements of the stump only are necessary to control the prosthesis.

LOWER EXTREMITY FUNCTION

The lower extremity is primarily concerned with locomotion and in walking on level ground. Reserve power and function of the leg are so great that even a patient with a hip disarticulation can walk reasonably well using a properly designed prosthesis. The increased energy requirements in walking on stairs or ramps is immediately reflected in a marked increase in the amputee's limp. Stability of the lower extremity is achieved by aligning the joints in such a way that the knee is inherently stable in the standing position.

Man's leg is classified as being graviportal, which means that in the standing position the joints lock so that little or no muscular activity is needed to prevent buckling of the hip and knee. Thus, during the stance or weight-bearing phase of walking, the amputee walks with the knee in slight hyperextension to maintain stability and uses the muscles of the stump to maintain this hyper-

extension and to help in forward progression. The muscles of the remaining leg are called upon to supply additional power for progression.

During the swing phase of walking, the muscles around the knee act to damp excessive flexion of the knee. In the amputee this action can be duplicated by a mechanical brake placed in the knee.

AMPUTATIONS IN CHILDREN

Amputations in children are usually indicated either because of congenital anomalies or because of trauma. With present available prosthetic techniques children should be fitted at a much earlier age than formerly thought feasible. Leg amputees should be fitted with standard prostheses as soon as they have standing balance on the remaining leg or if they are bilateral amputees, as soon as they give evidence of being able to balance themselves on their stumps. Arm amputees should be fitted with a simple socket to use in crawling as soon as they begin this activity. As they begin to use their hands more actively they can be given a prosthesis with a simple clamping type of hook. Between the ages of three and four they can usually be given a prosthesis with full type of adult controls. It is important not to let children with arm amputations become accustomed to using the stump bare since they may then never achieve a satisfactory adjustment to the prosthesis.

With modern amputation and prosthetic techniques the great majority of all amputees can be successfully fitted. A small percentage of patients who cannot be fitted are usually unsuitable because of some severe type of general physical or psychologic disturbance.

READING REFERENCES

- Atiken, G. T., and Franks, C. H. Juvenile Amputee. *J. Bone & Joint Surg.* 35-A:659, 1953.
- Aldredge, R. H. Major Amputations. *Surg. Gynec. & Obst.* 84:759, 1947.
- Aldredge, R. H., and Murphy E. F. Prosthetics Research and the Amputation Surgeon. *Artif. Limbs* 1-4 (Sept.) 1954.
- Batch, J. W., Spittler, A. W., and McFadden, J. Advantages of the Knee Disarticulation over Amputation through Thigh. *J. Bone & Joint Surg.* 36-A:921, 1954.
- Bechtol, C. O. Suction Socket, Proper Selection of Patients for the Above-Knee Artificial Leg. *J.A.M.A.* 146:625, 1951.
- Bechtol, C. O. Prosthetic Clinic Team. *Artif. Limbs* 1-9 (Jan.) 1954.
- Canty T. J. Amputations and Recent Developments in Artificial Limbs. *U. S. Armed-Forces M. J.* 1147, 1952.

- Fletcher M J: Upper Extremity Prosthetic Armamentation. *Artif Limbs* 1:18 (Jan.) 1951
- Hacklin, C. C., and Thomas, A. Status of the Above-Knee Section Socket in the United States. *Artif Limbs* 1:29 (May) 1951
- Jampol, H. Physical Therapy Program for the Upper Extremity Amputee. *Phys. Therapy Rev.* 32:553 1952.
- Kessler, H. H.: Psychological Preparation of the Amputee. *Indust. Med.* 20:107 1951
- Kirk, V. T. Amputations. In *Lewis Practice of Surgery* Hagerstown, Md., W. F. Prior Company 1949, vol. 3.
- Klopsteg P. E. and Wilson, P. D. *Human Limbs and Their Substitutes*. New York, McGraw Hill Book Company 1951
- McKeever F. M. Upper Extremity Amputations and Prostheses. *J. Bone & Joint Surg.* 26:660 1914.
- Spittler A. W., and Rosen, I. E. Cineplastic Muscle Motors for Prostheses of Arm Amputees. *J. Bone & Joint Surg.* 33-A:601 1951
- Thorndike A. Medical Progress. New Techniques and Materials in Upper Extremity Prosthetics. *New England J. Med.* 247:1022, 1952.
- Wilson, P. D., in *Nelson's Loose-Leaf Surgery* New York, Thomas Nelson & Sons.

THE HAND

By MICHAEL L. MASON M.D.

MICHAEL LIVINGOOD MASON has devoted himself to extending the principles laid down by Kanavel in the treatment of infections, injuries and deformities of the hand. His training and experience in the teaching of anatomy particularly suited him for this role in surgery. A patient conservator of damaged tissue in preserving the function of the hand, he is extravagant in his use of color in following his hobby of painting. Dr. Mason serves his undergraduate and medical alma mater, Northwestern University, as Professor of Surgery.

In the past fifty years the surgical cure of conditions of the hand has developed from a consideration of infections to a definite subspecialty in general surgery. A significant factor has been the realization of the great functional value of the hand and an appreciation of its economic significance. The difficulties to be overcome before surgical therapy could be established on a sound basis were a lack of appreciation of the anatomy of the hand, an imperfect knowledge of wound and tissue healing and lack of appreciation of the technique necessary for dealing with the tissues of the hand.

The cure of diseases and injuries of the hand demands techniques developed in several other fields of surgery, e.g., orthopedics, plastic surgery and neurologic surgery as well as expert knowledge in the handling of wounds. In the past, hand injuries were usually entrusted to the least experienced, whereas it is now recognized that usually the most expert care is required even in "minor" injuries.

The first epoch-making advance in surgery of the hand was the publication of Kanavel's book, *Infections of the Hand*, the first edition of which appeared in 1907 and the seventh edition, at the time of the author's

death, in 1938. It would be difficult indeed to estimate the influence which this book has had in the development of surgery of the hand. In the experimental and clinical observations contained in this volume, a sound anatomic basis was laid for the understanding and treatment of the crippling and baffling processes of infections of the hand.

Advances in wound care and a firm knowledge of infections soon led to greater attention being paid to the care of wounds of the hand and to the surgical treatment of disabilities of the hand.

INFECTIONS

Infections of the hand are important because of the serious disability which may follow particularly if treatment has been late or inept. The spread of infections in the hand is determined by three factors: the anatomy of the area involved, the resistance of the host tissues and the virulence of the invading organism. The peculiarities of the structure of the hand determine certain pathways of spread which must be understood by the surgeon if he is to diagnose and treat infectious processes correctly. If only the cellular and subcutaneous tissues are involved, the immediate result is quite like similar

processes elsewhere. If however the subcutaneous tissues happen to be the pulp of a digit, or the palm, a quite different situation obtains. If the deeper tissue, planes and spaces are affected, the spread and possible consequences of the condition are again far different. This applies especially to the tendon sheaths and deep fascial spaces of the palm which in a few hours may be the site of an extensive infection from a very insignificant wound. Consequently it is necessary to know these anatomic pathways to understand properly and "outguess" the possible course and site of an infectious process.

If a knowledge of anatomy is necessary to know where an infectious process is likely to localize, it is equally necessary to know the anatomy of the part in order to secure surgical drainage should this be indicated. The intricate and compact anatomy of the hand is such that irreparable damage may be done to tactile areas and to nerves and tendons if the surgical approach is not the correct one.

Some estimate of the virulence of an infection is likewise essential to intelligent care. In the infirm or debilitated patient with little resistance to an invading organism an infection may spread very rapidly and lead to serious complications in a short time. Similarly when a very virulent bacterium is involved, e.g., from scratches with autopsy knives or pinpricks while dressing patients with virulent infections a fulminant process may develop in a few hours even in persons in the pink of condition. In the days before antibiotics were developed, many surgeons, pathologists and nurses suffered severe infections and not a few lost their lives from hand infections. It is important to know if an infection is being brought under control by the body forces, i.e., if it is being or has been localized, or if the bacteria are multiplying and extending into the surrounding tissues as diffuse cellulitis or into the lymphatics as lymphangitis. When localization is occurring or has occurred, a favorable outcome may be anticipated and surgical drainage can be instituted. On the other hand, if the process is diffuse and spreading and particularly if there is evidence of invasion of the lymphatics, the surgeon knows he has to deal with a more serious problem. He must direct all of his efforts toward favoring localization and increasing the patient's resistance. Furthermore, he must avoid all manipulation of the part and making an incision until localization has occurred. Fortunately the antibiotics are of great value in bringing these

spreading infections under control. Nevertheless even under an antibiotic umbrella, too early active surgical treatment is to be religiously avoided.

A determination of the exact organism responsible for the infectious process should be made whenever possible: routine culturing, both aerobic and anaerobic, will lead to more intelligent care. The development of the chemotherapeutic and antibiotic agents has served to emphasize further the need for accurate knowledge, not only of the infecting organism, but of its sensitivity to these agents.

GENERAL PRINCIPLES OF CARE

Certain general principles of care apply to all types of hand infections.

An early diagnosis must be made of the location of the process and whether it is spreading or is localized.

The anatomic location of the site of infection is of great significance since only by knowing this can adequate and proper drainage be carried out and the possible course of extension be predicted and anticipated. The need for determining whether the body immune processes have succeeded in localizing the infection is likewise of great importance. In the adequately localized process prompt drainage is usually indicated. In the acute spreading processes or in any infection in its early stage before the spread has been stopped, surgical intervention is contraindicated. In the acute spreading infections surgical intervention during the stage of spread may be disastrous and lead to death of the patient or serious extension of the process. In the early stages of less severe infections, such as a carbuncle or furuncle, early incision may lead to extension of the process, increased tissue injury and delay in healing.

The proper treatment for the infection should be started at once when the patient is first seen. Delay in instituting care may lead to serious sequelae such as tenosynovitis, felon and acute spreading infections. In any infection, recovery is more prompt and complications less likely to occur if therapy is started early than if it is delayed for fear of discommoding the patient. Prompt care does not mean immediate incision necessarily; on the contrary it may mean strict avoidance of incision.

Rest of the part is an integral element of care in the treatment of any infection. In an infection of the hand, proper splinting is a necessary feature. The extent to which splinting is carried out and the means of obtaining



Figure 1 The position of function. An important milestone in the development of surgery of the hand was the establishment by Kanavel of the importance of this position in immobilizing the hand

rest will vary with the location and severity of the infection. In infections involving only one digit, such as a paronychia or felon, it may be necessary to put only the digit at rest. In the more extensive processes, the whole hand and forearm must be immobilized. If the process is lymphangitis or acute spreading infection, the hand, forearm and arm must be immobilized and the patient put to bed.

Whenever the hand or any of its parts is put at rest because of an infection, it is essential that it be splinted in the position called by Kanavel "the position of function" (Fig. 1). This briefly put, is the position of grasp and not the position of rest. The wrist is dorsiflexed about 45 degrees, the metacarpophalangeal and interphalangeal joints are flexed about 30 degrees and the fingers are slightly separated from each other. The thumb is abducted from the side of the hand and rotated forward so that its pad faces the pads of the other digits. The advantages of this position are several. If any stiffness results from the infection, the hand will be capable of some use even with a minimum of motion. With a hand in this position, restoration of function is much easier. The ligaments of the various joints are in the position of maximum length when the hand is in the grasping position; hence, if they become involved, they do not restrict motion. If the joints are in the extended position, the ligaments are short in that position and, if they become fibrosed, hold the joints stiffly in extension and restoration of function is difficult, if not impossible (Fig. 2). With the wrist in moderate extension, the grasping power of the fingers is greatest and diminishes as the wrist is straightened and flexed (Fig. 3).

This position of function is applicable, likewise, if only one or two digits or the wrist only is to be splinted. Fingers should be splinted in slight flexion, the thumb in rotation and apposition, the wrist in extension.

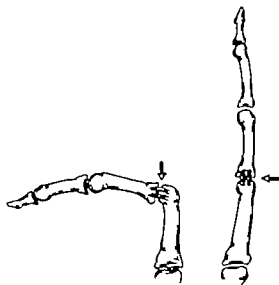


Figure 2 When the joints of the digits are flexed, the collateral ligaments are lengthened, when the joints are extended, these ligaments are shortened. Hence, when a joint is put at rest in a straight position, the ligaments are shortened and flexion may be difficult or impossible if immobilization is prolonged.

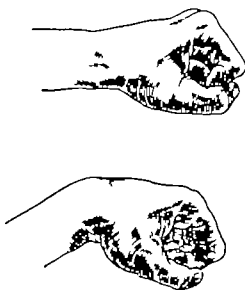


Figure 3 The grip is strongest with the wrist in slight extension.

The position of function will be found to be of importance in the care of all conditions of the hand which require splinting. In fact, if a hand is put on a splint in any other position, definite indications must be present for it.

The use of warm moist packs to favor localization of the infection probably by leading to increased blood flow is a time-honored therapeutic measure. The packs are of greatest value when they are applied as continuous dressings rather than intermittent

soakings, particularly in the acute and post-operative periods. They should be applied in such a manner that warm sterile physiologic saline solution may be poured into the dressings from time to time without the need of removing them (Fig. 4). Measures should also be provided to keep the dressings warm by external heat such as a therapeutic baker or hot water bottles. The need for maintaining sterility is particularly important following incision of an infection.

Operation to secure drainage of an infection is done with the care accorded an aseptic operative procedure. As a rule except in an early paronychia in which a tiny incision may be made without employing any anesthetic, general anesthesia is needed. Local infiltration is somewhat hazardous in an infected area and injection about the base of a digit may be followed by the development of gangrene of the finger tip. Incisions should be made in a bloodless field, secured by means of a blood pressure apparatus pumped up to 280 to 300 mm. Hg after the arm has been elevated for thirty to sixty seconds (Fig. 5). This bloodless field is necessary since the incision must be made with the care of an anatomic dissection. Hasty incisions within a field obscured by bleeding

are almost certain to cause trouble. Especially is this true of incisions for tenosynovitis or of the palmar fascial spaces where nerves, blood vessels and tendons are liable to serious injury.

Drainage is secured by adequate incision and not by the use of drains. When an incision has been made it is usually wise to insert a thin strip of rubber or petrolatum-soaked, fine-mesh gauze into one corner of the wound to keep the edges from becoming agglutinated. The strip is left in the wound not over forty-eight hours being usually removed at the first or second dressing. To be strictly avoided are stiff rubber or other hard drains that press against nerves and tendons. Likewise, through-and-through drains must be avoided.

After-care is of as much importance as the incision. Following incision, the warm moist packs are maintained for two or three days or until it is evident that the process is receding. This is manifested by recession of swelling and reappearance of the skin creases near the area of inflammation. As soon as recession begins the surgeon must start at once to secure mobilization in all joints not directly involved in the process. This can usually be accomplished by reducing the splinting to only those digits affected and leaving the other digits free. It is often necessary to stimulate the patient in the use of the

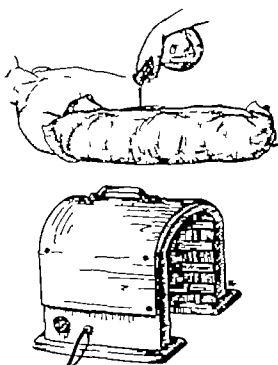


Figure 4. Warm moist dressings should be so applied that they need not be disturbed for the addition of warm sterile fluid (physiologic saline solution). The warmth is maintained by some sort of external heat (Mason, M. L.: *Infections of the Hand*. In *System of Medicine, Surgery and the Specialties*. J. B. Lippincott Company).

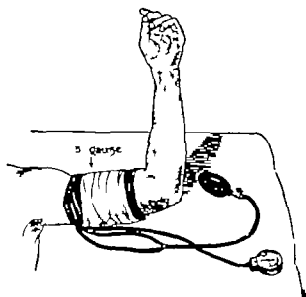


Figure 5. The use of a blood pressure cuff to maintain a bloodless field is standard practice in surgery of the hand. By this means, the surgeon prevents obscuration of the field by blood while identifying important structures. The cuff is applied loosely to the arm which is then elevated for thirty to sixty seconds to favor venous drainage. The pressure is then rapidly raised to 280-300 mm. Hg and the operation is carried out in a bloodless field.



Figure 6 All examinations and dressings are done under aseptic precautions with surgeon and assistant masked and using sterile instruments to handle all dressings.

uninvolved fingers which may have become quite stiff if the infection has been severe. Warm soapy baths in which the patient actively moves the fingers are instituted once or twice daily. It is when mobilization is attempted that the surgeon will be happy to have kept the hand in the position of function during the acute phase.

It need scarcely be pointed out that as long as the possibilities of adding secondary contaminants obtains, the infected hand should be accorded aseptic care. Every precaution is taken to preserve asepsis while removing and applying dressings and, of course, at operation. A mask should be worn and an instrument technique observed when the wound is being dressed (Fig. 6).

Antibiotics should be considered only as part of the treatment of an infection. In most instances before the offending organism has been identified, it is customary to give penicillin in good-sized doses once or twice daily preferably parenterally. The gradual emergence of penicillin-resistant strains of staphylococci has posed a real problem. They reflect the widespread use of antibiotics and appear to be especially prevalent in hospitals. On the other hand, many patients are developing sensitivity to penicillin, in which case some other antibiotic should be given.

As soon as the bacterium causing the infection has been identified, sensitivity tests should be made to determine which antibiotic is the most effective.

TYPES OF INFECTION

Folliculitis. This relatively minor infection of the hand involves only the dorsal surface which alone is supplied with hair follicles. It responds to simple care—usually it is enough to cover it with a small dressing. The temptation, however, is for the patient to pick and squeeze it or to pull out the hair. Not infrequently such treatment leads to extension of the infection, usually staphylococcal in nature, into deeper tissues.

Furuncle. The furuncle, or boil, frequently stems from a follicular infection which has been tampered with by the patient. It is due to staphylococcal infection, usually originating in a hair follicle or sweat gland, and starts as a small, reddish, painful swelling. In the early stage, the process may subside completely if covered with a dressing and if the hand is put at rest on a splint. Sometimes an injection of penicillin will bring about subsidence. More often, however, the process leads to necrosis of the surrounding tissues and a "core," or

material, will require release. Warm moist packs and a splint may produce spontaneous evacuation of the necrotic plug. If not, an incision will be required. It is best to incise the furuncle with the patient under light general anesthesia since the area of necrosis and the cavity are usually much larger than external appearances would lead one to believe.

Subcutaneous Abscess on Dorsum of a Finger. Folliculitis, or a furuncle, may give rise to a deep abscess on the dorsum of a finger usually on the proximal phalanx (Fig. 1). This process overlies the extensor tendon and may spread subcutaneously, over the whole of the dorsal surface of the phalanx. Operative drainage is indicated and must be done with the patient under a general anesthetic with bleeding controlled by means of a blood pressure cuff on the arm so as to enable the surgeon to visualize the whole extent of the abscess cavity.

Carbuncle. This oversized furuncle occurs on the hand only on the dorsum usually on the ulnar side, or over the dorsal surface of the proximal phalanx. It may start out as a tiny follicular infection and frequently there is a history of trauma. This type of infection should make one immediately suspicious of diabetes. The patient with a carbuncle should be put to bed with the hand immobilized on a splint and in warm moist packs kept continuously warm in a baker or similar apparatus. If diabetes is present control measures should be started. Penicillin or another antibiotic should be given in adequate doses parenterally. Under this regimen, the carbuncle may drain and evacuate itself spontaneously. If this does not occur incision will be required. It is seldom necessary to employ the wide cruciate incision usually required in the past, because warm

packs and splinting, plus an antibiotic, tend to minimize spread and favor localization.

Collar Button Abscess. The skin on the palmar surface of the hands and to some extent on the digits is thick and horny especially in the working man. It is subject to trauma, and small cracks, puncture wounds or blisters are frequent. These favor the deposit of bacteria beneath the thickened epidermis. Here a small abscess may form which spreads beneath the cuticular layer to form a purulent blister or *subcuticular abscess*. Such a blister causes few serious symptoms and usually subsides if it is derooft. However the blister may represent simply the subcuticular accumulation of pus which has found its way from beneath the corium into the subcuticular area. Arriving at the epidermis which is thick and impenetrable, it spreads out beneath, raising it up as it goes (Fig. 8). In a similar fashion, although infrequently, a simple subcuticular abscess unable to rupture to the surface may perforate the corium and form a deeper abscess. In either case, the subcuticular purulent blister is simply the superficial manifestation of a more deeply lying process. An infection of this sort is known as a collar-button abscess since there are two lakes of pus connected to each other by a narrow neck passing through the derma vera.

Collar button abscesses occur typically at the distal end of the palm, usually in the region between the metacarpal heads. They may lie, however directly over the flexor tendons at the base of the digits (Fig. 9). The deep portion of the abscess lies in the web space, from which it may extend distally to the dorsum and also into the subcutaneous tissues of the proximal phalanx which may be completely encircled.

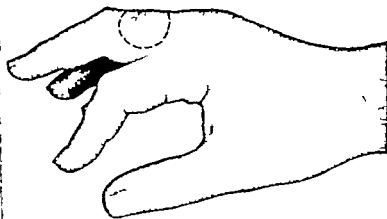


Figure 1. The dorsal subcutaneous abscess on the finger usually starts as a follicular infection which spreads over a much larger area than the superficial process would seem to indicate.

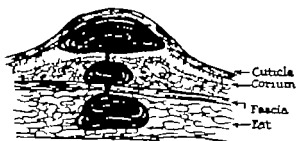


Figure 8 Any subcutaneous abscess on the hand may rupture through the corium and lead to subcuticular accumulation of pus. Drainage of this alone will not adequately evacuate the deeper pocket.

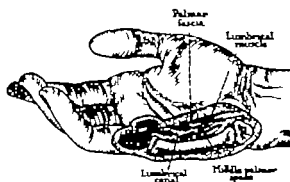


Figure 9 The frog felon or collar-button abscess is a typical type of bulocular abscess occurring at the base of the digits. A superficial subcuticular accumulation of pus simply indicates a deeper pocket (Mason, M. L. *Infections of the Hand*. In *Sajous System of Medicine, Surgery and the Specialties*. J. B. Lippincott Company.)

Treatment of such a process therefore consists in drainage not only of the purulent blister but of the deeper process as well. The surgeon should suspect the presence of such a deep abscess from the extensive swelling and redness and be prepared to drain the deeper pocket. It is always necessary when opening a purulent blister even if there are no signs suggestive of a deep abscess, to inspect carefully the floor of a blister to be certain that there is no tiny sinus leading to a deeper cavity.

Paronychia or "Run-Around." This infection starts in the nail wall or just between the nail and cuticle at the base of the nail. It is called "run-around" because it tends to spread under the cuticle from one side of the nail to the other. It is usually only moderately painful and the patient tends to temporize with it. If it is seen quite early pain may be quickly relieved and the process halted by means of a tiny incision in the nail wall or by lifting up the cuticle over the base of the nail with a blunt instrument (Fig. 10).

If the paronychia is neglected a more serious extension of the abscess takes place.

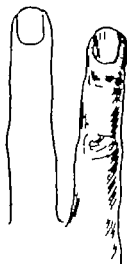


Figure 10 In its early stage, paronychia starts as a tender swelling in the nail wall which may usually be unroofed easily with a sharp scalpel or scissors without employing an anesthetic.

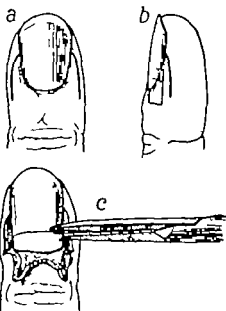


Figure 11 When the paronychia has extended beneath the nail, it is necessary to raise up the eponychium and excise the overlying nail to secure adequate drainage.

The pus finds its way under the nail, raising it up at the base and separating it from its bed. Treatment of such an infection requires meeting two indications. The cavity must be drained and the undermined nail must be removed since it acts as a foreign body which simply keeps up the process despite other wise adequate drainage. The cavity is drained by making incisions to either side of the nail base and then lifting up the cuticle and turning it proximally (Fig. 11). The nail is then inspected and if it is undermined, it is removed. Care must be taken to remove all

nail that has been undermined which usually necessitates removal of the proximal one-third. In any case sufficient nail should be removed that when the eponychium drops back it does not overlie the distal remaining portion of the nail and thus create a closed pocket. Following incision the hand is put at rest on a splint in a voluminous warm moist pack for twenty four to forty-eight hours until the acute symptoms have subsided. Thereafter, light dressings, which may be first moistened with Dakin's solution are applied. The nail will regenerate satisfactorily provided surgical drainage has not been too long delayed. The nail at first is likely to be quite rough and irregular but as it grows and slowly pushes off the distal portion it becomes smoother and eventually is quite a satisfactory nail.

The neglected paronychia which has been allowed to persist for weeks or even months without adequate drainage presents one of two pictures. The nail at the base may have been partly destroyed so that an exuberant plug of granulating tissue protrudes from the nail fold, covered with purulent discharge, or the eponychium at the base may be raised, red and swollen and an occasional drop of pus may be expressed from beneath it. In either instance adequate drainage is called for and the classical incision with nail removal and after-care should be carried out.

Chronic ulceration or exuberant granulations involving the nail bed may not be chronic paronychia, however and a careful history of the development of the process should be taken. For example a chancre of the nail bed is seen occasionally in physicians who have exposed themselves while carrying out vaginal examinations. A carcinoma of the nail bed may occur usually following an injury which has refused to heal after many months. A malignant melanoma of the nail bed, Hutchinson's melanotic whitlow is suggested by the black pigmentation, especially around the border. Rarely a subungual exostosis will raise up the nail and lead to a granulation tissue plug. A nail which has been subjected to excess irradiation will frequently break down and ulceration will occur.

Subungual Abscess. This process as distinguished from the subungual infection which occurs with paronychia, is usually seen as the result of a splinter or needle prick which enters the finger at the tip beneath the nail. Not infrequently a small bit of foreign material is carried in or the splinter breaks off. A painful infection develops which soon

leads to an abscess that can be visualized beneath the nail which is raised above it. It subsides rapidly when the process is unroofed by excising a small triangular slip of nail overlying the abscess. This may often be done without anesthesia, although it is sometimes so painful that brief general anesthesia is required.

Felon. In order to understand the felon and to treat it intelligently one must know the anatomy of the anterior closed space in the pulp of the finger (Fig. 12). The felon is a staphylococcal infection involving the resilient cushion which covers the volar surface or pad of the finger tip. Examination of this pad will readily show that it is uniquely adapted to the functions which it subserves namely grasp and fine touch. The finger pad enables the individual to pick up or cling to large or small objects, giving a firm resilient grip with tissues which do not slip or slide. As for touch we are all cognizant of the fine stereognostic sense of the finger tips which enables one to distinguish size, shape, consistency and texture of various objects. This pad, or cushion occupies approximately the distal two-thirds of the tip of the finger and is made up of a tough meshwork of connective tissue fibers somewhat like sponge attached

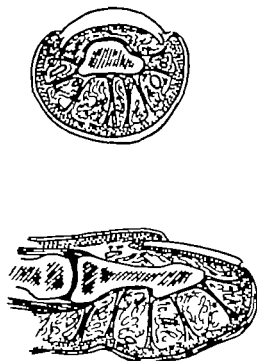


Figure 12 The pulp of the finger is a firm pad made up of interwoven fibrous trabeculae which run from the undersurface of the skin to the surface of the bone. It forms an admirably firm cushion useful in grasping. Since this area is unyielding, an infection leads to tense, painful swelling and early blockage of blood supply to the distal phalanx beyond the epiphyseal line.

on one side to the corium of the overlying skin and on the other to the volar surface of the distal phalanx. This latter insertion ends just distal to the point of attachment of the flexor profundus tendon. The anterior closed space is not connected with the tendon sheath of the finger.

The digital vessels and terminations of the digital nerves run through this space and, since this area is practically nonexpandable because of the dense fibrous cords which run through it, they are quickly pressed upon when inflammation occurs. This leads to early severe pain and, if tension is not soon released, to interference with the blood supply particularly to the bone.

The felon usually starts from a puncture wound of the finger tip which leads to throbbing pain within twenty four to forty eight hours. Along with the pain is tenderness and tension in the involved pad which can swell only moderately. The pain becomes progressively more severe and it is usually after a sleepless night that the patient sees his physician.

Examination will show the involved tip to be only slightly swollen, but tense, hard and tender. There will be little redness and the original puncture wound may be invisible.

Treatment should be started at once, since the combination of a closed infection, interference with blood supply and a necrotizing invading organism soon leads to necrosis of the soft tissues of the pulp and involvement of the bone. This is probably the most frequent cause of osteomyelitis of the bones of the hand. If the patient has reported early for care, it may be permissible to apply a splint, continuous warm moist packs over night and to administer an antibiotic. The infecting organisms are usually staphylococci. In an occasional case the process may subside overnight under conservative management. If, however, the symptoms are no better or are worse or if the tip is just as tender and tense as before, incision should be carried out at once.

Immediate incision is called for in the patient seen late or if very acute symptoms are present. The incision for the felon should divide the fibrous tissue bands or retinacula cutis which bind the undersurface of the skin to the anterior surface of the distal phalanx (Fig. 13). It should be placed along the lateral side of the phalanx midway between the anterior surface of the bone and the volar skin. After incising the skin, the knife enters the closed space parallel to the bone surface and sweeps across the space dividing the

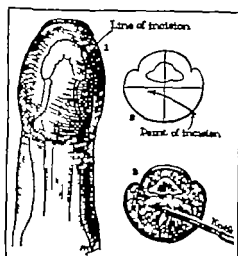


Figure 13 Drainage of a felon is accomplished by a single hockey stick shaped incision, along the side of the phalanx, which is designed to divide the flexor septa. Care must be taken not to make the proximal end of the incision too far proximally lest the tendon sheath be opened (Koch, S. L.: J.A.M.A., vol. 92)

retinacula. It is not necessary to divide the skin on the opposite side of the phalanx. Caution should be exercised not to carry the incision too far proximally lest the tendon sheath be opened and infected.

Following incision, a small rubber strip or strip of petrolatum gauze is placed in the wound to prevent agglutination of the edges. The hand is then placed on a splint in the position of function in continuous warm moist dressings. The originally administered antibiotic is continued unless sensitivity tests indicate that another is more efficacious.

If drainage has been adequate and early the process subsides fairly rapidly. If however a large necrotic plug has formed, this may take a considerable time to separate. If there is involvement of the bone, a conservative attitude is indicated. If drainage has been adequate the dead bone will slowly sequestrate or absorb. If drainage is not adequate further incision of soft tissues is required.

Osteomyelitis. Osteomyelitis of the bones of the hand is seen particularly in the distal phalanges following a felon or occasionally in association with other infectious processes which have been neglected. It may also occur following severe wounds which have become contaminated. It is practically never seen as a hematogenous infection. As a sequela to a felon, it is evidenced by persistent drainage and a granulomatous plug coming out of the drainage incision or from a sinus tract. Early x ray films may show rarefaction in the distal phalanx. ¹ be mistaken

for osteomyelitis. However while it is important to make the diagnosis early the treatment of this condition is the same as that for felon without osteomyelitis. Under no circumstances should the bone be attacked surgically. The soft tissues must be adequately drained down to the bone surface, but the bone itself must not be curetted or otherwise tampered with. Following drainage, the part is immobilized and sequestrum formation awaited. As soon as this is discharged, the wound closes. Occasionally in children the lost bone reforms but this seldom, if ever occurs in adults.

Tendon Sheath and Fascial Space Infections. In order to understand these infections a clear picture of the anatomy of these spaces is necessary (Fig. 14)

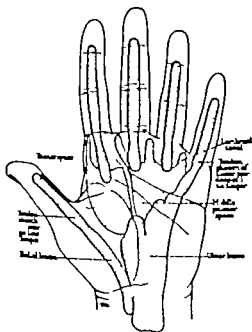


Figure 14 The tendon sheaths and fascial spaces of the volar surface of the hand and forearm form definite pathways for the spread of infection in the hand. When infectious organisms gain entrance into a tendon sheath, the process soon spreads throughout the whole sheath. When any of the three central digits are involved, the process comes to a momentary halt in the palm. If drainage is not soon provided, the process then ruptures into one of the fascial spaces in the palm—the middle palmar space if the middle or ring finger is implicated, the thenar space if the index finger is the original site. The pathway from the ulnar bursa on the little-finger side, however, ends in the lower forearm and an infectious process originating in this bursa ruptures into the retroflexor space here; similarly a process from the radial bursa extends into the lower forearm. To be remembered also is the fact that the radial and ulnar bursae usually communicate with each other and, hence, when one is primarily involved, the other is quickly invaded (Mason, *M. I. Infections of the Hand*. In *Sajous System of Medicine, Surgery and the Specialties*. J. B. Lippincott Company).

The flexor tendons of the hand are enclosed over a greater or lesser part of their extent by synovial lined sheaths, the synovial tendon sheaths. These structures are long, closed sacs which are invaginated by the tendons in such a way that the tendon is covered by a layer of the sac much as the bowel is covered by peritoneum. The sheath is then folded back on itself to line the space in which the tendon lies. Along the area through which the tendon invaginates the synovial sheath there is a space through which blood vessels enter and leave and this is often stretched out to form a wide membrane similar to the mesentery (Fig. 15). It has been called the mesotendon. In the digits this mesotendon is reduced in the region of the joints to ligament like structures known as the *vincula longa et brevia*.

The tendon sheaths of the index, middle and ring fingers extend from the insertion of the flexor profundus tendon on the distal phalanx upward into the palm where they end just proximal to the metacarpophalangeal joint, which lies at the level of the distal palmar crease. Within the digit, the tendon and its synovial sheath lie within a dense osteofibrous tunnel formed by a fibrous tendon sheath on the volar surface attached on either side to the phalanx.

The little-finger sheath does not terminate in the palm but proceeds on upward through the palm beneath the transverse carpal ligament and into the lower forearm for an inch or so. As it passes through the carpal tunnel, the sheath invests the tendons of the ring, the middle and then the index finger. This sheath is known as the *ulnar bursa*.

The sheath surrounding the thumb flexor likewise continues upward through the carpal tunnel to end in the lower forearm, an inch or so above the wrist. This sheath is known as the *radial bursa* and it usually connects with the ulnar bursa as the two pass side by side through the carpal tunnel.

Three fascial spaces lie deep to these ten-

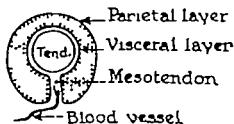


Figure 15. A cross section of a sheath-enclosed tendon shows the synovial sac which encloses it and also the thin membrane which forms the mesotendon through which pass the blood vessels nourishing the tendon.

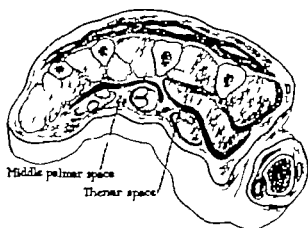


Figure 16 The relationship of the tendon sheaths to the fascial spaces in the palm (after Kanavel)

don sheaths and the relationship of them to each other is of importance. Two of these spaces lie in the palm in relation to the sheaths of the index, middle and ring fingers; the other lies in the lower forearm in relation to the radial and ulnar bursae.

In the palm (Fig. 16) a sheet of fascia from the palmar aponeurosis attaches to the middle metacarpal bone along the line of origin of the adductor pollicis muscle. On strictly anatomic grounds such a sheet is difficult to demonstrate, but, from a clinical standpoint, the presence of such a barrier is unquestioned. The retroflexor space to the radial side of this fascia is known as the *thenar space* and in its roof lie the flexor tendons to the index finger. The space to the ulnar side of the sheet of fascia is known as the *middle palmar space* and in its roof lie

the flexor tendons of the middle and ring fingers. The proximal ends of the digital tendon sheaths therefore lie in the roof of the two spaces.

The *retroflexor space* in the lower forearm lies just above the wrist beneath all the flexor tendons and in direct relation to the ends of the radial and ulnar bursae (Fig. 17).

Infections starting in a tendon sheath tend to travel throughout the whole length of the sheath almost at once—the sheaths are liquid tunnels in the fingers, palm and wrist (Fig. 18). In any of the three central digits, the infection is confined to the one digit until tension gets too great or the synovial sheath ruptures from some other cause. When it ruptures it does so as a rule through the least protected area, which is its proximal end, in the palm. When rupture occurs, the fascial space in relation to the sheath is involved. This means that if the index finger is involved the *thenar space* will be invaded, while if the middle or ring finger is implicated the *middle palmar space* is involved. Occasionally infection in the middle finger sheath will lead to infection of the *thenar space*.

The situation with regard to the *ulnar* and *radial bursae* is somewhat different. When these bursae are infected, the process extends upward through the palm and into the lower forearm. Since the radial and ulnar bursae are usually connected with each other at the wrist involvement starting in one is almost certain to affect the other—a condition which is called by the Germans a *V phleg*.

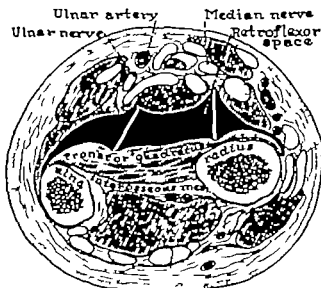


Figure 17 The relationship of the radial and ulnar bursae to the retroflexor space in the lower forearm (after Kanavel)

mon. When either the radial or ulnar bursa ruptures, it ruptures in the lower forearm into the *retroflexor space*.

Acute Tenosynovitis. This infection starts usually from a puncture wound, most often at the site where the sheath is nearest the surface, namely on the volar surface of the finger at the proximal or distal Interphalangeal joint. Once in the sheath, the infectious organisms quickly set up a reaction and the whole of the sheath is soon the site of inflammation. The sheath forms a liquid channel through which infection may spread.

As the inflammatory process develops and fluid accumulates in the sheath the tension increases and, since the synovial sheath is surrounded by a dense fibrous vaginal sheath, expansion is impossible. This is an example of the phenomenon of the closed space infection. Extrinsic pressure upon the tiny vasa which carry blood to the tendon leads to blockage of the vessels and, hence, if not soon relieved, to necrosis of tendon. Since rupture of the synovial sheath is retarded by the vaginal sheath, the synovial outpocketing in the palm or lower forearm bulges and ruptures. Thus, the infection is introduced into one of the three retro-tendinous fascial spaces, depending upon

which tendon sheath is involved. In case of the index, middle or ring digits the fascial spaces in the palm are involved. The process from the index finger invades the *thenar space* that from the middle finger ruptures into the *middle palmar* or occasionally the thenar space and that from the ring finger into the *middle palmar space*. If the radial or ulnar bursa is involved, the retro-tendinous space of the lower forearm is invaded.

In case of radial or ulnar bursa invasion, the course of events is somewhat more extensive than indicated above. It should be recalled that these two bursae are very closely associated at the wrist where both pass through the carpal tunnel. In most instances the two bursae actually communicate there and consequently an infectious process starting in one is quickly transmitted to the other. The extent of area involved is therefore much greater than that of the digital sheaths of the index, middle and ring fingers. The retro-tendinous involvement in radial-ulnar infection is in the forearm, in index, middle and ring sheaths in the palm.

Before these processes were well understood and logical methods for draining them established, these infections led to most serious crippling of the hand and occasionally to loss of life. The infection itself usually led to necrosis of tendons which would eventually be extruded through adventitious sinus tracts or be removed by surgical intervention. The "finger worms" of Paracelsus were necrotic tendons in infected digits which refused to heal until the "worm" was removed, usually by spontaneous separation. If tendons do not become necrotic, but somehow manage to survive both the effect of the infection and the vascular shut-off they become frozen in the sheaths and functionless. Not only the tendons but the whole fabric of the digit, palm and lower forearm may be involved in the matting of exudate consequent upon infection, and the whole hand may become frozen and stiff usually in a vicious position.

Added to the havoc wrought by infection itself were the damages due to inadequate, inept and incorrect surgical care. Until the surgeon understood the process, its site and localization, drainage was often delayed for long periods, either to permit the process to point or because he did not know where to drain. It was not appreciated that early drainage was essential to saving tendons and forestalling widespread extensions. If drainage was attempted without a clear idea of the site of infection, inadequate or illogical

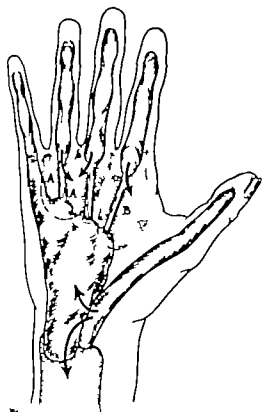


Figure 18. The spread of infection through the various tendon sheaths and fascial spaces of the palm and forearm is shown diagrammatically.

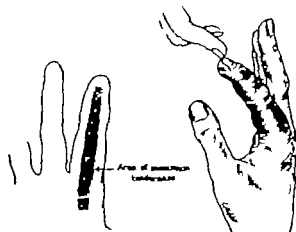


Figure 19 The cardinal signs and symptoms of acute tenosynovitis are represented in these sketches: 1 the finger is uniformly swollen, 2, it is held slightly flexed at all its joints, 3, any attempt to extend the finger passively causes exquisite pain, 4, the maximum area of tenderness maps out the anatomic outline of the sheath.

Incisions were made which either drained poorly or left serious scar contracture in their wake. Most common was an incision straight down the front or volar surface of a digit which drained the sheath but which permitted the tendon to bowstring forward out of the incision and become necrotic. If this tendon necrosis did not occur the midline incision still led to flexion contracture of the finger and an almost irreparable situation obtained. It was not infrequent for incisions to be made through and through either finger or palm and for hard rubber drainage tubes to be led from side to side in the digit or from palm to dorsum of the hand. These drains led to pressure necrosis and further increased the already crippling effects of the process. Those in the fingers caused tendon necrosis those in the palm carried infection to the dorsum where it was not at the start, and frequently caused osteomyelitis of the metacarpals.

It not infrequently happened also that incisions were made hastily and without due appreciation for the intricate anatomy of the hand. They were usually made without benefit of a bloodless field and many blood vessels, nerves and tendons were not visualized.

The recognition of acute suppurative tenosynovitis is not difficult if one remembers the nature of the process and if one keeps in mind the four cardinal symptoms established by Kanavel (Fig. 19). The patient with acute suppurative tenosynovitis usually reports fairly early to his physician because of the pain and inability to secure relief from simple home remedies and be-

cause he usually feels sick. It is apparent to the patient that he has something seriously wrong with the finger or hand.

Upon questioning, the patient will usually give a history of a minor injury pin or needle prick or knife injury to the volar surface of the involved digit twenty-four to forty-eight hours previously. Usually nothing was done about it. Aching pain will have begun in the finger the night following the injury and will probably have been severe enough to interfere with sleep.

On examination it will be noted that the patient is protecting the finger that he holds it slightly flexed and that the wrist likewise is flexed the forearm is supinated and supported on the opposite forearm. Jarring motion and dependency of the part are all avoided. The patient is reluctant to permit any except the gentlest examination. The finger itself will be swollen and this swelling will involve the whole finger which assumes a sausage-like shape. Attempts to straighten the finger passively lead to marked pain. Finally if the examiner takes a blunt probe and gently tests out the digit for an area of maximum tenderness, he will find this to coincide with the anatomic extent of the involved sheath.

These symptoms and findings hold true for all tenosynovitis however there are some extra features in case of infection of the radial and ulnar bursae. These bursae almost always communicate with each other hence, while the initial symptoms and findings occur in the bursa first affected, it is not long before the other is also involved and one will find evidence of its invasion. Thus, with suppurative tenosynovitis of the radial bursa, the thumb will be swollen, flexed and resist passive extension and there will be typical tenderness, which will extend from the distal phalanx of the thumb upward into the wrist and just above the transverse carpal ligament. Added to this will be evidence of ulnar bursa invasion which will present evidence of tenosynovitis though in the early stages not as severe as it may become later if adequate therapy is not carried out. Kanavel pointed out that the earliest sign of ulnar bursa invasion is an area of maximum tenderness in the palm at the point where the flexor tendon sheath of the little finger comes closest to the surface. This point lies in the palm where the distal flexion crease crosses the hypothenar eminence. Gentle pressure here leads to pain if the ulnar bursa is infected.

If the infection

little finger

and first involves the ulnar bursa, the chain of events is just the opposite: first there will be maximum symptoms and findings on the ulnar side of the hand and later evidence of radial bursa invasion.

It is important not only to be able to make the diagnosis of these cross bursal infections, but very important to suspect them in any patient in whom one of these bursae is involved.

The general symptoms associated with acute suppurative tenosynovitis are those associated with other types of surgical infections. However the rapid extension of the process within a closed space with relatively large surfaces of absorption often makes for quite marked general symptoms out of proportion. It may seem to the small wound of entrance.

The treatment of acute suppurative tenosynovitis must be prompt and efficient if tendons are to be saved and extension prevented. Penicillin or another antibiotic is started at once and preparation is made for immediate drainage. Only in exceptional circumstances should drainage be delayed twelve to twenty four hours.

As with all hand infections, incision is made in a bloodless field, secured by means of a blood pressure cuff pumped up to 280 to 300 mm. Hg after the hand has been elevated for sixty seconds. The actual incision is made with all the care of an anatomic dissection since important nerves

blood vessels tendons and uninvolved spaces of the hand must not be damaged (Fig. 20).

The incisions vary somewhat depending upon the space or spaces to be drained. The incision on the digits must be made on the lateral or medial side of the finger and, since the fingers are quite swollen, it is often difficult to know just where the lateral side is. As landmarks, the surgeon takes the ends of the transverse creases of the fingers which appear at the joints. Since it is a rule never to cross flexion creases in incisions of the hand this guide for incisions is not difficult to remember. Without following these guides the incision on the side of a badly swollen digit will be found to lie very close to the midline when the swelling subsides. Such incisions lead to midline contracture scars which produce flexion deformities of the finger.

Incision on the index, middle and ring fingers (Fig. 20 a) extends from the base of the distal phalanx up to the proximal flexion crease of the digit. It does not extend into the palm. The proximal cul-de-sac of the sheath of these fingers is drained by a transverse incision in the palm at about the level of or slightly distal to the proximal flexion crease in the palm. Since this incision passes directly across nerves (Fig. 20 e) vessels and tendons it is obvious that a bloodless field permitting clear vision is necessary.

On opening of the finger the edematous

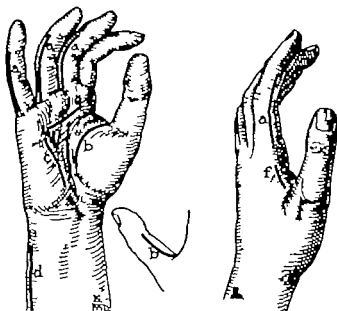


Figure 20. Incisions for drainage of the tendon sheath and fascial spaces must be made with the care of an anatomic dissection in a bloodless field. The incision in the digit is made on the midlateral side and, since the finger is swollen, it may be difficult to place this incision correctly. The proper location of the incision is indicated by the ends of the transverse creases on the digits which are never obliterated, regardless of the degree of swelling (Mason, M. L. Infections of the Hand. In *Sajous System of Medicine, Surgery and the Specialties*. J. B. Lippincott Company).

fat will bulge out into the operative field. The surgeon must have assistance to keep this fat retracted so that he can first identify the digital nerve and vessels. Passing volar to these structures, the tendon sheath is exposed and opened throughout its length in the digit. It is thought by some to be important to attempt to save parts of the vaginal ligament or fibrous sheath. To this end, some surgeons advise making the distal half of the incision on one side of the digit and the proximal half on the other.

When the incisions have been made and culture taken for identification and sensitivity tests, warm moist compresses are held in the wounds and the blood pressure cuff is released. After a few minutes the incisions are inspected for arterial spurs which are ligated. A soft petrolatum gauze strip is laid in the incision to prevent agglutination of the edges and a voluminous moist pack is applied with the hand laid on a splint in the position of function.

The radial and ulnar bursae call for further incisions than those on the digit. The digital portion of the incision is made as described, however drainage of the palmar and forearm prolongation of the two bursae is also required. The palmar portion of the ulnar bursa is opened by an incision which roughly parallels the hypothenar eminence (Fig. 20 c) and its distal end may have to cross the proximal palmar crease to secure an adequate opening. Proximally this incision ends at the level of the transverse carpal ligament. The forearm portion of the ulnar bursa is then drained by an incision (Fig. 20 d) about 3 inches long just anterior to the ulna on the medial side of the wrist.

The palmar portion of the radial bursa is drained by an incision which skirts the ulnar border of the thenar eminence (Fig. 20 b). It extends from the region of the proximal flexion crease of the thumb upward to within a thumb's breadth of the transverse carpal ligament. It must stop there, otherwise there is danger of dividing the motor branches of the median nerve to the thenar muscles. In fact, this portion of the incision of the radial bursa is difficult since the sensory ramus of the median nerve to the thumb also crosses the tendon sheath and must be visualized.

The proximal, or forearm, portion of the radial bursa is opened along the radial side of the wrist by an incision about 3 inches long, made just anterior to the radius. Usually if the ulnar bursa is to be drained at the same time the ulnar incision will suffice for

drainage of the proximal ends of both bursae.

The middle palmar space usually is involved by extension from a digital sheath and the thenar frequently is. However the thenar space is just as frequently if not more often primarily involved.

Evidence of middle palmar involvement may be surmised if in association with tenosynovitis of the middle or ring fingers, there is brief relief from the pain followed quickly by exacerbation of both local and general symptoms. Locally the normal palmar concavity is lost and the palm becomes flat, tense, hard and gray. The tenderness, which at first was confined in its maximum intensity to the tendon sheath, becomes localized also in the center of the palm. The general symptoms increase in intensity, pain becomes more severe, and the swelling of the whole hand increases. The patient becomes very ill and the fever increases along with other symptoms of a severe infection.

The involved thenar space produces similar but usually not quite such severe symptoms. The swelling is typical. The hand looks as if a golf ball had been inserted in the soft tissues between the metacarpals of the thumb and index finger. The thumb is pushed away from the hand and the whole area is markedly edematous. Maximum tenderness is in the first interosseous space particularly in the palm. Early drainage of these spaces is indicated. If the associated tendon sheath is infected and requires drainage, the fascial space is drained at the same time.

The middle palmar space is opened through the same incision which drains the proximal cul-de-sac of the sheath (Fig. 20 e). Forceps are inserted beneath the tendon and passed proximally and enter the space. In the case of the thenar fascial space the incision is made on the dorsal surface of the web space between the first and second metacarpals on a line joining the metacarpal heads and about $\frac{1}{2}$ to $\frac{3}{4}$ inch proximal to the border of the web (Fig. 20 b). A hemostat is passed forward over the first dorsal interosseous muscle and posterior to the flexor tendons of the index finger into the thenar space.

The after-care of these infections is the same as for tenosynovitis: warm moist packs, splinting and appropriate antibiotic therapy.

Lymphangitis and Acute Spreading Infections. Most infections of the hand begin as a small diffuse process which soon becomes localized to certain areas of the hand or lower forearm. Many of them require a

longer or shorter period of rest and immobilization; often the use of warm moist packs is permissible to favor adequate localization before drainage. The one real exception to this is suppurative tenosynovitis which demands early incision.

Acute lymphangitis and acute spreading infections are caused by highly virulent organisms which spread rapidly in lymphatic and tissue spaces before body resistance can be developed to combat or control them. Many are due to bacteria of highly developed virulence against which the body has no resistance. These are the infections which, in the days before the chemotherapeutic agents and the antibiotics, often led to the death of the patient.

The courses of extension of these two processes are essentially the lymphatic system of the hand and the associated areas through which the lymphatics pass. The problem of the lymphatic spread was carefully studied by Kanavel. On the basis of Boer's original work, the various typical patterns of spread and eventual localization of the acute spreading infection were described by Koch.

The hand is richly supplied by lymphatic vessels, especially on the palmar surface (Fig. 21). The lymphatics from the palm drain roughly in two directions. Those on the heel of the palm, that is the proximal portion of the palm, drain upward along the volar surface of the forearm toward the elbow and axilla, following in general the main venous channels. The remainder of the lymphatics of the palm and those of the digits pursue the shortest course to the dorsum of the hand and then pass upward to the axilla, some passing through the epitrochlear nodes, others through the axillary nodes, while some skip both axillary and epitrochlear nodes to empty directly into lymphatic channels in the neck. Kanavel pointed out that the lymphatics of the ring and little fingers pass first through the epitrochlear nodes before reaching the axilla and that those of the thumb and index finger pass directly to the axilla. Those from the middle finger may skip both sets of nodes and pass directly into the neck. It was for this reason that the middle finger was known as the "dangerous finger" since lymphangitis starting there may quickly lead to systemic infection unless brought under early control.

The acute spreading infections start as lymphangitis which quickly leads to diffuse involvement of the soft tissues of the ex-

tremity. The patterns of the different types of involvement were established by Koch and are explicable on the basis of lymphatic drainage.

The source of infection for the acute spreading process is usually a minor injury of some sort, frequently one whose treatment has been neglected. At the start there will usually be lymphangitis which will be manifested by the usual red streaks in the areas of drainage of the involved part. Soon, however, this process will be complicated by a much more diffuse and tense swelling, particularly of the back of the hand and forearm. Unless the infection is so fulminant as to cause death rapidly, localization of the infection follows certain predictable patterns depending upon the original site of inoculation (Fig. 22).



Figure 21 The lymphatics of the hand pursue the shortest course to the dorsum, where they form into a number of channels which pass up the forearm and arm. The drainages from the thumb and index finger usually skip the epitrochlear nodes and empty into the axillary nodes. Those from the ring and little finger pass first through the epitrochlear nodes before emptying into the axillary nodes. Those from the middle finger may miss both sets of nodes and drain directly into the lymphatic vessels in the supraclavicular area (Mason, M. L. *Infections of the Hand*. In *Sajous System of Medicine, Surgery and the Specialties*. J. B. Lippincott Company).

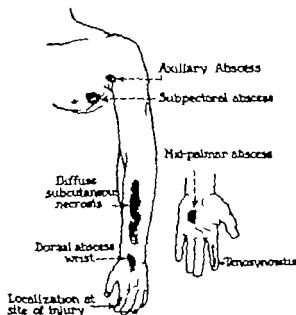


Figure 22. The acute spreading infections show several quite definite patterns of spread and localization as described by Koch. This illustration shows diagrammatically the various typical pictures seen in these dreaded infections.

If inoculation has occurred on the volar surface of a finger the lymphatics in passing from volar to dorsal surface lead to a tenosynovitis of the digit. They then pass on to the dorsum of the hand and the infection not infrequently becomes localized on the dorsal surface of the hand and wrist. In fact, in practically every instance, localization occurs over the dorsum of the wrist and is associated with extensive necrosis of subcutaneous tissues and sloughing of overlying skin. This necrosis of subcutaneous tissues and skin may in some instances be very extensive and involve almost the whole of the subcutaneous tissues of the dorsum of the hand and forearm. Such extensive invasion and local necrosis do not of course, always develop. There may be simply marked lymphangitis and inflammation of epitrochlear and/or axillary nodes, which may occasionally break down into abscesses. When treatment is started early no localization whatever may occur; there will be the streaks of lymphangitis and the nodal areas will show swelling and tenderness, but the process subsides often as rapidly as it began, without any abscess formation. This latter course is the one usually seen today when antibiotics are readily available and are promptly administered.

These infections, which were among the most feared of all and ranked with peritonitis as the cause of death of victims, have tended to disappear since the advent of the

antibiotics. While it is no doubt true that antibiotic agents are used much too promiscuously and often unnecessarily it cannot be denied that their use has forestalled the blossoming of many a case of lymphangitis with a serious life-threatening infection. Although one is tempted to accord to the antibiotics the major credit, it must be apparent that there are other factors which have helped to reduce the development of the acute spreading infection. A significant factor has been a real appreciation of the importance of hand infections and a more cautious attitude toward the care and management of even small wounds of the hand, which in the past have been the most potent source of the serious infections.

Other factors may also be at work which are as yet not completely understood. It may be likely that the greatly improved nutritional status of the general population and possibly even differences in the bacterium-human relationship are of great significance.

The therapy for acute spreading infections must be aggressive but conservative—aggressive in that measures must be instituted at once to combat the process, conservative in that the process must be handled with the greatest gentleness and that all temptations and attempts at persuasion to operative intervention must be firmly resisted. Operative intervention before the process has been brought to a standstill is certain to lead to disaster. In the preantibiotic days, not a few patients, and among them many doctors and nurses lost their lives and many patients became hand cripples from the sudden and uncontrollable spread which followed ill-advised active intervention.

The patient with a spreading infection must be immobilized immediately preferably in bed in a hospital and measures instituted promptly to favor localization of the process. The hand, forearm and arm are placed on a splint, enclosed in a voluminous warm moist pack and kept at rest to prevent any motion and the consequent milking of lymphatics and the breakdown of whatever barrier is being laid down by the body defense processes. Proper administration of fluids and sedatives for pain constitute the general measures which are instituted. Antibiotics are given in large doses from the start. When no bacteriologic diagnosis can be made, penicillin is the agent of choice given in doses of 400,000 to 600,000 units once or twice daily. If material is available for bacteriologic identification and study of sensitivity the appropriate

This regimen is

ven

process has been brought under control which may occur in twenty four hours or may require several days. The subsidence of the process will be evidenced by the disappearance of local signs of lymphatic inflammation and diminution of the general symptoms. In many instances no localization whatever will occur and the findings and symptoms subside completely over a period of several days. If localization occurs the involved area, usually over the dorsum of the wrist, suddenly softens the overlying skin may show areas of necrosis and when these are opened, copious discharge of necrotic subcutaneous tissue and pus will take place. If the infection had its onset on the volar surface and a suppurative tenosynovitis has been present its presence will have been evident and the severe pain associated with it will have pretty well subsided.

At this time, drainage should be instituted if necessary. Drainage is especially needed when suppurative tenosynovitis has been present. The dorsal subcutaneous processes often drain spontaneously, although incisions will speed up the process and broken-down lymph nodes will need incision. Following drainage, the warm moist packs and splints are maintained for a further two or three days, following which they may be discontinued and dry dressings or dressings moistened with Dakin's solution are instituted.

Human Bite Infections. Injuries to the hand from contact with the teeth of an opponent are not particularly infrequent. If these wounds are not recognized for what they are when first seen or if the individual neglects to seek help very serious infections may occur. Most often the wound is received when one person strikes another his knuckles coming in contact with his opponent's teeth. The skin and tendon are tightly stretched over the metacarpophalangeal joint and are easily perforated, leading to direct contamination of the joint cavity (Fig. 23). Likewise, at the same time several other spaces

are entered above and beneath the extensor tendon. As the joint straightens out the tissues slide back in layers and effectively seal off the wound. From here may develop suppurative arthritis, subfascial and subtendinous infection and if the process lasts long enough extension of the infection may lead into the palm via the lumbrical canals.

The tissues are not just perforated, but crushed and hence are not resistant to infection. In the second place the organisms introduced into the tissues are extremely virulent and immediately invasive. Both aerobic and anaerobic streptococci and staphylococci as well as various spirochetes and numerous other organisms will be found on smear and cultures. These lead to immediate development of infection characterized by production of foul-smelling pus. Unless soon brought under control, a severe acute spreading infection supervenes and may lead to a fatal process. Locally the process invades and destroys bones and joints.

When these injuries are seen early they should be widely opened excised and irrigated. Under no circumstances should these wounds be closed. The extensor tendon, which is not infrequently divided, should not be repaired. The hand should be splinted in the position of function and a voluminous warm, moist dressing applied. There is evidence that the use of zinc peroxide is of value locally if applied according to Meloney's directions. The patient is preferably hospitalized. Antibiotics should be started at once in large doses. Penicillin is usually chosen initially and it would seem advisable in view of the mixed nature of the infection and the presence of gram-negative organisms to add streptomycin. Bacitracin applied locally has proved of great service in these cases. As soon as bacteriologic studies have been completed, it may be necessary to change the antibiotic therapy.

The further management consists in securing proper drainage when and if required. There will be a temptation to attack bones surgically when osteomyelitis appears. It must be remembered that the roentgenologic findings may simply represent calcium absorption and not actual destruction. The osteomyelitis is managed, as in the case of felon, by watchful waiting and maintaining adequate soft tissue drainage, and removing sequestra when they appear.

Chronic and Unusual Infections. Although most chronic infectious processes may involve the hand, only a few of them



Figure 23. Human bite injuries (more correctly human tooth injuries) of the hand usually result from tooth perforation over a knuckle. The tooth penetrates in several different spaces in its passage—first, the subcutaneous space, then the subfascial space beneath the tendon, then the joint space. Serious involvement of all these areas soon occurs (redrawn from Mason, M. L., and Koch, S. L.: *Surg. Gynec. & Obst.*, vol. 51).

are of sufficient importance or sufficiently peculiar to the hand to deserve special description. Such infections as actinomycosis, blastomycosis, coccidiosis, leishmaniasis, anthrax and syphilis may be seen on the hand. Tuberculous tenosynovitis, erysipeloid, sporotrichosis, tularemia and milkers' granuloma are more common.

Tuberculous tenosynovitis. Tuberculous tenosynovitis is a specific chronic infection of the bursae and tendon sheaths of the hand and foot.

It is slightly more frequent among females than males. It is most often seen in young adults and involves the right hand more than twice as frequently as the left. Tuberculosis is not often found elsewhere, but it is imperative to examine carefully for other foci, especially in the chest. Direct inoculation may rarely occur from safety pins (attendants in sanatoriums) or bone spicules (butchers); usually, however, there is no clue as to the source of the lesion.

The radial and ulnar bursae of the hand are the most frequently involved, next in frequency are the dorsal tendon sheaths of the hand, then the digital sheaths of the index, middle and ring fingers and, lastly, the tendon sheaths on the foot. The radial and ulnar bursae are almost always infected together, but their distal prolongations into the thumb and little finger may remain free from disease. The process is so rarely bilateral that the presence of such involvement practically excludes tuberculosis.

The sheaths may be but slightly thickened and injected and contain a moderate amount of yellow serous fluid. More often, however, both parietal and visceral layers are thick and shaggy and the enclosed tendons are gray and lusterless, while in the small amount of yellow fluid are fibrin flakes and rice bodies. There may be extensive formation of granulation tissue with caseation and destruction, with or without rice body formation. As the disease progresses the tendons are invaded; in fully 75 per cent of the patients, serious tendon destruction will be found and, in some 50 per cent, whole segments of tendon will be destroyed or so badly fragmented as to require removal. In neglected cases, the associated structures—bones, joints and muscles—are invaded and cold abscesses form, with their attendant danger of secondary infection. The median nerve, though compressed and swollen, is never infiltrated.

The rice bodies or melon-seed bodies found

in about 50 per cent of cases are small, shiny grayish, ovoid or kidney-shaped, flattened or faceted masses varying in length from 1 to 15 mm., and in number from one or two to many hundreds. They are probably the result of the mechanical action of the moving tendons on villi and fibrin-encrusted products of necrosis of the tendon sheath.

The disease is characterized by the insidious development of a slowly progressive, painful or painless swelling over the anatomic area of the involved sheaths. In one third of the patients the swelling is preceded by sensory or functional disturbance, such as pain, tingling, numbness or prickling sensations or by stiffness in one or more fingers. These premonitory symptoms may persist for many weeks or months before the actual swelling appears. A leathery crepitus, as in traumatic tenosynovitis, may occasionally usher in the tuberculous process.

The swelling often appears as a sudden diffuse edema which responds to heat and rest only to be replaced later by the more tense localized, persistent swelling. With involvement of the radial and ulnar bursae, the swelling begins at the wrist and extends slowly into the palm, whence it may proceed down over the volar surfaces of the thumb and little finger. With involvement of the digital sheaths of the index, middle and ring fingers, the swelling first appears over the proximal phalanx and then extends upward into the cul-de-sac of the sheath in the palm. In cases of long standing, the disease spreads beyond the limits of the sheaths and leads to swelling in the middle palmar space, thenar space or fascial spaces of the forearm.

The pain is often quite mild or even absent. In some patients a severe burning or throbbing pain occurs which radiates upward over the extremity and is increased by use of the involved hand. It may occur only at night or in the early morning hours and lead the patient to seek relief by the application of heat or cold. After persisting for several months or a year the pain usually subsides even though the swelling slowly increases.

In the early stages of the disease, the patient has good use of the part except for the associated pain. Stiffness, weakness and difficulty in closing the fist soon develop while later on swelling interferes mechanically with movement and destruction of tendons leads to its actual loss.

If the disease is allowed to progress, bone and joint destruction may render the hand

useless and the formation of cold abscesses and secondary infection may necessitate amputation and threaten the patient's life.

Tuberculous tenosynovitis produces a fusiform swelling which in the early stages may not be apparent unless compared with the sound side. The swelling at the wrist is in completely divided into a proximal and distal half by the transverse carpal or dorsal carpal ligament. On the dorsum of the wrist the several longitudinal compartments can be made out corresponding to the dorsal tendon sheaths. Disease of the digital sheaths produces a swelling resembling spina ventosa. If rice bodies are present they give a sensation resembling lead shot in a leather bag and produce a peculiar grating crepitus when the patient is asked to move his fingers. A moderate amount of atrophy of the associated muscles occurs. There is little or no increase in the temperature of the part and the skin is normal unless cold abscesses, sinuses or secondary infection is present.

Tuberculous tenosynovitis must be differentiated from other forms of chronic tenosynovitis and from tumors of the tendon sheaths.

Chronic traumatic tenosynovitis, most often on the dorsum of the hand is diagnosed by the history of an acute onset followed by unaccustomed strenuous use and its rapid response to immobilization.

Chronic nonspecific tenosynovitis produces a clinical and gross pathologic picture so closely resembling that of tuberculosis that microscopic section is required to make the diagnosis. Bilateral chronic tenosynovitis is almost always nonspecific.

Syphilitic tenosynovitis is very rare and may produce a clinical, gross and microscopic pathologic picture so closely resembling that of tuberculosis that the differentiation is extremely difficult. An acute or subacute inflammation may occur in the early secondary stage and the syphilitic hygroa and chronic gummatous type in the tertiary stage. The condition is likely to be bilateral, is rarely painful and causes few or no functional symptoms. The diagnosis is established by the presence of associated lesions, a positive serum reaction and response to specific therapy.

Gonorrheal tenosynovitis occurs most often in males, is metastatic, secondary to a urethral infection or more rarely to vaginitis or ophthalmia, and may follow instrumentation. The onset is acute with rapidly developing pain and loss of function. A tender swelling is produced over the course of

the tendon sheath with little or no change in the overlying skin. Aspiration of the swelling yields a cloudy serofibrinous or even purulent fluid.

The common dorsal ganglion produces a tense circumscribed globoid swelling on the dorsum of the wrist, most often on the radial side, and rarely reaches greater than hazelnut size.

Xanthomatous tumor or villinodular synovitis, of the tendon sheaths may occasionally lead to confusion when it occurs as a diffuse tumor at the wrist instead of a circumscribed smooth nodular swelling of a finger. It is however firmer and more nodular than tenosynovitis and fluctuation is absent.

Arborescent lipoma is a rare condition in which a fatty branching tumor spreads throughout the sheath. It grows slowly and insidiously and causes few or no functional symptoms.

Extrasynovial lesions are rarely confused with tuberculous tenosynovitis. A roentgenogram of the hand and wrist should be made to determine the presence of bone or joint involvement.

The prognosis for tuberculous tenosynovitis is good. Few patients will be found with active tuberculosis elsewhere and unless grossly neglected the local process should not threaten life. If the condition is treated early and properly a primary cure should be obtained in the majority of patients but repeated operations will be necessary in a few. The functional results are good. In an occasional patient, amputation of a digit or an extremity may be necessary.

Management should be directed both toward the local lesion and toward the general state of the patient, as emphasized by Adams, Jones, and Marble. The treatment of choice for the local lesion is complete surgical excision of all diseased tissue. If the diagnosis is uncertain, the surgeon may temporize for a short time with immobilization and heat, but this should not be persisted in after the diagnosis is made. Complete surgical excision is extensive and time consuming but if carried out thoroughly justifies the pains taken. The operation is performed, with the patient under general anesthesia, in a bloodless field secured by means of a blood pressure apparatus. Appropriate incisions are made, avoiding as much as possible midline incisions and incisions crossing flexion creases, and great care is taken to identify and isolate all important nerves and blood vessels. The tuberculous sheath is opened and each tendon taken in

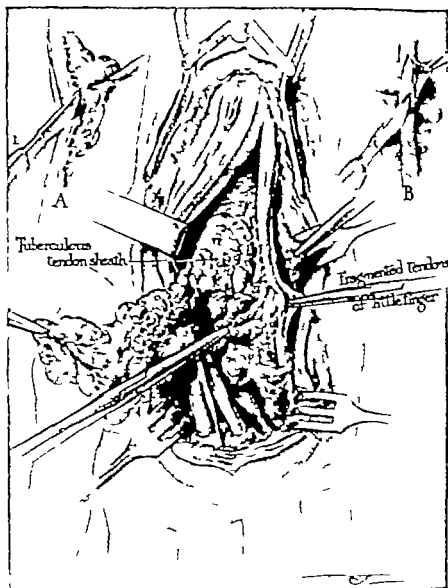


Figure 24 Details of operative treatment of tuberculous tenosynovitis. After the median nerve has been carefully retracted, each tendon is taken in turn as in A and the tuberculous tissue removed. Great care must be exercised to remove the visceral layer of the sheath as in B (Kanavel, A. B.: *Surg. Gynec. & Obst.* vol. 37)

turn and carefully stripped of diseased tissue (Fig. 24) after which the parietal portions of the sheath are excised. Fragmented and diseased portions of tendons must be removed and it may be necessary to repair tendon defects by means of a suture or tendon graft.

Other less radical procedures are not to be recommended. Partial excisions, simple drainage, simple removal of rice bodies and injection of various medicaments have not proved successful.

Following operation, it seems advisable to give a course of streptomycin or streptomycin and PAS, since it is doubtful if even the most meticulous dissection removes all diseased tissue.

Erysipeloid. This condition, resembling a chronic type of erysipelas, is occasionally ob-

served in fishing towns among fishermen or fish handlers, dealers of poultry, cooks and veterinarians and in others who come in contact with fish.

The site of inoculation becomes red and indurated and burns and itches in a manner similar to that attending any type of bacterial inoculation. Few general symptoms are present, although the patient may complain of malaise and there may be a slight elevation of temperature. As the disease progresses, lymphatic streaks may be present. The lesion takes on a purplish color, the borders become raised and papular and the condition tends to spread. As extension occurs, the central portion clears and heals.

The disease is self-limited and usually subsides in a few weeks, leaving no immunity to further attack.

minant attacks may occur and the condition may become chronic.

Tularemia. This infection known commonly as rabbit fever is a widespread disease, having been described in almost all countries of the world although it has been recognized only since 1910. The natural habitats of the causative organism—*Pasteurella tularensis* which is carried among wild life by such vectors as ticks, lice and deer flies—are rabbits, squirrels, woodchucks, muskrats and wild rats.

Inoculation may and frequently does occur in the hand in persons who handle and skin wild game. Rabbits, and to a lesser extent squirrels, have been the source of most human infections.

The site of inoculation becomes manifest in from one to six days after injury as a red papule, producing moderate pain. There may be chills, fever, headache and general aching, such as occurs in many infections. The general symptoms usually last for two to three weeks and subside gradually.

The local lesion, at first apparently subcutaneous, becomes larger, more indurated and attaches to the overlying skin. The induration increases and the center of the nodule breaks down into a necrotic core which soon sloughs out, leaving a granulat-

ing ulcer that heals slowly. The lymphatics leading from the area may become inflamed, the epitrochlear and axillary nodes may enlarge. There may or may not be splenic enlargement.

Some six types of the disease are described depending upon the manner of the original inoculation and the virulence of the infecting strain. The ulceroglandular type is seen on the hand and upper extremity. Other types described are the pulmonic, glandular, ingestion typhoid and oculoglandular.

Treatment of the condition consists first in avoidance of contamination with the organisms. General measures of treatment for the condition when it once develops consist in bed rest and general supportive treatment. Before the introduction of the antibiotics, Foshay's serum was the mainstay in the therapy and was remarkably efficient. It has been replaced by streptomycin.

Diagnosis is made on the history of exposure plus identification of the organism from the secretions and guinea pig inoculation. The intradermal test of Foshay is specific and should be carried out in all suspected cases.

Sporotrichosis. *Sporotrichum schenckii* is responsible for this fungus infection which is peculiar to the hand and forearm being

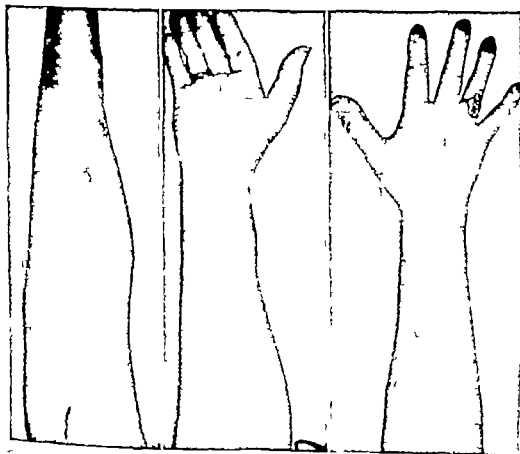


Figure 25. Sporotrichosis from thorn injury of the hand.

seldom seen on any other portion of the body. It is a common fungus of plants and shrubbery and seems to be particularly frequent on roses. A number of the patients whom we have seen have been pricked by rose thorns.

The condition starts as a subcutaneous nodule several days or so following injury (Fig. 25). The nodule may reach an inch or more in diameter before it becomes fixed to the overlying skin. It produces few symptoms, little pain or tenderness and eventually breaks down into a dirty ulcer or sinus tract, discharging gummy brown pus. Soon the lymphatics leading from the area become thickened and cordlike and nodules develop along their course. The nodules may likewise break down as did the original lesion. The associated lymph nodes also enlarge but seldom actually break down.

The general reaction to the disease is remarkably slight, the patient is not sick and there is little if any elevation of temperature.

Diagnosis is made on the rather typical findings and is confirmed by demonstration of the organisms in a smear. The reaction to the agglutination test is positive.

The disease runs a slow chronic course and tends to be self limited but prolonged. Large doses of potassium iodide, 60 to 150 grains a day should be administered and are quite helpful.

Milkers' nodule and hair granulomas of barbers. Injuries to the palm due to the penetration of hair are known to occur in association with two occupations—milking and barbering. The mode of penetration of hair is obvious, the explanation of the chronicity of the process is likewise obvious when one considers the make-up of hair. The hair is a scaly structure in which the scales lie on the surface like shingles on a roof. The free ends of the scales point away from the distal end of the hair. Consequently when the hair penetrates the palm, the scales act like the barbs on a fishhook, they tend to penetrate deeper and deeper but retraction is hindered by the scales.

These injuries produce chronic tender red nodules and tiny draining sinuses in the creases at the bases of the fingers and in the interdigital clefts. They may become acutely inflamed and require surgical drainage; they are cured only by removal of the offending hair.

INJURIES

Injuries to the hand account for 35 to 50 per cent of all wounds. When one considers the great economic importance of the hands

and the fact that all of us make our living with our hands the need for special attention to wounds and infections is evident. The very fact that the hands are our tools which come into contact with all the objects of our daily life subjects them to many hazards.

Injuries to the hand may be conveniently divided into sharp, clean, lacerated wounds, such as those caused by broken glass, knives and tin cans, and crushing, tearing, lacerating wounds (Fig. 26). This distinction between the "tidy" and the "untidy" injuries is of considerable significance insofar as treatment is concerned. In the sharp wounds, the damage to the tissues is confined to the actual line of division. The tissues themselves are not devitalized except insofar as vascular supply may be interfered with. With crushing and tearing wounds, there is the tissue division as seen in the sharply lacerated wounds, but added to this is the extensive damage to the tissues which extends beyond the area of actual tissue division or destruction. The significance of this difference is reflected in the manner of healing of the different types of wounds and the treatment afforded them.

The management of acute open wounds of the hand is based upon certain principles of care which apply to all open injuries. Since however many open injuries of the hand are often small, and to the uninitiated, simple there is a tendency to take too casual an attitude and not to afford such injuries the care they demand.

The care of open wounds of the hand begins with first aid and is not completed until the hand and patient have been restored to the greatest degree of function compatible with the injury. First aid should be simple and confined to measures which prevent secondary contamination of the wound, the control of bleeding and swelling and the insurance of rest of the injured part. The wound should be covered at once with a voluminous compression dressing bandaged on snugly but without constriction and a splint should be applied to put the hand or digit in the position of function (Fig. 27).

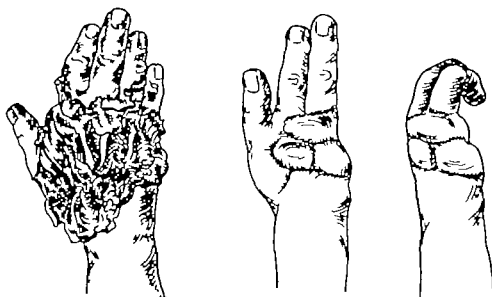
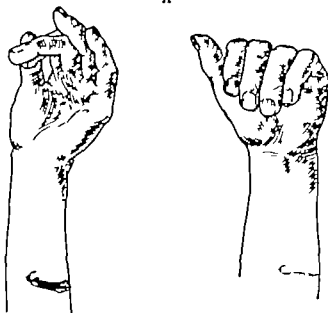
Control of bleeding is often overstressed to the detriment of the wound. Actually most bleeding is controlled by a firm compression dressing and elevation of the extremity. The use of a tourniquet should be restricted to those patients in whom compression will not work or as a purely temporary measure until compression can be applied. If a tourniquet is applied, it should be put on correctly, firmly enough, and not too tight.

and left on until the patient reaches a place where it can be removed under aseptic surgical conditions. This means the patient with a tourniquet must be transported at once to a hospital where the tourniquet is removed immediately. The practice of releasing a tourniquet from time to time on the journey to the hospital has resulted in fatal hemorrhages. The danger of shutting off the blood supply to the part by the tourniquet is not as great as that of compressing the nerves by the tourniquet, which may lead to paralysis from which recovery is very slow. Obviously, both dangers are im-

portant. Curiously enough it usually happens that a patient with a tourniquet is brought into the emergency room with the wound still bleeding. As soon as the tourniquet has been removed, the bleeding stops, indicating that the tourniquet has merely served to obstruct venous return and does not control arterial inflow.

When the patient is brought to the emergency room for care a history is secured at once. This history should include first of all the circumstances of the injury itself and then a general history of the patient. This latter is of particular importance in patients

A

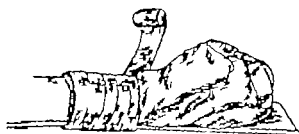


B

Figure 20 A convenient classification of wounds of the hands has been suggested by Rank and Wakefield. A, the tidy wounds are those produced by sharp-cutting implements which produce clean-cut injuries, B the mangled injuries, due to crushes, tears, abrasive burns, etc., are associated with extensive tissue damage, loss of covering skin, severe devitalization of tissues and, frequently extensive contamination.



Hand in physiological position.



Application of voluminous dressing and splint.



Strong wrapped.

Figure 27 First aid consists in the application of a voluminous compression dressing, wrapped on snugly enough to control bleeding, and a splint to put the hand at rest.

with extensive injuries requiring operative care under a general anesthetic.

The object of the history is obviously to assist the surgeon to care properly for the injury and the patient. The nature of the injury is of great importance, since wounds due to sharp objects such as knives and glass are different from those due to crushing forces, crushing abrasions and brush burns. Crushed, abraded and burned tissues heal poorly sloughing may occur if too great demands are put on them, while sharply lacerated wounds are frequently amenable to extensive primary repair.

It is essential to find out the degree of initial contamination of the wound. Initial contamination is usually not with immediately invasive bacteria but there are some instances in which virulent invasive bacteria may be immediately introduced on the traumatizing agent, for example, in a person working in a meat packing or butcher shop who cuts himself with a knife used to cut up meat, a plumber working in sewers or toilets, a pathologist working on a cadaver-

a person who sustains a tooth injury of the knuckles.

The interval following injury is also of importance. It is known that bacteria accidentally introduced into wounds require a certain amount of time to acclimatize themselves to life in human tissues. A certain latent period obtains which is usually assumed to be six to eight hours. A wound seen before the period of acclimatization is over can be assumed to be merely contaminated and not infected. After this time has elapsed however one should assume that the wound is infected. The extent of reparative surgery permitted in the wound which is simply contaminated is greater than that which may be carried out in a wound in which infection has already developed.

The nature of first aid and any other treatment previous to admission is also important. The surgeon is especially interested in discovering any possible secondary contamination or injury which the wound might have sustained subsequent to its incurrence. Was the wound exposed to contamination from human sources, e.g., droplet infection from nose and throat of patient, onlookers, doctors or nurses? Was the wound promptly covered so as to prevent this contamination? Was the patient taken to an office or elsewhere and treatment started under unsterile conditions? Occasionally a patient is received who has had an attempt at surgical repair at which time the wound was found to be more serious than expected, a dressing was applied and the patient was sent elsewhere for treatment. It is usually wise to assume under such circumstances that secondary contamination has occurred and that extensive repairs, especially of tendons, are contraindicated.

It happens not infrequently that previous operative procedures will make diagnosis difficult. The injection of a local anesthetic may produce an anesthesia which stimulates that due to a sensory nerve division. A reduction of an open dislocation of a joint may confuse the examiner who cannot make the diagnosis even on x-ray examination. This may be important in open dislocations, since the reduction may have carried dirt and contaminants into the joint cavity. The surgeon can never be sure of the conditions under which the previous operation was carried out and repairs which may be permissible in a wound not previously tampered with may be contraindicated because of possible contamination.

If simple first aid was not out, consid

ing only of covering of the wound with a sterile dressing, the possibilities of secondary contamination may be safely ruled out.

A general history of the patient is important if anything more than a simple laceration is present. The possibility of complications due to diabetes, hypertension and other general diseases cannot be overlooked.

Examination of the wound should be carried out under aseptic precautions. The surgeon, his assistants and the patient should be masked. Mouth and nose organisms are easily introduced by droplet contamination and, being acclimatized, may lead to more or less serious infection.

The wound itself should not be meddled with at this examination. Diagnosis of its extent and some idea as to need for preparation for skin grafting can be secured by observation without poking around in the wound with probes and forceps. Diagnosis of nerve and tendon division is made by testing for function of the wrist and fingers and this may be done with the wound covered.

A sufficiently accurate diagnosis of sensory nerve division may be secured by touching the patient's finger tips gently with a wisp of cotton (Fig. 28). It should be remembered that not only must the examiner test

for the median and ulnar nerves, but, when injuries involve the palm and digits, each side of each digit must be tested. The individual common digital or lateral digital nerves may be involved. The examiner must always suspect the possibility of nerve involvement whenever a wound lies in a likely location. Motor functions of the median nerve may be tested by asking the patient to rotate and appose the thumb to the tip of the little finger. This tests the integrity of the motor branch of the median nerve to the thenar muscles. It must be recalled in this connection that a complete median nerve lesion may be present in the forearm and still the thenar muscles may not be paralyzed because of crossed innervation from the ulnar. The motor function of the ulnar nerve may be tested by requesting the patient to abduct and adduct the digits. This function is never taken over by the median nerve (Fig. 28).

By and large, the sensory findings are by far the most important, since, if the tests are carefully made and the patient is cooperative and intelligent, the findings are unequivocal. In children the examination is quite difficult and conclusions as to nerve division must often be made on probability.

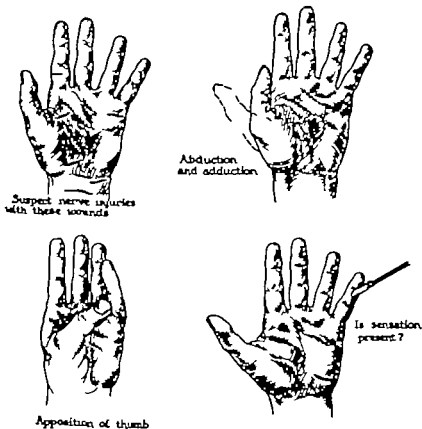


Figure 28. Diagnosis of nerve damage in acute injuries may be made by testing for light touch with a wisp of cotton applicator and testing for motion of the intrinsic muscles. Nerve damage must be suspected and archly sought for when wounds are present over the course of any of the nerves of the hand.

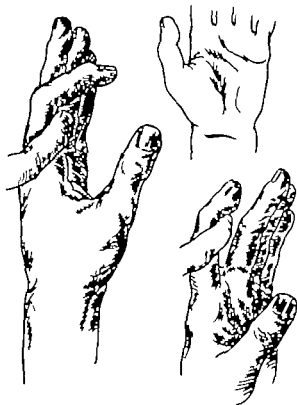


Figure 29 Diagnosis of tendon injury is made by testing motion of the fingers and wrist against slight resistance.

Examination for tendon division is made by testing the action of the fingers against slight resistance (Fig. 29). The patient is directed to flex each joint. It is sometimes difficult to make the diagnosis of sublimis tendon division in the presence of an intact profundus tendon although it will usually be noted that the initiation of flexion may be somewhat delayed. The metacarpophalangeal joints, being flexed by the interossei and lumbrical muscles, may act normally even if the tendons are divided, provided the median and, particularly the ulnar nerves are intact.

It should be remembered that it is possible to have a complete median and ulnar nerve division at the wrist and still have flexion of the digits if the tendons are intact, since innervation of the sublimis and profundus muscles comes from the nerve trunks in the forearm well above the wrist. The superficial position of the median nerve at the wrist, just beneath the palmaris longus tendon, renders it susceptible to injury and, if any tendons have been divided at the wrist, it is almost certain that the median nerve is involved.

X ray examination of the hand should be carried out in any patient in whom there is the slightest suspicion of fracture. Certainly

any hand with a crushing wound should have a roentgen ray examination, as should one with a saw cut or deep laceration.

Examination should not stop with the hand but a general examination of the patient must be carried out. Blood and urine tests are essential especially if a general anesthetic is to be administered. It must also not be forgotten that the patient may have other wounds which require care and, at times these may take priority over the wound of the hand.

It is rare for a wound of the hand to lead to sufficient hemorrhage to cause shock, however this possibility should be kept in mind and, if present or threatening, measures should be taken to combat or prevent it.

With the diagnosis made and all other necessary factors under control, the surgeon then proceeds to take care of the wound itself. This operative care must be given under strict operating room conditions, whether it be a small wound requiring simple cleansing and closure or an extensive wound demanding multiple nerve and tendon repair and requiring two to three hours of the surgeon's time. The surgeon scrubs and wears cap, gown and gloves whether he is putting in two sutures, laying a small graft on a finger tip or carrying out open reduction of several fractured metacarpals. Except for brief and small procedures a general anesthetic is preferable. The anesthesia need not be as deep as that required, for example, in abdominal surgery since the amount of relaxation needed is not so great.

The operation should be carried out in a bloodless field secured by means of a blood pressure cuff pumped up to 280 to 300 mm Hg. The cuff is applied rather loosely to the upper arm which is then elevated for a minute or so, and is then rapidly inflated to the desired pressure. A bloodless field has a number of advantages. It enables the surgeon to identify structures which ordinarily would be covered with blood. It obviates the necessity for constant time-consuming sponging and, by reducing sponging to a minimum it automatically reduces the amount of trauma to the tissues from coarse gauze. The blood pressure apparatus is left inflated until the initial phases of the operation have been carried out and all dissection has been accomplished. The cuff is then deflated. After maintaining sponge pressure on the wound for from five to ten minutes, the wound is examined and all bleeding points are clamped and ligated. This

accomplished, the arm is again elevated and the cuff reinflated. The operation is completed and a splint and compression dressing are applied before the cuff is finally released.

The first act in the surgical operation on the wound consists in a thorough cleansing of the hand and forearm and then of the wound itself (Fig. 30). This cleansing is carried out in the operating room under aseptic technique with the individual who is doing the washing scrubbed for surgery and wearing rubber gloves. It may be necessary to anesthetize the patient before the cleansing is carried out, although often cleansing may be done before the anesthetic has been started. For extensive wounds it is usually preferable to have the patient asleep. It may be necessary to have the blood pressure cuff inflated during the washing since this maneuver may loosen clots and bleeding may begin again.

Large cotton pledgets and liberal amounts of soap and water are used and the washing is done as carefully as the surgeon washes his own hands before operation. First, the wound is covered and the skin of the hand and forearm exclusive of the wound is carefully washed for some ten minutes. At the end of this time, the part is rinsed with warm sterile water. Then the dressings are removed from the wound and after the surgeon dons fresh gloves and obtains a fresh basin of soap and water the wound itself is carefully and gently washed. This is not a scrubbing with brushes but gentle washing

with well lathered cotton squares which are frequently changed. After ten minutes, the wound is thoroughly irrigated with warm normal saline solution and the part is ready for surgical draping. Under no circumstances are antiseptics used in the wound and they are not necessary on the surrounding skin.

The wound is now ready for careful study and for the first step in surgery which is excision of tissue so badly damaged by the trauma that it is certain to become necrotic. It is now that the surgeon first really realizes the significance of the distinction between the sharply lacerated and the crushed wound. In the former with little tissue damage very little tissue will require excision. In the crushing wounds however there will be flaps of crushed or brush-burned skin which must be excised, and muscle fascia and even digits so severely damaged that they cannot survive. Wound excision is a most important and most difficult procedure. Upon its careful performance will depend the healing of the wound. Excision must be thorough but not reckless; all tissue which will not survive is removed and, at this stage of the operation, all tissue which will live is retained. Considerable experience is required to make the decision to err on the side of conservatism may lead to serious wound disturbance and to even further tissue loss from infection. To err on the side of radicalism may lead to needless sacrifices of structures and even fingers which could have been saved with proper care.

Each tissue is taken in turn and all that will obviously succumb is sharply excised. Probably the greatest difficulty will be experienced in the care of the skin where two temptations present themselves. The first temptation is to be too conservative, since wide excision of skin will cause great difficulty in closure. The other temptation which must be resisted is excessive excision of skin particularly of irregular tabs and flaps which do not seem to be of any value, or sacrifice of skin from digits which must be amputated. In sharply lacerated wounds, very little skin excision is required. Often it is not necessary to remove any of the skin edge. In crushing injuries, however the judgment as to the amount to excise is somewhat difficult. The surgeon can get some idea as to the desired extent of excision from a knowledge of the severity of the injury. Heavy weights and crushes, severe abrasions and degloving injuries usually leave the skin not only torn loose from underlying and lateral blood supply but so

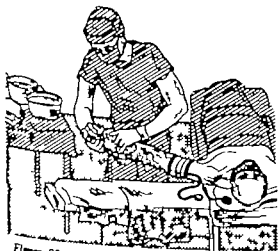


Figure 30 The surrounding area and then the wound are cleansed with soap and water under aseptic conditions. This process will require fully ten to fifteen minutes or longer and is not a perfunctory washing with soap water but careful, actual, thorough but gentle washing. No antiseptic is applied either to the wound or to the surrounding skin.

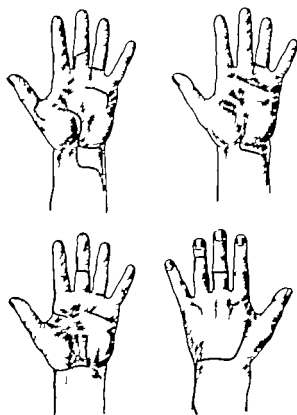


Figure 31 Certain general principles apply to the location of incisions on the hand. Incisions should avoid the midline; they should not cross flexion creases but should parallel them as closely as possible and should be planned so as to produce flaps of skin and subcutaneous tissue to overlie the operative area. The same principles apply to enlargement of accidental wounds which should be incorporated as well as possible into the general pattern of ideal incisions (from *Surg. Gynec. & Obst.*, vol. 70)

severely damaged that survival is impossible. It is a real error simply to replace this skin. It is likewise seldom feasible to attempt to use it later as a free graft since it is not only badly crushed but seriously soiled and contaminated. One can often get some idea as to viability of skin by watching the return flow of blood into it after the blood pressure cuff has been released.

Subcutaneous tissue and fascia also require removal and judgment here is likely to be very difficult. Muscle which is non-contractile and gray should be excised. Bones, however, are sparingly removed. Even tiny fragments, if they are connected with some connective tissue, are likely to survive and may be molded into the reconstruction of digits and metacarpals that have been badly damaged. Tendons are not excised unless the digits to which they are attached have been amputated or reconstruction is obviously impossible. Nerves are not sacrificed, regardless of their condition un-

less they too are destined for areas which are already gone or will likewise be sacrificed. If nerves are excised, care is taken to divide them high and away from the area of trauma so they may retract into healthy tissue, preferably muscle.

When wound excision has been completed, only viable tissue should remain so that the surgeon can consider then what reconstruction he can carry out. The wound is carefully surveyed and all structures are identified. It is wise at times to insert fine sutures into the nerves and tendons to permit easier exact and rapid recognition when the time comes for repair.

Decision as to the permissible amount of deep repair is made on several considerations. This concerns largely the matter of nerve and tendon repair since fracture reduction and closure of joints are indicated in every patient except those with already developed severe infection. The surgeon must be capable of carrying out this repair. He must know how to do it. He must have the patience to carry it out and the assistance and equipment to do so. It is not possible to speed up these repairs and unless a surgeon is willing to spend the time in doing so, he should not undertake surgery of the hand. Assistance is necessary. These are not procedures for a surgeon operating by himself in an emergency room. Proper instruments, fine needles, fine suture material, fine forceps and small retractors are needed, since the usual equipment of the general operating room is not suitable for this fine surgery.

It is never feasible to repair tendons over the site of a fracture since the repaired tendon will surely become adherent to it and the repair be useless. With badly crushed wounds, tendon repair is likely to prove a failure since the tissues heal poorly and adhesions are sure to develop. Likewise, with badly crushed wounds or extensively lacerated ones, the blood supply may be so precarious that the enlarging incision necessary to expose tendon stumps may be more than the tissues can withstand.

The matter of infection and contamination is another important factor. In wounds over six to eight hours old, it is wiser to conclude that one is dealing with a potentially infected rather than a contaminated wound and only under exceptional circumstances is tendon repair permissible. The site of tendon injury is also important. In general, one can be somewhat more liberal so far as time limits is concerned with

wounds of the dorsum of the hand than with those on the volar surface. This is because dorsal wounds involve tendons surrounded by paratenon rather than by synovial sheath. The paratenon-covered tendons do not retract as far as do the sheath-covered tendons and, hence, it is not necessary to make such a wide exposure to secure retracted stumps. On the digits, careful consideration should be given to indications for primary flexor tendon suture. Only under exceptionally favorable circumstances is primary repair to be undertaken and then only by one well skilled in the art.

Repair of the nerves should be carried out at the primary operation in all but grossly infected wounds. This too requires a special technique and is to be undertaken only by one who understands it and only if proper suture material is available. If proper repair is not possible, it is well to tack the nerve ends together with a fine suture passing only through the perineurium at the very tip of the divided ends. This will prevent retraction during healing.

Some types of wounds are so severely contaminated initially that repair or even closure, is out of the question. These include wounds inflicted by human teeth and wounds from objects similarly contaminated such as autopsy knives.

While the surgeon is considering the feasibility of primary nerve and tendon repair the wound is covered with warm compresses and firm pressure is applied. The blood pressure cuff is then deflated and manual compression is maintained for from five to ten minutes. During this time, the hand first becomes hyperemic and then this slowly recedes. When this happens the compresses are removed and bleeders are caught and ligated with firm, nonabsorbable ligatures. As soon as bleeding has been controlled, the arm is again elevated for a minute or so and the cuff is again inflated to 280 to 300 mm Hg and the operation completed.

One further consideration should be in the surgeon's mind when excising wounds of the hand. Many tissues of the hand may be quite valuable for later reconstruction. An obviously irreparable digit may form a valuable "bank of material for this purpose.

If deep repair has been decided upon the tendons are usually repaired first (Fig. 32) then the nerves, although the order of repair will depend somewhat upon the individual case. Since the hand and fingers must be held in such a position as to relax the structures which it is sought to repair the surgeon

may be at some disadvantage if more deeply lying structures are not repaired first.

Following whatever deep repair is indicated the next step consists in the closure of the wound. Here again the surgeon is called upon to exercise judgment since, in wounds already infected when first seen or wounds contaminated initially by virulent invasive organisms closure at first instance is not permissible. All other wounds, however are to be closed primarily. Lacerations and wounds in which there has been minimal skin loss may be closed by suture. When it comes to closure the surgeon will be happy if he has not sacrificed skin unnecessarily but has saved even irregular tags and flaps. These may often be dovetailed into each other to secure cover and will often serve well since hand skin is best for closure of the hand (Fig. 33). In instances in which digits may require amputation because of irreparable damage the skin of much of the digit may often be left attached and drawn into the palm or over the dorsum to cover defects which would otherwise have to be covered by means of grafts.

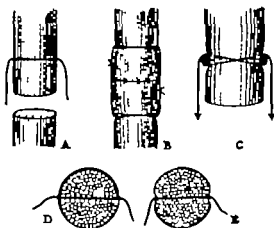


Figure 32. Tendon repair is carried out with the finest possible nonabsorbable sutures, the tendon is handled with extreme care to avoid traumatizing the delicate tissue.



Figure 33. Wound closure by suture is carried out wherever it can be accomplished without tension. The dovetailing of irregular flaps will often secure adequate closure with hand skin, which is always preferable.

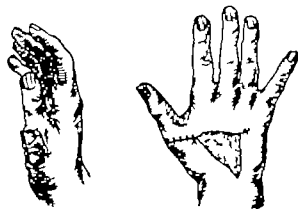


Figure 34 When bones, joints and tendons are exposed, rotation flaps may be used to cover the area and the bed from which the rotation flap is raised is covered with a split graft.

If the wound cannot be closed by suture, some type of skin graft is indicated. The type of graft used will depend upon the bed upon which it is to be placed. If the bed is composed of soft tissues, it is usually possible to lay a split thickness skin graft over it and secure satisfactory healing. If raw bone, joints or bare tendons are exposed in the wound, however, it is not possible to lay free skin grafts over the surface. Instead, a flap of skin and subcutaneous tissue carrying in its own blood supply is indicated. This may be accomplished in one of several ways.

If the defect occurs on the dorsum of the

hand, it is often possible to rotate a flap of skin from the side of the defect and bring it over the bone or tendon. The bed from which this flap is raised is then covered with a split graft (Fig. 34).

At other times there is no possibility of bringing a flap in from the side of the wound and, in these instances, it is necessary to raise a flap from some other part of the body usually the abdomen and to place the hand under it (Fig. 35).

Following closure of the wound, the hand or part is placed in a voluminous compression dressing on a splint in the position of function (Fig. 36). If only one finger is injured, only that digit is immobilized; if several fingers are involved, the entire hand is put at rest under a compression dressing in the functional or grasping position. Care is taken to keep the fingers separated by gauze so that maceration does not occur where skin surface lies against skin surface.

The compression dressing helps to reduce the swelling which always accompanies injury and which is such a factor in the subsequent stiffness which may occur. Compression likewise helps to control the capillary oozing which, if unchecked, may separate apposed surfaces despite every effort at adequate hemostasis at the time of operation. Compression obliterates dead spaces and

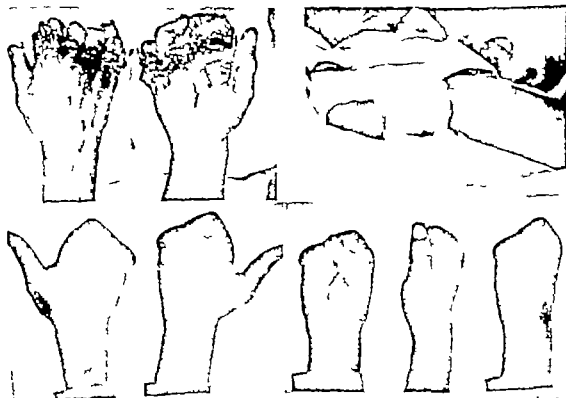


Figure 35. A pedunculated flap may be needed when there has been great loss covering these.

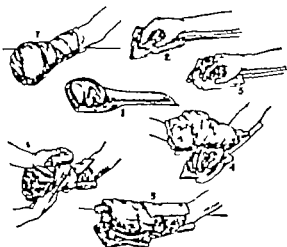


Figure 36 A volutinous compression dressing is useful in all phases of surgery of the hand. When it is desirable to cover the whole hand, it is put at rest on a splint (1) in the position of function (2) the fingers are separated by stuffed gauze (3) and a large amount of resilient dressing is laid over the hand (4 and 5) This is then bandaged on firmly and evenly without producing constriction (6 and 7)

brings the tissues into correct relationship and under normal tension. The dressing will also hold fractured bones in position a feature especially valuable in the severe crushes in which the surgeon molds the hand into the position of function over a suitable splint. The dressing is applied before the blood pressure cuff is finally released so that the hand is not engorged with blood in the reactive hyperemia which follows sphygmomanometer release.

Finger Tip Amputations. Preservation of the length of digits is important in preserving function of the hand. A common injury is an amputation of a segment from the tip of a finger. Since it is not possible to close these wounds by suture, a common temptation is to amputate the bone higher up in order to procure flaps to cover over the tip. This produces a good stump but necessitates sacrifice of length. The preferred procedure is to cover the amputated tip with a split thickness skin graft taken usually from the



Figure 37 Amputations through the finger tips may usually be closed with small split grafts taken from the for-

volar surface of the forearm (Fig. 37) a procedure which can be done with the patient under local anesthesia and without special equipment.

Abrasive Burns of the Hand. These injuries are frequently due to a worker catching his hand in a pulley belt or to falls from moving vehicles. Areas of skin usually from the dorsum, are either completely removed or so badly crushed and affected by heat that they cannot survive. Such areas should be immediately excised and replaced at once with split grafts (Fig. 38).

Extensive Crushing Injuries. These present some of the most difficult surgical problems. At first glance many of these wounds look as if nothing could be done in the way of repair and if a pessimistic attitude is assumed needless sacrifice of tissue may result. It is frequently not possible to assess these injuries until the hand has been cleansed and after all nonviable tissue has been excised. Great care is taken in this excision to leave all viable tissue even though it may appear that much of it will be useless eventually. When complete excision has been accomplished, the surgeon then reviews what is left and tries to fashion from the viable remnants as functional a hand as the tissues left by the trauma will permit.

The approach to the problem is to restore function, not appearance. To make the decision on function the surgeon must understand what the functions of the hand are. Decision in such cases must be made on the basis of the most essential or primary functions. The finer functions and motions may eventually be restored, but, in cases of this type effort is first made to restore the basic ones of sensation, grasp and pinch.

If these principles are followed in the crushing injuries it follows that in all instances nerve repair should be carried out, since a hand without sensation is almost useless. Restoration of grasp and pinch may seem hopeless, especially in the presence of multiple tendon injuries which must await secondary repair. Loss of skin may be extensive and repairs of tendons must await not only healing of the wound, but also the application of a pedicled flap under which tendon repair may be safely undertaken. Multiple fractures and loss of bone and digits may leave a poor framework and little to work with. However the surgeon need only remember that molding the hand or its remnants into the position of function will give the part the best chance of function. This molding will bring fractures into alignment and digits or their remnants into the grasp-

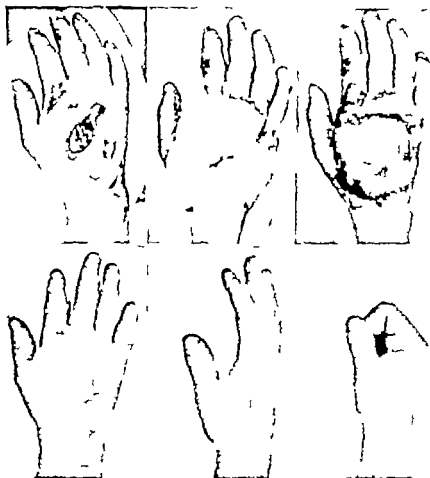


Figure 39 Abrasive or brush burns often lead to damage over large areas. Such badly damaged skin is irreparable and must be adequately excised and the defect covered by a split-skin graft.

ing position. With the hand brought into this position, the surgeon will be able to visualize the functional possibilities of the remnants and can utilize flaps to the best advantage. If no more than closure can be accomplished with fractures reduced and the hand in the position of function, a great step will have been achieved.

Later flaps of skin may be applied to replace skin grafts and to make a suitable bed for tendon repair. If such are needed, a hand in the position of function makes the best possible starting point.

Tendon Injuries. Repair of divided or ruptured tendons of the hand is based upon a knowledge of tendon healing, an appreciation of the delicate structure of the tissues to be dealt with and a willingness to follow a strict and careful technique in handling these tissues. It is not a question of simply obtaining union of tendon to tendon or tendon to bone. This will occur quite readily if these structures are kept in contact for a sufficiently long time. It is a matter of obtaining union where it is wanted and free gliding along the remainder of the course. It is a

matter of obtaining primary reactionless healing at all stages of surgery of maintaining mobility of joints which stiffen with rapidity when they are kept immobile. It is a matter of knowing when to start motion and how and when to encourage active and purposeful use—when to permit full function. The surgeon must continually watch, encourage and, if necessary chastise his patients. He must not permit his own patience to be worn down in the process.

When severed tendons are first brought together or when tendons are sutured to bone, a definite healing process begins which may conveniently be divided into four phases (Figs. 39 and 40). The first, or exudative phase of union is characterized by an outpouring of body fluids at the site of union. This exudate soon coagulates into fibrin which glues the ends of the tendons together rather weakly so that they can be separated by very slight pull. Soon, however this fibrinous area is infiltrated by fibroblasts which come from various sources, more especially at the onset by fibroblasts from the surrounding tissues and sheath elements of the tendon.

This fibroblastic union forms the second phase of repair and while it is much stronger than the fibrinous phase, it is still quite weak and reacts to motion by becoming more and more prolific and may produce heavy callus which may be palpable. Obviously if such callus lies near to bone or fibrous tissue it will become adherent to it and the subsequent gliding of the tendon is seriously jeopardized or even completely prevented. Toward the end of the third week, the tendon callus begins to soften up about the tendon, while that part which lies between the tendon ends become more dense and organized, its cells and collagen fibers tend to line up in the direction of tendon pull. Toward the end of the third week, the union has become quite strong and will withstand a fair pull without rupturing. Indeed it would seem at this phase of the healing process some motion is helpful and tends to lead to

strengthening of the union. The final phase of healing begins at this time and is known as maturation. At the start motion and use must be restricted for a week or ten days and probably strenuous use and sudden forceful tensions are best avoided until the end of the fifth or sixth week after repair. Maturation lasts for many weeks or months until the site of union has become as strong as or stronger than the normal tendon. It is always possible to detect the site of union, or the tendon graft which has been put in since the new tendon never regains entirely the glossy sheen of the normal tendon. Microscopically however little difference between them may be detected.

Relaxation of the sutured tendon is necessary to prevent separation of the tendon ends during the early stages of repair since the union at this time is weak and easily separated. During the second ten days following suture strenuous use will lead to separation while even mild motion without much force may lead to increase in the size of the tendon callus and its consequent firm adherence to the surrounding tissues. Hence following tendon repair the parts are splinted in relaxation for a period of three weeks (Fig. 41) permitting no motion of any sort until the end of that time. In case of the flexor tendons the wrist is held flexed at about 30 degrees and the involved fingers in what would approximate the position of function, i.e., slightly flexed, more so at the

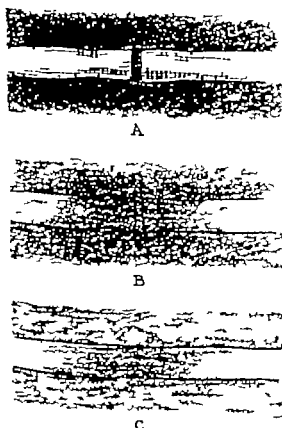


Figure 39 Diagrammatic representation of the manner of healing of a tendon. A, First week—proliferation of peritendinous and intratendinous connective tissue. B, Second week—tendon cell proliferation and obliteration of gap. C, Third to fourth week—consolidation of gap tissues and beginning stages in adaptation of surrounding tissues to subserve gliding function. From the fourth week on, and for many months thereafter the union undergoes slow changes of maturation resulting in reduced cellularity an increase in collagen and in tensile strength (Mason, M. L. Surg. Gynec. & Obst., vol. 70)

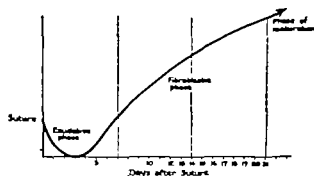


Figure 40 Diagrammatic representation of tensile strength of sutured tendons following suture. When first sutured, the tendons are held together by the sutures only. However very soon the tendon ends soften in the process of repair and the sutures tend to pull out. Union during this phase is due only to fibrinous exudate. Not until the seventh day when fibrous proliferation has started, has the line of union approached in tensile strength that equal to the holding power of sutures alone. From then on, a gradual increase in strength occurs and by the nineteenth to twenty-first day is sufficient to permit some active but guarded use. Thereafter increase in strength, as a process of maturation, continues over a period of many months.

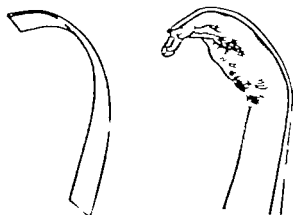


Figure 41. Following suture of the flexor tendons, the hand is splinted in flexion so as to relax the suture line for a period of three weeks.

metacarpophalangeal joints than at the interphalangeal joints. At the end of three weeks, the wrist is kept flexed on a splint and the fingers are provided with check reins which permit flexion but prevent more than just slight extension. This small splint and the check reins are worn for a week to ten days during which time the patient is permitted to flex the digits and mobilize them actively. After about ten days of this restricted motion, the splint is removed entirely and the patient is permitted and encouraged to use his hand in his daily life activities. Strenuous duties are however not permitted for another couple of weeks. When dressings and splints have been removed, the patient is instructed in "daily soaks." These are not just soaking of the hand in warm water but consist in warm soapy hand washes, using a cloth or simply washing one hand against the other for four to five minutes, four to five times daily. Both hands are washed since it is the motion of the fingers and intrinsic muscles of the hand which act on the vessels to pump the blood through the tissues and upward into the forearm veins. It must be stressed to the patient that perfunctory fluttering of the fingers and waving them in the air is not accomplishing anything. The patient is told to feel the flexor muscles of his forearm and to make them hard by contracting them forcefully.

Splinting following repair of extensor tendons (Fig. 42) is, of course just the opposite of that for flexor tendons. In this instance the wrist is dorsiflexed about 40 degrees and the fingers are extended. However one must avoid holding the metacarpophalangeal joint out to complete 180-degree extension, since this joint stiffens quickly at complete extension or hyperextension and correction is difficult. The finger joints, likewise, are seldom

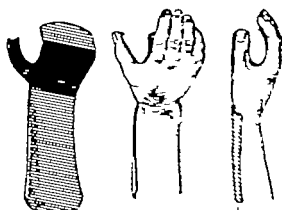


Figure 42. Following suture of the extensor tendons, the hand is splinted in extension for three to four weeks.

splinted in 180 degrees of extension unless the tendon division has occurred on the digit itself i.e., over the phalanges. In case of the thumb the digit is abducted and usually extended fully.

Immobilization of the extensor tendons is carried out somewhat longer than is immobilization of the flexors—usually three and a half to four weeks for complete immobilization and then another week with restricted motion. Following this, the patient is fitted with a padded splint with straps which he wears at night for a few weeks. The night splint is necessary for the extensor tendons since the constant flexion, often strong, which occurs during sleep may lead to stretching of the site of union and loss of a certain amount of extension.

Rupture of the Extensor Pollicis Longus Tendon. Rupture of this tendon was described many years ago as drummer's palsy since it was seen so frequently in drummers in the German army. After years of drumming, these men would suddenly lose the power of extension of a thumb. The condition would come on painless but completely and no amount of conservative therapy would cure it. It was assumed for some time that it was an isolated paralysis of the extensor pollicis longus muscle of unknown cause. However when some bold surgeon cut down on one of the tendons, it was discovered that the condition was actually a rupture of the tendon in an area of attrition along that part of the tendon which moved back and forth over the lower end of the radius, obliquely transverse across the wrist and finally distally to the thumb. Age seems to be of some significance in the development of spontaneous rupture since the circulation of the tendon becomes less as the tendon ages.

Similar rupture of this tendon is seen infrequently following Colles fracture. It comes on suddenly in the second or third week and is thought to be due to irritative damage to the tendon as it passes over the site of fracture. It is a condition inherent in the fracture and has nothing to do with the reduction or manipulation. It may occur in instances in which reduction is perfect and has been accomplished with little trauma.

The findings are perfectly typical of loss of function of the extensor pollicis longus tendon (Fig. 43). The thumb droops for ward in flexion at the metacarpophalangeal and interphalangeal joints and the patient is unable to extend it. Although the extensor brevis and abductor longus are functioning, the great significance of the extensor pollicis longus in all motions having to do with extension and abduction is clearly revealed in this injury. When there is doubt as to the diagnosis, the crucial test is extension of the distal phalanx, which is not possible in loss of the extensor longus.

Treatment of the condition is operative. No amount of splinting will restore function. Suture of the tendon is seldom possible or feasible. The ends are degenerated and repair is impossible. It is quite possible to

insert a tendon graft between the distal stump at the base of the metacarpal thread it through the proper tunnel and then attach it to the proximal stump. However this is a formidable procedure. The proximal stump is difficult to find and requires an extensive dissection in the forearm. Besides these disadvantages, the tendon graft would have to run through a tortuous tunnel with rigid walls and stands a good chance of being stuck therein.

A procedure which fortunately works out well because of availability of tendons is that of tendon transference. There are two radial extensors to the wrist, either one of which can be detached and transferred to the distal stump of the extensor pollicis longus. In some patients, however this transfer has been followed by a spastic condition of the thumb in which attempts at flexion are accompanied by extensor spasm. This was assumed to be due to differences in excursion of the two tendons, and such a difference does exist. The extensor pollicis longus has an excursion of 5.5 to 6.0 cm. the extensor carpi radialis brevis and longus, 3.5 to 4.0 cm. Consequently another double extensor supply has lately been utilized, namely the extensor indicis proprius. Curiously

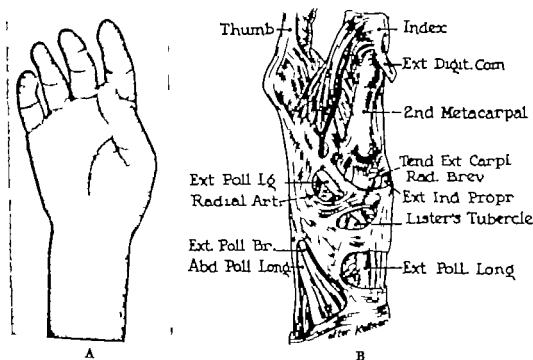


Figure 43. A, Isolated rupture of the extensor pollicis longus tendon occurs occasionally following Colles fracture. It was at one time seen frequently in drummers in the German army and was shown to be due to striction of the tendon due to repetitive trauma. B, The complex course of the extensor pollicis longus explains the liability of this tendon to damage at times of Colles fracture and also illustrates the difficulties of suture and repair following injury. The close relationship of the extensor indicis proprius to the extensor pollicis longus makes tendon transfer a feasible solution to the problem (Kaplan, E. B., *Functional and Surgical Anatomy of the Hand*, J. B. Lippincott Company).

enough, although this tendon has an excursion of 3.0 cm. or less, it seems to function satisfactorily.

After-care for tendon transference consists in splinting of the thumb in the position of abduction and extension for three weeks, then application of a removable splint which the patient takes off for a few hours a day for the next two weeks and wears at night for another two weeks. Any vigorous use of the thumb is prohibited for at least six weeks.

Baseball Finger (Drop Phalangette Mallet Finger) The insertion of the extensor tendon into the base of the distal phalanx of the finger is extremely thin and is frequently torn when the extended tip is forcibly flexed. This is seen especially in baseball players when a ball lands on the tip of an outstretched finger and flexes it acutely (Fig. 44). One of two things may happen—the tendon tears across, usually at the level of the joint, or an avulsion fracture occurs with the bone of insertion fracturing with the pull of the tendon. It is wise, therefore in these circumstances to secure x ray films to determine if there has been a fracture.

The deformity resulting from the injury is quite typical and unmistakable. The distal phalanx drops forward in flexion and cannot be actively extended, producing a dropped finger tip. It can be pushed back into extension but drops down immediately to an angle of 125 degrees or less as soon as released. The finger flexes normally but when the active flexion ceases, the finger comes back to about 125 degrees. This must not be confused with active extension, it is simply the angle which the tip assumes at rest.

Treatment of the condition varies with the pathologic condition present with the probable extent of the damage and with the time elapsed since the injury. When there has been avulsion of the tendon with a chip of bone from the phalanx, healing will often occur satisfactorily following splinting of the finger in extension, with the distal phalanx in hyperextension. This must be maintained for at least five weeks and the patient will have to wear a splint at night for several weeks longer.

For rupture of the tendon without bone avulsion, splinting is likewise often successful, especially if the patient is seen quite early after the injury and if he wears the splint continuously for a period of five weeks and at night for a further two to three weeks.

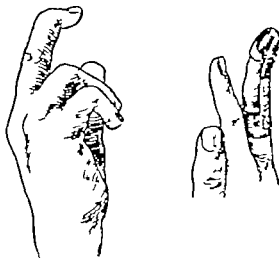


Figure 44 Baseball, or mallet, finger is a characteristic deformity due to rupture or avulsion of the extensor insertion into the base of the distal phalanx.

The tendon heals quite rapidly; however it is tissue-paper thin at the site of rupture and the thin callus stretches easily so that even though a full extension may be present early the tip will droop later. It is sometimes difficult to prevent a certain amount of dropped tip even under the best of conditions. Operative repair in the early case is of some advantage since it permits the surgeon to remove bits of capsule and tendon from the joint cavity which may have fallen into it. The period of immobilization following operative repair must be just as long as after simple splinting.

When the condition is seen late an open repair may be necessary since the gap between the torn ends of the tendon fills up quickly with callus and splinting in extension cannot be expected to shorten this callus. The open procedure consists in tendon shortening or rather callus shortening, since the distal stump is short and thin. The callus is divided obliquely in a longitudinal fashion and then with the finger in maximum extension, the two flaps are overlapped and tacked together with a few fine sutures. The essential step is to maintain the hyperextension since the sutures themselves serve little function after the first day. In fact, the secret of all tendon repair is the maintenance of relaxation and, in this particular instance the importance of this is markedly emphasized. At times, it may seem indicated to put a tendon graft into the digit to secure more stable union.

After-care following operation is the same as the treatment of the acute injury, namely, continuous uninterrupted extension of the finger for a minimum of five weeks and a

splint at night for a further two to three weeks.

Buttonhole Dislocation. This descriptive term applies actually to the typical deformity following the division of the central slip of the extensor usually just distal to the proximal interphalangeal joint (Fig. 45). In order to understand the deformity produced by this division and to rationalize its treatment it is necessary to understand the anatomic arrangement obtaining with regard to the insertion of the extensor tendons on the digits.

The common and proper extensors of the index, middle, ring and little fingers originate in the forearm and after passing through their tunnels on the dorsum of the wrist reach the region of the metacarpophalangeal joints. Here they pass over the joint capsule attaching to it to greater or lesser extent. Certain of the fibers attach to the base of the proximal phalanx where they serve as extensors of the joint. The capsule and these tendon slips form what is known as the extensor cuff. Approaching the cuff from the sides, and between the metacarpal heads, are the

tendons of the lumbrical and interosseus muscles which form the so-called lateral slips of the extensor aponeurosis. These slips however become attached to the central slip or that portion of the extensor aponeurosis coming from the common extensor tendons, distal to the metacarpophalangeal joint, passing along the side of the proximal phalanx and proximal interphalangeal joint to reach the dorsum of the finger. These lateral slips come together just distal to the proximal interphalangeal joint and then proceed as a single band distally to insert in the base of the distal phalanx. The central slip in the meantime has inserted into the base of the middle phalanx where the majority of its fibers stop.

This arrangement accounts for the fact that the lumbrical muscles and interosseus muscles act as flexors of the metacarpophalangeal joint and extend the interphalangeal joints. It also accounts for the fact that the central slips coming from the common and proper extensors extend the metacarpophalangeal and proximal interphalangeal joints but do not extend the distal interphalangeal joint unless the whole extensor expansion is put under tension by flexion of the wrist and flexion of the metacarpophalangeal joints. When the central slip of the extensor aponeurosis has been divided, the proximal interphalangeal joint drops down into flexion and the extensor communis pulls upward or retracts. At the same time, the lateral slips become tense and hence they exert pull on the distal phalanx and draw this joint into extension. At the same time with the central slip divided, the lateral slips tend to dislocate forward, thus accentuating the flexion of the proximal interphalangeal joint which then projects dorsally between the slips. A typical deformity is thus produced. The proximal interphalangeal joint tends to become more and more acutely flexed and the distal joint goes into more and more extension and eventually becomes hyperextended. This deformity is called buttonhole dislocation since the joint, while not actually dislocated, projects dorsally between the lateral slips of the extensor aponeurosis.

This deformity occurs usually from an open division of the central slip across the dorsum of the proximal interphalangeal joint. Repair of this injury entails repair of the divided tendon, which is not as simple as it sounds since the situation is usually not seen early and contracture has already occurred. It is usually necessary to bring the lateral slips forward as well as to repair the central

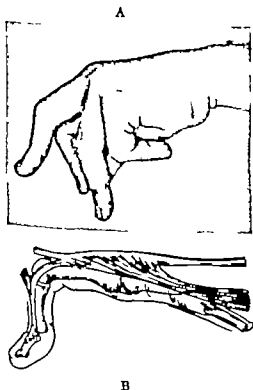


Figure 45. A, Division of the central slip of the extensor aponeurosis produces a typical deformity known as buttonhole dislocation. The proximal interphalangeal joint is flexed and the distal interphalangeal joint is hyperextended. B, Diagram adapted from Kaplan to show the mechanism of buttonhole dislocation (redrawn from Kaplan, E. B.: *Functional and Surgical Anatomy of the hand*. J. B. Lippincott Company).

slip For this procedure, tendon grafts are often necessary

It is sometimes possible in the early case to secure satisfactory healing by splinting of the proximal interphalangeal joint in extension with a splint which permits some flexion of the distal interphalangeal joint. If a patient is seen late, this same splinting should be carried out to correct the deformity before operation. In some instances, even when the patient is seen late this splinting may correct the condition

Rupture of the Flexor Tendons. Flexor tendons rarely rupture. In certain types of injuries, the tendon may pull out along with a digit, tearing through at the musculotendinous junction. Rarely a flexor profundus will rupture through the insertion into the distal phalanx. When it does so it will retract variable distances up the sheath, often restricted by the vincula. Symptoms are those one would expect from loss of the proper flexor of the distal phalanx. Repair usually entails a tendon graft since, except in early cases, it will be impossible to bring the stump distally and reinsert it into the distal phalanx.

Grease-Gun Injuries. These injuries are purely a product of the machine age, although similar lesions pathologically were produced in the early days of cosmetic surgery by the introduction of paraffin for the rounding out of contours and the building up of noses and breasts. Similar pathologic tumors were caused, in the old Russian and German armies by recruits seeking to avoid military service by the injection of various oils to cause tumors. Occasionally the injection of medicaments in oil, especially if given subcutaneously will lead to tumors (oleomas).

Grease from such guns, with small nozzle opening and very high pressure may penetrate the tissues leaving little or no mark of their entrance. The grease under great pressure is forced into the tissues, filling all spaces with an irritant foreign material. If the grease enters the tendon sheaths, they are completely filled.

The immediate reaction is a severe inflammatory process associated with moderate pain. Subsequently the wound of entrance may open and a purulent greasy discharge develops. Later on a hard, fibrotic sarcoma-like mass forms in all the involved tissues and may lead to complete disability of the part. The tumor tends to grow slowly and take over large areas. Although not malignant at the start, it may become so. When

first seen efforts should be made to remove as much grease as possible by adequate incisions, which should be left open to drain grease which will still soak out on the dressings. If seen later when tumor formation has occurred, extensive excision is often called for and various plastic procedures may be needed to reconstruct the hand.

BURNS

While the principles of care are the same wherever a burn may occur there are certain special features in burns of the hand which require emphasis

It cannot be stressed too strongly that, from the very start of care, it is important to place the hand in the position of function and maintain it on a splint under a compression dressing until healing has been secured. The hand goes so easily into a vicious position if great precautions are not taken to prevent it that this must be a constant care of the surgeon attending a patient with a burned hand. A compression dressing is of importance for although this does not entirely prevent the formation of swelling and edema, it does tend to minimize it. Much of the stiffness of the hand which occurs after burns is due to the organization of the exudate which comes with burns. If this exudate can be reduced in amount and, if the hand can be kept in the position of function, much of the disability following burns will be avoided.

A good result following care of a burned hand depends on securing early healing. The longer sloughs are allowed to remain and the longer raw surfaces are allowed to stay open, the greater will be the scar.

Many deep second degree burns of the hand become converted into third degree burns by infection. Hence every means possible should be taken to prevent the development of infection.

Burns of the dorsum of the hand especially even if not full thickness, tend to heal with thick keloid scars which contract and form an armor like covering which seriously interferes with function.

Full thickness burns of the hand should be excised early and the raw surface covered with split grafts. These grafts will seldom be the definitive cover for these areas, probably because it is not possible to carry out a complete excision in depth and in extent of the burn. However they will secure early healing and will minimize deep scarring. Later on it will be possible to excise these areas and apply more suitable

Dorsal burns of the hand especially when the fingers are affected tend to expose the tendons and the interphalangeal joints especially the proximal joints. This unfortunate condition may result when the burn originally perhaps deep second, becomes converted into third degree or full thickness loss from infection. It is therefore especially important to get rid of slough or destroyed areas as early as possible and to secure healing.

Maintenance of the position of function is not as easy to accomplish as it may seem when deep burns and massive destruction are present. It is, however, not enough to keep the hand in the position of function. Unless the surgeon also secures early coverage simply splinting the hand is of no avail since the raw surfaces pile up granulations and the scar becomes thicker and more contractile.

Further care of the deep destructive burn with loss of tendons, joint fusions and severe contractures consists in the provision of adequate covering tissue, in securing the position of function and in providing as much motion to the hand as the contracture permits.

VON VOLKMANN'S ISCHEMIC CONTRACTURE

This crippling condition when fully developed is characterized by necrosis, followed by fibrous replacement of variable extent of the flexor muscles of the forearm and wrist, with subsequent contracture of the tendons leading to flexion deformity of the wrist and hand (Fig. 46). Associated with it are varying degrees of nerve involvement, particularly of the median but also of the ulnar nerve, and general wasting of the extremity from the elbow down. It is most frequently seen following supracondylar fractures particularly those treated in flexion with tight casts, but may occur without any of these etiologic factors. A similar muscular condition may occur in the small muscles of the hand alone, often due to constricting bandages and leading to a typical contracture deformity from shortening of the interossei.

The pathogenesis of the condition is not clear. In most instances it would appear that swelling due to hemorrhage and edema in an extremity which cannot expand, because of tightness of fascial compartments or tight dressings, or both, is the prime factor. However in those instances in which tight dressings are not present, and in which the mechanical factor of compression does not obtain, it is thought that arterial damage

over the fracture site or arterial spasm is present to account for the necrosis due to lack of blood supply. Considerable experimental work has failed to explain the condition satisfactorily. Brooks was convinced that the condition was due to interference with the outflow of blood caused by venous obstruction and he produced some ingenious experiments to prove his point. Subsequent workers have not been able to reproduce his results.

The situation with regard to the nerves is not clear. In some instances, the median and ulnar paralysis recovers gradually while the patient is undergoing conservative treatment. In other cases exploration of the forearm in patients in whom nerve recovery has failed to take place has shown the nerves to be reduced to scarred cords for a considerable area. In some patients the median nerve at the elbow is actually divided or compressed by bone fragments or is constricted by scar involving the pronator teres. The nerve condition however is not the cause of the necrosis but rather part of it or secondary to it.

The brachial artery likewise may be impinged on bony fragments at the elbow and its flow stopped. The significance of this in causing true von Volkmann's contracture is likewise not clear. Certainly arterial blockage is more likely to lead to gangrene than to the condition of von Volkmann's contracture.

The significance of compression is real, as evidenced by the beneficial effects of release of compression by division of the cast and straightening of the elbow.

The onset of von Volkmann's contracture is ushered in by severe pain in the forearm and hand, usually after reduction of a supracondylar fracture at the elbow. The radial pulse disappears and the hand becomes swollen and blue. If the situation is recognized early if tight dressings are removed and if the flexed elbow is straightened out, the initial symptoms may subside, the pulse return and the pain disappear. If however these symptoms are ignored, or if the patient is not under hourly surveillance irreparable damage results. The pain will slowly subside after reaching a peak, the fingers gradually contract down into flexion and the swelling recedes. When the cast is finally removed, areas of necrosis of skin and subcutaneous tissues of the forearm will usually be found. When this process has cleared, the typical deformity will be present.

When fully developed, the deformity pro-

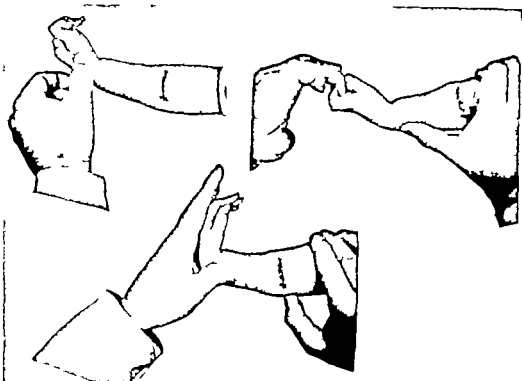


Figure 48 Von Volkmann's contracture is due to massive necrosis of the flexor muscles in the forearm, with variable degrees of median and ulnar nerve destruction. This produces the flexion contracture of wrist and fingers, adduction of thumb and pronation of the forearm, which are the unmistakable signs of this dread condition.

duces an unmistakable picture. The forearm is markedly wasted, especially the volar surface. The fingers are thin and atrophic and flexed at the interphalangeal joints. The wrist is held flexed and the thumb tightly adducted to the side of the hand. The fingers can usually be straightened if the wrist is flexed, but when one attempts to extend the wrist, the fingers flex tightly into the palm. The forearm is pronated and attempts to supinate it meet with resistance.

Sensation may be absent over the median and ulnar nerve distributions. Growth of the forearm and hand almost ceases and, since the condition is seen particularly in youngsters, it is not long before this difference in size is quite noticeable. The hand becomes an almost useless appendage. There may develop a modicum of flexion, but the range is limited and extension is restricted by the contracted flexor muscles.

Treatment depends upon the stage in which the condition is encountered. When the premonitory symptoms occur in the patient under treatment, immediate steps must be taken to release all constricting casts or dressings and to readjust the elbow to the point where the radial pulse returns. It may be necessary to attempt some other means of controlling the fracture, but, at the moment,

the most serious problem is that of combating the threatened von Volkmann's contracture.

If these conservative measures fail, the surgeon must consider releasing tension under the forearm fascia, by wide splitting of the fascia, leaving it open, although the skin may usually be closed. At this time, opportunity should be taken to inspect the brachial artery which may be damaged or in spasm. The spasm may release spontaneously or infiltration of Novocain in the area may effect release. The median nerve should likewise be inspected at this time.

When the contracture has become established, the treatment in any case is long and tedious. Corrective tension-splinting should be started very early. This is designed to prevent or correct the flexion contracture by exerting constant but gentle pull of the fingers and wrist into extension, to hold or pull the thumb into abduction and extension and rotate the forearm into supination. Numerous ingenious splints have been devised to accomplish this end. None is perfect and all require constant watching and attention. The pressure must be accurately adjusted and inspection must be frequent lest the patient develop pressure areas under the straps and elastic bands.

Splinting and physical therapy if started early and continued for a long time, may restore a hand to considerable function. However, it all depends upon the extent to which the muscles and nerves have been damaged. Obviously, if there are no living muscle fibers left in the flexor group, one can not hope for return of function. If, however, some motion can be detected in them, there is a chance that with use and a determined patient a fair amount of function can be developed.

If it is not possible to bring the digits out into a functional position and there appears to be a fair amount of muscle present which is too short, the surgeon may consider some means of compensating for this discrepancy. Several possibilities present themselves, such as detaching the flexor group at its origin and advancing it an inch or so down the forearm. Another possibility is to shorten the forearm by cutting segments out of the radius and ulna. It has even been suggested to remove one row of carpal bones. The general principle is to shorten the forearm or lengthen the tendons so as to permit the fingers to be extended.

In those patients in whom sensory and motor recovery does not occur after a reasonable time, it is advisable to explore the median and ulnar nerves and to release them from their constriction. This may or may not be successful. When no motor function can be detected in the entire flexor group, the only remedy is to attempt transfer of functioning tendons into the nonfunctioning flexors. Usually the extensors of the wrist are functioning and may be utilized for this purpose.

DUPUYTREN'S CONTRACTURE

In 1831, Baron Dupuytren of the Hôtel Dieu in Paris had the opportunity to dissect a hand which was the seat of a peculiar contracture causing flexion deformities of the digits. Although the condition was known before this time, and, although it would appear that Sir Astley Cooper had previously pointed out the exact site of the trouble, Dupuytren's dissection and careful description were definite and precise and the condition has been known by his name ever since.

Dupuytren's contracture, as originally described, is a condition involving the palmar aponeurosis and its prolongations and connections, leading to the formation of hard fibrous nodules and bands in the palm, interdigital clefts and digits and producing contractures of the skin and fingers (Fig. 47 and 48).

To understand Dupuytren's contracture, the anatomy of the fascia of the hand should be reviewed, particularly the palmar fascia (Fig. 49) which is the structure involved in the disease. Not only must one know the palmar fascia, but also the deep connections of this structure with the fibrous septa passing between the flexor tendons from the region of the metacarpophalangeal joints.

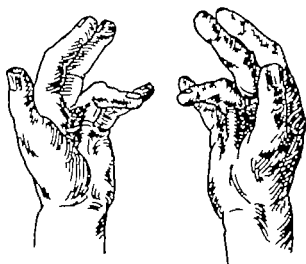


Figure 47. Typical Dupuytren's contracture—flexion of the ring finger, moderate flexion of little and middle fingers and a hard, fibrous band beneath the palmar skin.

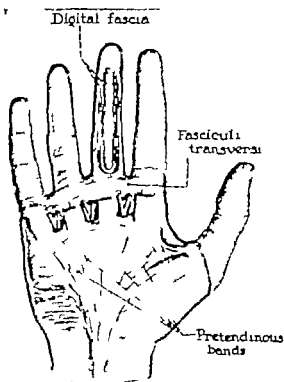


Figure 48. The superficial palmar fascia or palmar aponeurosis. This is the tissue involved in Dupuytren's contracture (Kusnetz, A. B., Koch, S. L., and Mason, M. L.: Surg. Gynec. & Obst., vol. 48).

fascia and to be attached to the proximal interphalangeal joint capsules.

One other anatomic fact is important when operative correction is undertaken. The skin of the palm is supplied by vessels which take origin from the common digital vessels in the palm and rise palmward between the tendons to perforate the fascia and reach the undersurface of the skin. Here they spread out for a variable distance to anastomose with other vessels from neighboring arteries. This anastomotic network, however, is not very well developed or extensive. The blood supply to the palmar skin therefore is easily jeopardized by the division of these vessels, especially so if the undersurface of the skin is badly contracted by fibrosis, such as occurs in Dupuytren's contracture. This jeopardy is further increased if following removal of the fascia and closure of the incision, a large hematoma forms under the skin creating compression of the fine anastomotic vessels on its undersurface.

The etiology of the condition is not known. Dupuytren thought that repeated trauma was the cause. His coachman who handled the whip and his wine handler whose hands were constantly subjected to injury from hoisting wine barrels, go trooping through the pages of history. With the advent of the automobile, the emphasis was shifted from the whip to the gear shift and the brake. Nowadays, with automatic gear shift, we still see Dupuytren's contracture in laborer and nonlaborer alike. In other words the cause of Dupuytren's contracture remains a mystery today as it did a century and a quarter ago when Dupuytren first described it. Many studies have been made to attempt to correlate Dupuytren's contracture with rheumatism, with cervical arthritis, with diseases of the spinal cord and sympathetic nervous system, with diabetes with repetitive trauma, but none has stood the rigid test of mathematical analysis. No single factor has shown itself predominant. The factor of hereditary influence has been demonstrated in a good percentage of the patients and this, while not universal, has been more consistent than any other. The association with epilepsy so clearly brought out by Skoog, is attractive and certainly there are many instances of this condition appearing in epileptic patients. From our observation, it can be said that Dupuytren's contracture is often seen in epileptic patients, but very rarely have we been requested to care for a patient with Dupuytren's contracture who gives a history of epilepsy. The same may be said

for age—the majority of patients with Dupuytren's contracture are fifty to sixty years of age, but it is seen in younger persons though not so frequently. So far as sex is concerned, most of the patients are males but it is not a disease of males alone and the studies of Stuart Gordon of Toronto seem to indicate that the figure of 10 to 15 per cent in females may be too low.

As far as trauma as an etiologic agent is concerned this seems to have the strongest support. However the evidence is equivocal and circumstantial. The history of trauma is by no means universally present, not even in an overwhelming majority of individuals afflicted. The hands obviously are the most subject to trauma of any part of the body so that one would be hard put to find a patient who would not give a history of some sort of trauma to the hands. Usually the condition begins in one hand, often the right. The patient may give a definite history of an acute injury years before or of excessive use of the hand with repeated trauma to the palm. This seems to fit the condition quite well until several years later the same condition develops in the opposite hand or is discovered there by the examining surgeon. Nor does Dupuytren's contracture occur only in individuals who do manual labor. It is seen quite frequently in persons whose severest work is the use of pen and pencil and whose only palmar trauma consists in pressure of the steering wheel or golf club.

Heredity is thought to play a role in the occurrence of the condition. It is found not infrequently in a family for two or three or more generations. It must be admitted that heredity is by no means always implicated, although it is surprising how many patients will deny it initially only to return later with the statement that upon inquiry they had discovered that some relative usually but not always, on the father's side had some sort of crippling condition of one or more fingers.

The gross pathologic state of the condition shows the pretendinous bands to be thickened and shortened (Fig. 50). Often, in the region over the metacarpophalangeal joints thick nodules will be seen in the bands—1 to 2 cm. in diameter. Similar thick nodules will be found in the volar surfaces of the digits when these are involved. As these bands shorten, they pull the overlying skin into folds and dimples. These are however by no means simple skin folds, as the surgeon soon discovers on attempting to free the skin from the underlying fascia. The fascia will

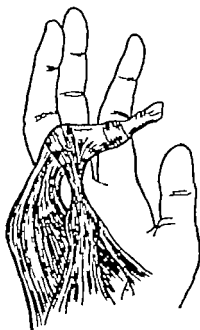


Figure 50 Gross pathology of Dupuytren's contracture (Adams, W., *Brit. M. J.*, 1878, vol. 1)

be found to be intimately attached to the overlying undersurface of the skin and separation will be difficult and, in some instances, impossible, so that sacrifice of the skin will be required.

As the fascia is pulled up away from the underlying tendons, it will be seen that the septa passing dorsalward to the sides of the metacarpals to either side of the tendons are likewise thickened and, as the fascia is followed farther distally the neurovascular bundle comes to be enclosed between two sheets. This portion of the fascia may likewise be involved and, when this obtains the nerves are drawn out of their course often to the midline of the digit.

The joints and tendons are not primarily involved in the process. Some compensatory shortening may occur and if the joints have been held for a long time in acute flexion, the capsules may contract and even a mild subluxation may be present.

Occasionally the patient will have pads on the dorsal surfaces of the proximal interphalangeal joints (Fig. 51). These, known as knuckle pads, are not frequently seen, nor is it generally realized that they are part and parcel of Dupuytren's contracture. Likewise, in a small percentage of patients, a nodular thickening of the plantar fascia will be found similar to that seen in the hand. In extremely rare instances plaques of similar nature will be found in the fibrous septum of the penis—Peyronie's disease. However Peyronie's disease is seldom seen in patients who apply for



Figure 51 Knuckle pads in a patient with Dupuytren's contracture (patient of S. L. Koch)

treatment for Dupuytren's contracture, although urologists say that patients with Peyronie's disease often suffer from Dupuytren's contracture.

Microscopically the thickened fascia is seen to be made up of very dense fibrous tissue scanty in blood vessels and nuclei. Here and there however one sees areas of increased vascularity and round cell infiltration. These areas have never yielded evidences of inflammation or bacteria. Skoog, who has written an excellent monograph on Dupuytren's contracture, thinks these areas represent healing of microscopic tears in the fascia. On this finding, he bases his contention that Dupuytren's contracture is due to trauma in the form of tiny repeated fascial tears which, upon healing, lead to scar formation with its attendant thickening and contracture of the fascia. After a careful histologic study Nézelof and Tublana could find no evidence to implicate trauma, tumor or inflammation. There is no evidence that the condition is a tumor although many contend that the process is a type of fibroma. Nerve fibers are scanty in the mass and show nothing unusual. The dorsal nodules and the plantar thickening reveal the same pathologic picture.

When once seen, the condition is always subsequently recognized. It starts out usually as a nodular thickening in the palm in the region of the distal palmar crease generally over the fourth metacarpophalangeal joint. The digits are involved in the following order of diminishing frequency ring, little, middle, index and thumb. Similar thickening also may occur in the web spaces. It is unusual for it to begin as nodules or bands in the digits themselves, either over the proximal phalanx or along the side of the finger.

As time goes on, the

more

prominent and a thickened hard cord may be felt to extend proximally and often distally mistaken by the uninitiated for the tendon. Soon, as this band thickens and shortens the proximal phalanx of the finger is drawn into flexion; at first, flexion is slight, but as the condition progresses the finger becomes more and more flexed, at both metacarpophalangeal and proximal interphalangeal joints. This may draw the finger down until the tip lies in the palm. In severe neglected cases, it may actually dig into the palm. The distal interphalangeal joint instead of going into flexion tends to remain extended or even hyperextended because of involvement of the dorsal expansion.

There are ordinarily few subjective symptoms other than the inconvenience caused by the contractures. Patients complain that it is difficult to shake hands to hold a golf stick or grasp a hammer or that they poke themselves in the eye while washing the face. Occasionally, one will complain of vague discomfort, rarely of actual pain.

Examination reveals the contractures and thickening in the palm and digits. In the very early case there may be only the nodule in the palm and the surgeon may be somewhat in doubt if he is dealing with an early Dupuytren's contracture. However careful palpation will often reveal the thickening of the pretendinous band. It can often be more easily demonstrated if the fingers are passively hyperextended. When this is done, the nodule will become slightly more prominent and the pretendinous band becomes evident. Likewise, at this time, the skin over the transverse carpal ligament between the closely opposed bases of the thenar and hypothenar eminences will be drawn distally as a triangular fold. If the process is of long duration and marked contracture has taken place, there may be maceration in the skin folds and between the digits.

The treatment for Dupuytren's contracture consists in operative excision of the palmar fascia. It is preferable to remove the whole of the palmar plate, even if only certain segments are involved. The digits are not opened unless there is actual palpable involvement. Removal of the palmar portion of the fascia is carried out in a bloodless field, since it is imperative that the surgeon visualize the nerves, blood vessels and tendons at all times. The palmar portion of the aponeurosis can usually be removed through an incision made transversely across the palm at the level of the distal transverse crease. The skin is carefully raised from the under-

lying fascia by sharp dissection. Unless the condition has been present for a long time and the skin is completely adherent, it is usually possible to save the skin which will live provided it has not been too badly traumatized if the blood supply has not been too seriously damaged and if hematoma is prevented from forming beneath it after operation. As the skin is raised proximally the surgeon looks for the perforating vessels which come up through the fascial plate to the undersurface of the skin. With care many of these vessels can be saved. When the skin has been dissected free proximally as far as the distal border of the transverse carpal ligament, the medial and lateral attachments along the hypothenar and thenar eminences are divided and then one cuts across the origin of the fascia from the ligament. The fascial plate may then be drawn distally to about the region of the proximal palmar crease. Here the fascia sends down its vertical longitudinal sheets to either side of the underlying tendons. The nerves and blood vessels have been in view at all times up to now. From here on special pains must be taken to visualize the neurovascular bundle. The surgeon identifies the nerve and divides the fascia only when both it and the nerve are under visual control. This visualization becomes increasingly difficult as the web spaces are approached and it is just here that the fascia may pull the nerve far out of position. If the digits are not involved, the excision of the fascia stops at, or just distal to the proximal end of the digital tunnel.

If the digits are involved it is necessary to enter them and remove the fascia. The first task, after entering the finger is to identify the digital nerves on either side before making any attack whatever on the fascia itself.

The incisions made to remove the fascia are meticulously closed after bleeding points are controlled. This is ascertained by releasing the blood pressure cuff which has been securing the bloodless field. As soon as all bleeding points are secured and ligated, the cuff is again inflated and the incisions are closed. If it has been necessary to remove skin the defect left by this excision is filled with a free full thickness skin graft.

The most serious complication likely to follow this operation is the formation of a hematoma in the palm. This will lift up the already poorly vascularized palmar skin and necrosis is certain to follow. The postoperative course will be unduly prolonged and the

recovery poor. To avoid this complication the palmar dressing is applied firmly with large amounts of resilient gauze. It is usually advantageous to hold this dressing firmly in the palm by means of silk sutures fastened to the skin to either side of the palm and tied over the dressings.

The hand is no longer dressed in complete extension following the operation. Complete extension of the fingers puts a great deal of tension on the palmar suture line and this may lead to necrosis. Full extension also prevents the skin from being pressed firmly into the palm so that hematoma formation is more likely to occur. Finally the fingers if held for some time in complete extension tend to develop joint capsule changes so that, although the patient may be able to extend the digits, full flexion is seldom regained.

STENOSING TENOSYNOVITIS

The fibrous sheaths which hold the flexor tendons in position over the metacarpophalangeal joints and the sheath enclosing the abductor pollicis longus and extensor pollicis brevis alongside the styloid process of the radius may become thickened and inflamed (Fig. 52). When they do so, they constrict the tendons beneath them, interfering with their action. In the digits, the constriction may cause actual indentations on the tendons. When the patient attempts to flex the finger it comes down to a certain degree of flexion where it appears to stick and then only with real effort and with pain can the patient complete the flexion. This release may occur suddenly often with an audible snap always with a palpable snap. For this reason, this condition is usually called "snapping finger." When an attempt is made to straighten the finger the process is reversed, that is, the patient is unable to extend the finger actively but must assist it with the opposite hand. It is a troublesome condition which is a handicap and the patient hesitates to use the painful finger.

It may occur on one or several digits on one or both hands but is usually single, involving the middle or ring finger. When it occurs on the thumb, it seldom affects the fingers.

Snapping thumb appears both as a congenital and an acquired condition. It is seen in children at birth or is recognized shortly afterward, since the child refuses to use this digit. In adults, snapping thumb often appears to be an occupational injury since it is seen not infrequently in persons who strike the base of the thumb frequently



Figure 52. Stenosing tenosynovitis occurs typically in two locations on the hand—in the fibrous sheaths of the flexor tendons over the metacarpophalangeal joints, where it causes snapping finger and snapping thumb and over the radial styloid where it is called de Quervain's disease.

against some object or tool with which they are working.

Stenosing tenosynovitis over the radial styloid is known as de Quervain's disease. It affects usually only one hand, but may be bilateral, and is most often seen in females. It, too, would frequently appear to be due to trauma, usually to blows over the area, or to wringing actions of the wrist. Thus, it is occasionally seen in young mothers whose infants sit on the arm, resting against the lower end of the radius. It is thought by some that in these patients it may be due also to diaper wringing.

The pathologic picture of both snapping tendon and de Quervain's disease is quite similar. When the fibrous sheath is uncovered, it will be found to be thick, tight and constricted about the tendons. Since the patient is usually operated upon under local anesthesia one can request him to move the affected digit and can actually see the tendon snap in and out beneath the constricted sheath. The tendon itself may present an actual constriction. Occurring in the index, middle, ring and little fingers where there are actually three tendon slips, it sometimes appears that the block occurs when the two slips of the sublimis move up in the palm about the profundus which lies between them. Occasionally there may seem to be an actual thickening or nodule in the profundus tendon. In rare cases, a tumor such as a ganglion may lie within the tendon and cause the trouble. When the thumb is involved, it is in the

valved, the surgeon more often finds a very definite constriction of the flexor pollicis longus tendon.

Seldom there is only an increase in the fluid content of the synovial sheath and only rarely is the synovial sheath thickened. In de Quervain's disease the pathologic picture is quite similar except that actual tendon constriction leading to snapping is not seen and there is usually a moderate inflammatory reaction of the synovial lining and not in frequently increase in the fluid content. Microscopically the tissues show simply a chronic inflammatory process with fibrosis and moderate round cell infiltration.

The symptoms and findings in snapping tendon in the digits differ somewhat from those of de Quervain's disease. When the digits are involved, the patient usually complains of moderate pain which is referred to the proximal interphalangeal joint. This may persist for some time before actual blocking of tendon movement occurs and even when this happens the patient, and frequently his medical adviser tend to localize the trouble in the proximal interphalangeal joint. By this time, however palpation of the proximal end of the sheath in the palm will show that there is thickening and some tenderness here. If the surgeon palpates this region while the patient flexes or extends the finger the tendons will be felt to snap back and forth beneath the thickened sheath. Watching the process of flexion and extension will show the temporary stoppage which occurs just before the finger finally "snaps" into full flexion or is brought back, often by use of the opposite hand, into complete extension.

In de Quervain's disease or tenosynovitis over the radial styloid, the symptoms are somewhat different in that there is more pain, and interference with motion is due more to pain than to actual mechanical block. On examination, the radial styloid is more prominent than normal, in contrast to the unaffected side. X ray examination will usually have been made and it will reveal no abnormality of the bone but will show the soft tissue swelling. Pain is especially noted when the thumb is adducted, thus interfering with grasp. A sign which is almost pathognomonic of the condition is based on the fact that adduction of the thumb with ulnar deviation of the wrist is extremely painful. The examiner grasps the hand with the thumb pressed into the palm. The wrist is then adducted toward the ulnar

side. This causes severe pain felt over the radial styloid.

Treatment of snapping tendon and de Quervain's disease consists in division of the fibrosed sheath. Usually it is best to excise the roof of the thick tunnel, although this is not absolutely necessary. Incisions for approach to the fibrosed sheath are made transversely and are therefore at right angles to the tendons nerves and blood vessels. The operation may be easily done with the patient under local anesthesia, but must be done in a bloodless field. The incision in the palm lies at the level of or just proximal to the distal palmar crease. The sheath is uncovered, care being taken not to damage the neurovascular bundles which lie to either side. The sheath is then split longitudinally to permit free motion of the underlying tendons which may be tested by asking the patient to move the affected digit. The whole roof of the proximal half of the sheath may be excised without leading to functional disturbance. In the thumb the approach is the same however the surgeon must be extremely cautious since the digital nerves lie to either side of the thumb, quite close to the surface and with scarcely more than the tendons breadth between them. They are quite easily injured if not visualized during operation.

In de Quervain's disease, the approach to the sheath or compartment is made transversely across the wrist at the level of the radial styloid. This incision lies directly across

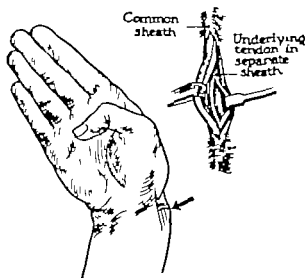


Figure 53 In de Quervain's disease, adduction of the thumb and ulnar adduction of the wrist cause exquisite pain. The tunnel containing the extensor pollicis brevis and abductor pollicis longus may be divided into several compartments which must all be opened at the time of operation.

the sensory fibers of the radial nerve. For this reason, the surgeon should be very cautious after the skin has been excised. Dissection should be in a longitudinal direction and the nerves must be identified. The sheath is then exposed and divided longitudinally and the interior is inspected. There may be more than two tendons passing through this tunnel. In fact, one or both of the enclosed tendons (extensor pollicis brevis and abductor pollicis longus) may be represented by two or three slips each. The importance of this fact is that one or several of these slips may lie in separate tunnels in the wall of the main tunnel (Fig. 53) and, unless these tunnels are also opened, the pain will not be relieved. It is well to splint the involved digit for a week or ten days after operation to insure sound healing.

TUMORS

A great variety of tumors occur on the hand and their accessibility and ease of examination should make for early diagnosis and proper therapy. The vast majority are benign and are amenable to successful surgical removal. Malignant tumors comprise only about 10 per cent of all tumors of the hand and, of these nearly 90 per cent are carcinoma, most of which arise as the result of irradiation dermatitis. Malignant tumors should be diagnosed or at least suspected early and the cure rate should be high.

The anatomy of the hand is such that removal of the superficial tumors frequently entails the necessity of skin replacement since there is little lard of cutaneous tissues, particularly on the digits and palmar surface. The deep tumors present their problems in demanding a careful anatomic dissection lest important nerves and tendons be damaged during removal of the tumor mass. Extensive and, particularly neglected growths may require major reconstructive procedures and their treatment should not be attempted without adequate experience with this type of surgery.

Trauma appears to play a significant role in the development of carcinoma of the hand and may have certain medicolegal significance.

One third of all tumors of the hand will be ganglia, one-third will be made up of four other types of tumors in approximately equal numbers, namely xanthoma, epidermoid cyst, angioma and carcinoma arising from irradiation dermatitis. The remaining third may be divided into two approximately equal groups. One group, or one-sixth of the

whole will be made up of carcinoma (not due to irradiation) lipoma fibroma, neuroma and enchondroma in approximately equal numbers. The remaining sixth comprises the remainder of the tumors of the hand, some very rare indeed. Among these are synovium, glomus tumor fibrosarcoma, giant cell tumors of bone osteosarcoma, osteoma, Ewing's tumor osteoid osteoma, lymphangioma sweat gland carcinoma, myxoma, leiomyoma and metastatic tumors.

This classification is based on relative frequency of tumors of the hand and disregards tissue origin. It is customary however to group tumors according to the tissues from which they spring or in which they are considered to arise. Thus, there are tumors arising from the skin from fibrous subcutaneous tissue from fatty tissues bones and joints, joint capsules tendon sheaths and tendons, nerves blood vessels, lymphatics, smooth and striated muscle. Some growths classified as tumors may very well not be tumors in the real sense of the word, e.g., the xanthoma, ganglion angioma and glomus tumor. Tumors arising from nerves and fibrous tissue may be confused and their true status may frequently be in doubt.

Tumors Arising from the Skin. The common wart is of importance on the hand for several reasons. *Verruca vulgaris* is generally thought to be due to some type of virus infection and the vagaries of its appearance and disappearance under varied and most illogical treatments are well known. It is, however often subjected to illogical types of treatment either by the patient himself or a physician and what has started out as a benign lesion may undergo malignant changes. The patient often keeps the wart trimmed down with his pocket knife or irritates it by picking it off with his finger nails. The wart may also be subjected to x-ray therapy which may or may not destroy it but often leaves in its place an area of skin damaged by irradiation which, in time may prove more serious than the original condition.

The treatment for warts on the hand is their removal. Many of them are removed by nonsurgical methods—cautery escharotics, carbon dioxide snow and fulguration. Surgical excision with the patient under local anesthesia seems preferable even though other methods are often more successful. Certainly if the warts have failed to respond to other treatment or if they appear to be growing and are ulcerating, they should be excised. It is always comforting

microscopic

section of such masses since it is possible for carcinoma to develop in them. Excision must obviously be done carefully as with all operations on the hand. The surgeon should be prepared to graft skin, particularly in case of the volar digital and palmar warts, since closure of the defect may be difficult.

The *subungual wart* is especially a nuisance, since it forms an unsightly mass at the finger tip, partly under the edge of the nail, and is difficult to keep clean. Removal usually demands excision of part of the distal nail bed and not infrequently a tiny skin graft is needed. The surgeon is inclined to be timid and to leave part of the wart behind and the mass promptly regenerates.

Keratosis and *hyperkeratotic lesions* are frequently seen on the dorsum of the hand in older people, particularly those who have exposed the skin to wind, sun and weather. They present themselves as roughened, slightly raised light brown plaques rough on the surface and not tender. They should be excised since they may be the forerunners of malignant disease.

Squamous papillomas may occur on the hand as well as elsewhere. They resemble soft warts, are quite benign and may be removed surgically under local anesthesia.

Epidermoid cyst often referred to as *inclusion cyst*, occurs not infrequently on the hand, particularly on the palm and volar surfaces of the digits (Figs. 54 and 55). It is thought in some instances to be due to implantation of small bits of epithelium into the subcutaneous tissues by pricks of thorns or

other penetrating types of injury. Tumors of this nature have been produced experimentally in animals. These cysts differ from the sebaceous cyst in that they are derived from the epithelium only and not from the sebaceous glands. Hence they contain no sebaceous material and their wall is formed from flattened cutaneous epithelium in which the various epidermal layers may usually be identified. The contents of the cyst consist of desquamated epithelial cells and other cutaneous debris. They are harmless tumors but may occasionally become infected. They may be confused with ganglia and with xanthomas particularly when the xanthoma is small and has only one lobule.

The treatment is, of course, surgical removal, usually under local anesthesia. These cysts show no tendency to recurrence.

Sebaceous cysts occur very rarely on the hand when they do it is on the dorsum. They are treated as similar cysts elsewhere by complete removal.

Pigmented moles are rather infrequently seen on the hand. Since the hand is frequently traumatized and exposed, it would seem wise to excise these blemishes to forestall any possibility of their injury. Fairly wide and deep excision should be carried out. Under no circumstances should they be irradiated, fulgurated or cauterized. They should either be left strictly alone or excised widely.

An area of hyperkeratosis may occasionally be neglected in elderly people and develop into a *cutaneous horn*. This curious

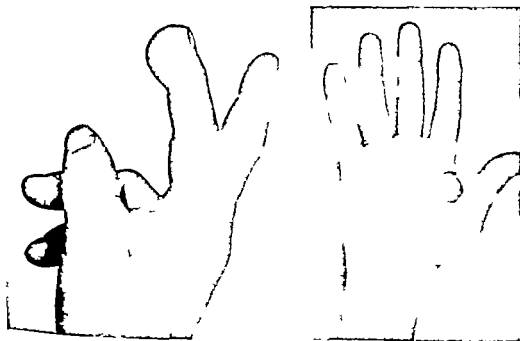


Figure 54 Epidermoid cyst of digit.
Figure 55 Epidermoid cyst of the hand.

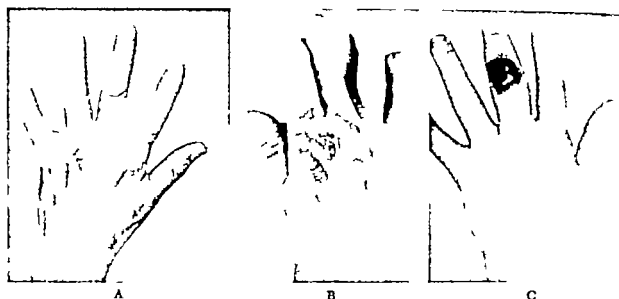


Figure 56. A, Carcinoma of the dorsum of the hand associated with exposure to sunlight. B Carcinoma of the dorsum of the hand arising from a wart. C, Carcinoma arising from area of chemical irritation.

appendage is not malignant and may be removed easily by excision. Skin grafting may be necessary to compensate for the resultant defect.

Carcinoma. This is the most frequent malignant lesion to occur in the hand and accounts for 90 per cent of all cancer of the hand and for some 10 per cent of all tumors of the hand. Another interesting feature of carcinoma of the hand is the frequency with which some irritative factor can be discovered in the patient's history (Fig. 56). In a fairly large series of hand carcinomas, irritation of some type or another was present in between 70 and 80 per cent of the patients. In only 20 to 30 per cent did it appear that the carcinoma arose from previously normal skin. Because of the frequency of some irritative factor it is possible to group hand carcinomas into seven groups, depending upon the type of irritation—exposure to sun and weather, irradiation with x rays and radium, chemical irritation, burn scars, internal medication, chronic infection and irritation of previously benign lesions. The 30 per cent or less of carcinomas of the hand arising from previously normal skin appear to be the most malignant of cutaneous carcinomas.

Hand cancer occurs with greater frequency in persons whose skin is exposed for long years to the vicissitudes of the sun and weather. A similar higher percentage of carcinoma of the skin of the face likewise is noted in sailors, farmers, ranchers and others who spend much time in the great outdoors.

It is not generally appreciated that irradi-

ation with x rays and radium is so frequently the cause of carcinoma of the hand. In our experience the majority of all cases of carcinoma of the hand are due to changes in the skin subsequent to exposure to x-rays or radium. In the overwhelming majority if not all these instances, the exposure has been unnecessary or unwise.

Carcinoma most often results from exposure to small, frequently repeated doses or minute daily exposure over a long time. Thus, when x rays were first introduced into clinical medicine their dangerous potentialities were unknown and the older roentgenologists took few or no precautions to guard against the rays. These men—the roentgen ray martyrs—were seriously afflicted and many died of carcinoma after numerous operations on the hands and arms. Later precautions were taken to prevent or minimize direct exposure. However it was then realized that even minute exposure to infinitesimal amounts of x radiation, if kept up day after day would eventually cause a skin reaction which was persistent and which failed to clear with simple medication.

This skin reaction might not become manifest for several years after exposure had been initiated and it persisted even if the individual stopped exposing himself to irradiation. Not only this, but years later often as much as twenty-five years, the original dermatitis would break down into areas of carcinoma.

At the onset of this type of chronic irradiation dermatitis, the skin becomes dry, often smooth, atrophic and then shiny. The hair might disappear or become quite sparse.

on the backs of the hands the nails rough and ridged (Fig. 57) The tiny cutaneous vessels in the area become apparent as telangiectases. This dry skin is easily traumatized and quite bothersome to the patient who treats it with various oils and greasy ointments. Soon, however numerous areas of keratosis and roughening appear. Areas of skin, which at first appear normal, may begin to show changes of dermatitis. Possibly, because of a minor injury or because of exposure to intense sunlight or other irradiation, an area of keratosis may break down into an ulcer. This ulcer may be exquisitely tender so that the patient hesitates to permit anyone to dress or examine it. Pain is more frequent and severe in the relatively acute ulcers than in the chronic ones. The development of an ulcer in chronic dermatitis usually though not always, indicates malignant disease. In many instances, the malignant lesion develops "in situ," often in several areas of the skin, and, if numerous sections are taken from a segment of skin which has been excised in treatment of the dermatitis, one will usually find early changes in the epithelial cells in numerous areas which indicate early malignant change.

The therapy of irradiation carcinoma, therefore, must be considered in three phases: first, prophylaxis of irradiation dermatitis, second, prophylaxis of carcinoma in irradiation dermatitis and third, treatment of irradiation carcinoma once it has developed.

With regard to the prophylaxis of irradiation dermatitis, it should go without saying that one should avoid needless exposure to radiation from any source. The professional

roentgenologist and radiation specialist, of course now take adequate precaution against the constantly repetitious exposure to which they were subjected in the old days. The nonprofessional, however, who uses x-rays especially in diagnosis is not so careful and exposes himself to frequently repeated x-rays, at the same time not taking the precautions his professional brother takes. The dentist, likewise, is inclined to be thoughtless in exposing himself to irradiation while taking dental films of his patients. He, too is becoming more cautious in this practice and has developed techniques which do not entail exposure to irradiation. Many surgeons expose themselves to irradiation while carrying out certain treatment or diagnostic procedures. For example, in the nailing of fractured hips, it is much simpler to carry this out under a fluoroscope and direct vision, but the orthopedic surgeon has learned the dangers of this procedure. Foreign bodies in the hand, if radiopaque, are easily seen in the fluoroscope and the temptation is great to search for them with forceps through a small skin incision. However this has often led to serious acute radiation dermatitis both in the patient and the surgeon. Few surgeons now carry out this hazardous procedure. Radium laboratories frequently hire nurses to pack and handle radium and radium emanation. The hands of these girls are exposed day after day to radium and, after a few years of such exposure the skin is irreparably damaged, although the evidence of it may not show up for many years.

But professional exposure is not the only cause of chronic irradiation dermatitis of the hands. One of the most frequent types of ex-



Figure 57 An "x-ray hand" in a dentist who held films in patients' mouths while taking exposures of the teeth.

posture is therapeutic, incurred in the treatment of various chronic recurrent skin diseases. Eczema, ringworm and psoriasis are all often treated by irradiation. The evil of this treatment is that the dermatologic condition may clear up either spontaneously or as a result of x rays only to recur. The patient returns for more treatment. If it is refused by his first physician, the patient frequently seeks help elsewhere and often does not inform the second practitioner of the previous x ray treatment. Such a performance may be repeated several times until the patient has received a very large dose of x rays delivered over a period of years. Since the rays used are those which especially affect the skin i.e., are not deeply penetrating, the damage produced is quite serious. Irradiation for the treatment of pruritus is seldom used on the hands, although we have seen one patient whose fingers were irradiated while he held his scrotum out of the way during irradiation therapy for pruritus ani. Carcinoma may eventually develop in these subjects years later although the incidence is slightly lower than in the professional group.

The carcinoma which develops is almost always of a squamous cell type and occurs not just in one spot, but in many. Numerous theories have been promulgated as to the mechanism by which this irritant gives rise to carcinoma, especially so many years following its discontinuance. One theory long held has been that the effect the irradiation has upon the circulation of the skin leads to chronic oxygen deprivation and that this eventually leads to nutritional disturbances which end in carcinoma. Another theory somewhat similar in nature has likewise to do with the effect of the irradiation on the vascular system. Dermatitis and telangiectatic vessels occur about the periphery of areas of irradiation. From time to time, these vessels thrombose and the thrombus lying beneath the thin skin forms a dark black speck, known as a "coal spot." The theory is that this coal spot or spots signify areas of tissue irritation and devitalization and that they stimulate the epithelium to repeated efforts at regeneration. Eventually from such irritative stimulation carcinoma develops.

These theories seem somewhat unfounded and are not borne out by careful studies of microscopic material. True enough, the arterioles of the irradiated skin are markedly narrowed by endarteritis and by fibrosis about them and there is no question but that the

skin is poorly nourished above them, but this is no proof that the poor nourishment in itself causes carcinoma. The overlying skin, situated as it is on densely fibrous corium, is easily traumatized, is constantly subjected to mechanical irritants and lacks protection by the natural skin oils which have been suppressed by the partial or complete destruction of the oil glands. Therefore, this irritative factor rather than just the circulatory one, could easily be as responsible for carcinoma. This same factor may well be at work in the production of burn scar carcinoma.

As for coal spots, if they are a cause, they must only rarely be implicated. In many sections studied by Teloh, Mason and Wheelock, coal spots were by no means a constant or even frequent occurrence. Carcinoma, on the other hand, has been quite frequent and seldom is found in the vicinity of coal spots. It has seemed that the rays must have produced some deep changes in the actual cutaneous cells. These changes, probably in the intricate chemical make up are fundamental and are carried from one cell generation to the next so that the growth processes in them over a period of years show the results of the process. It is possible to follow these changes in the skin cells from normal cellular structure, and minor variations in polarity and arrangement, to complete disorganization and carcinoma. These changes are seen not just in one area, but in many areas of the same specimen.

The treatment of this type of carcinoma may be considered separately from carcinoma arising from apparently normal skin, since the prognosis if the lesion is properly treated at the start, is very good and since prophylactic measures may be taken both to prevent the development of the dermatitis and to head off the occurrence of carcinoma in skin already affected by dermatitis. Likewise even if carcinoma has developed in the skin, the prognosis is quite good if the initial and local operation is thorough. If there has been delay in treatment and invasive carcinoma with lymph node involvement is present, a 25 per cent mortality can be anticipated.

It is wise practice to excise the whole of the involved skin and to replace it at once with grafts. It is not always possible to determine accurately the true limits of the skin involvement at the first observation. An apparently adequate excision may prove later to have been incomplete, since skin which at first appears perfectly normal may later show evidences of dermatitis al- borders of the

crafts. Why this is so is difficult to explain. The gross appearance of the skin suggests no trouble; however, there must be significant basic changes in the cells which are latent but which inevitably manifest themselves as time goes on. For this reason, persons who have such lesions should be examined periodically and new manifestations treated. The surgeon will often be surprised when the pathologist reports areas of carcinoma *in situ* in what appears to be only moderately irradiated skin.

When carcinoma is present it is treated as is cutaneous carcinoma elsewhere, by wide excision. If it is attacked early, there will very probably be no deep invasion, since the dense fibrosis of corium and blockage of lymphatics tend to hold the process localized. However, once these barriers have been passed, x-ray and radium carcinoma metastasize as do other squamous cell lesions. Fortunately, the great majority are of low-grade malignancy. However, the roll of roentgen ray martyrs stands as evidence of the malignant possibilities. Lymph node invasion may well occur and is present in about 50 per cent of the patients in whom enlarged nodes can be felt. Hence, epitrochlear and axillary dissections are indicated if enlargement is felt; otherwise such dissections do not appear necessary. With deeply invasive carcinoma, in which amputations are needed to effect removal of the local lesion, lymph node removal is advisable regardless of the findings.

Chemical irritation has long been recognized as leading to the development of carcinoma of the skin of the hands. Among the chemical irritants used commercially of particular importance are coal tar and its derivatives, aniline oils and greases, paint and coal soot. These are important only when the hands are constantly exposed to them over long periods. Certain industries are particularly hazardous in this respect; this has been recognized and measures are taken to avoid or minimize exposure. The liability aspects of the exposure cannot be ignored.

It is well known that *burn scars* of long duration, especially those of deep third degree burns with marked keloid formation, may break down into carcinoma. The thin epithelium overlying the dense scar beneath is frequently traumatized, breaks down easily into ulcers, cracks in usage and is frequently infected. Many years, often as many as sixty, may elapse between the burn and its breakdown into carcinoma.

Internal medication may be responsible for carcinoma of the hands. Fortunately, the use

of Fowler's solution has been very much curtailed, since this medication used over a period of years often tended to lead to arsenical keratosis which eventually broke down into squamous cell carcinoma. The lesions occur especially on the palms and soles but may be found wherever there is squamous epithelium and have been seen in the larynx. The condition is especially treacherous, since not one but multiple carcinomas develop on many regions of the body and continue to do so over a long time until metastases and a fatal outcome occur.

Chronic infection may be associated with carcinoma of the hand. At one time areas of cutaneous tuberculosis were irradiated and these occasionally developed carcinoma in association with the tuberculous lesion. One occasionally sees chronic osteomyelitic sinuses on the hand down which epithelium has grown and in which carcinoma has developed.

Irritation of previously benign lesions may be responsible for carcinoma of the hand. The ordinary wart if irritated sufficiently by picking and shaving it off or by overdoses of irradiation may occasionally break down into carcinoma. Several of the most extensive carcinomas of the hand which we have seen have been of this origin.

Carcinoma of the hand is almost always on the dorsal surface. Why the dorsal skin should more frequently develop carcinoma is not known, although it is tempting to speculate on the possibility of greater exposure of this area to various types of irradiation—solar x-ray and radium. Certainly the palm is more subject to mechanical trauma than is the dorsum and, if mechanical trauma were a factor here is where carcinoma should occur. Possibly the histologic difference between palmar and dorsal skin is the real reason.

If recognized early for what it is, the lesion is seldom found to be invasive, although it has the same malignant, locally invasive potentialities and tendency to lymphatic spread as do any of the cutaneous carcinomas. Wide excision, if there is no deep extension, beyond the skin, is frequently sufficient in the very early case. When invasion has occurred, more extensive procedures are called for and often partial amputations of a metacarpal and its digit will suffice. The extensive lesions involving wide areas of the palm or dorsum, however, will require amputation of the hand.

Dissection of the nodes draining the hand should be carried out in all patients in whom these nodes are enlarged or deep invasion has taken place. Whether or not routine axillary

and epitrochlear dissection should be carried out in every patient rests on the judgment of the surgeon. Axillary and epitrochlear dissection is not necessary in patients with superficial lesions without deep invasion. However in all in whom there is deep invasion or palpable nodes dissection should be carried out. In the latter group about 50 per cent will have evidence of involvement of the nodes.

Melanoma. The melanoma of the hand is most often seen involving the nail bed—the melanotic whitlow of Hutchinson (Fig. 58). It was first clearly described by Hutchinson who pointed out that these lesions are most often treated for some time as chronic paronychia. He also pointed out that these lesions show a pigmented border even if their central portions do not show any pigment. Their apparent innocuousness in the early stages may be misleading and the lesion may go toward deep invasion when its surface appears quite innocent. Nothing should be done to irritate the area. Biopsies are out of the question unless the surgeon is prepared to carry out at once the initial surgery. In the nail bed melanoma, amputation of the digit should be performed. The generally accepted course of action is then to carry out an axillary and epitrochlear dissection some four to six weeks later. This procedure has the authority of time-honored acceptance. Whether it is logical or the best treatment has not been determined. On grounds of analogy and logic, dissection starting at the axilla and working down along the supposed line of the lymphatics would seem more reasonable. It does not appear that the terrifically mutilating

forequarter amputations have offered much better prospects in regard to prognosis than have less extensive procedures.

Tumors of Fibrous Tissue. *Fibroma.* Fibromas occur in many locations in the hand. They are usually subcutaneous and produce superficial masses which are frequently diagnosed as neurofibromas, implantation cysts or early xanthomas. They may occasionally be pedunculated and hang from the skin by a thin pedicle. The early nodules of Dupuytren's contracture are usually diagnosed by the uninitiated as fibroma of the palm, and only as the fascial bands become more evident is the true identity suspected. Deep fibromas are seen occasionally in or on the tendons where they may cause symptoms of blockage due to mechanical interference with gliding. The fibroma is quite benign and does not recur after removal. However a peculiar fibroma does develop on the hand which does recur and usually is more extensive at each operative procedure. The microscopic appearance of this recurring fibroma may suggest nothing unusual. After several postexcisional recurrences, however its structure may change somewhat. This type of tumor probably should be included in the sarcomas.

Sarcomas (Figs. 59 and 60). There are many varieties of sarcomas of the hand. Many of them probably take their origin from nerve sheaths and are neurofibrosarcomas. They may however arise from any of the soft mesodermal tissues and vary from hard fibrous tumors to cellular masses containing only slight amounts of stroma. They may begin as tiny nodules in the lower layers of the skin or subcutaneous tissues but can arise deeply from any fibrous tissue as hard, usually painless, masses. They become fixed early to surrounding tissues and may cause pain. They may grow slowly for months or even years before their malignant nature becomes apparent. On the other hand, some of them develop very rapidly, become invasive and lead to early metastases and death. They may be composed of round cells, spindle cells or a mixture of the two and present a varying amount of stroma.

There is a variety which, while not peculiar to the hand, appears as a recurring fibroma that involves the overlying skin. This tumor is usually excised quite easily and apparently well beyond its limits. The defect left by the excision will often require a skin graft. Several months or even longer afterward, following an apparent cure, the tumor reappears in or along the border of the previous excision.

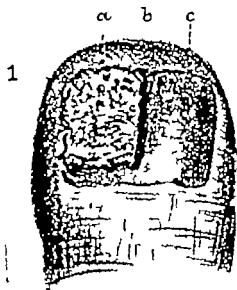


Figure 58. Hutchinson's melanotic whitlow (melanoma of the nail bed) (Hutchinson's original sketch.)

A second, and often a third or fourth excision, each wider than the preceding, will fail to lead to a cure. Careful study of microscopic sections may show nothing incompatible with a diagnosis of fibroma. After numerous recurrences, however it will become apparent that nothing but very wide excision or amputation will suffice and often at this time the microscopic characteristics will become more com-

patible with a diagnosis of sarcoma. Despite this however distant metastases are seldom encountered.

Lipoma. Lipoma is a fairly frequent tumor of the hand, where it forms a rather typical soft mass (Fig. 61) It is most often seen as a subcutaneous mass on the dorsum. Occasionally it is lobulated particularly if crossed by tendons or other dense structures. The

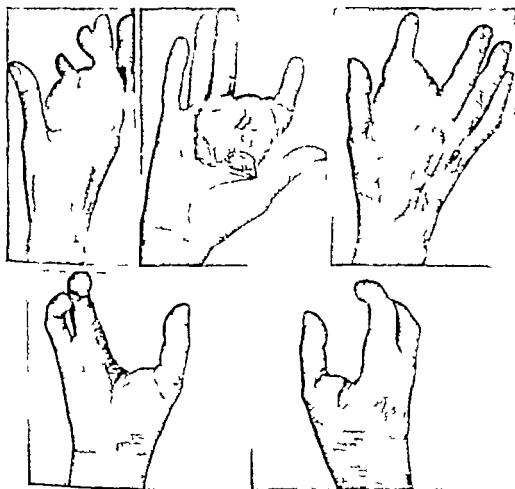


Figure 59 Fibrosarcoma.

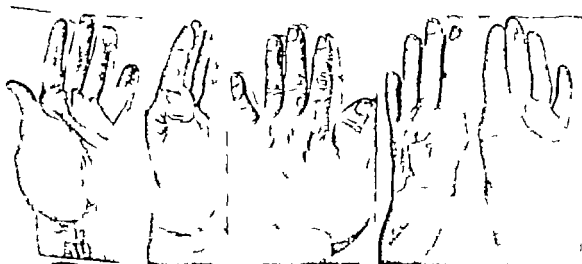


Figure 60 Malignant xanthosarcoma.

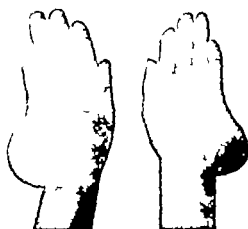


Figure 61 Lipoma.

superficial lipoma may be mistaken for an epidermoid cyst or xanthoma, however the characteristic consistency is usually a definite clue to its nature. These tumors are said to become hard when the hand is plunged into cold water. Deep lipomas may arise within muscle bellies about nerves or on or about tendons and within tendon sheaths. The intramuscular ones are very difficult to diagnose because of their location. They are said to become malignant occasionally. One variety of lipoma known as an arborescent lipoma occurs within the tendon sheaths where it spreads throughout the involved sheath and produces a picture quite similar to that seen in chronic tenosynovitis. Its removal entails an extensive dissection. It may be confused preoperatively with a tuberculous or other type of chronic tenosynovitis

with xanthoma of the tendon sheath or with villonodular tenosynovitis.

The prognosis for lipoma is good as far as recurrence or malignancy is concerned. In rare instances a liposarcoma may occur.

Tumors Arising from Joint Capsules and Tendon Sheaths. This interesting group of tumors should probably be called synovial tumors except that the name *synoviomia* carries with it the connotation of malignancy. King has studied these tumors more thoroughly than anyone else and has come to the conclusion that they are derived from cells destined to form the joint membranes, capsules and ligaments. They are, in fact, true tumors of synovial cell origin and should be known as *synoviomias*.

The most frequent of these tumors is the *ganglion* (Figs 62 and 63). It is also one of the most frequent of all tumors but it is at the same time one of the most obscure. It is held by many to be due to trauma and is said to occur especially in persons who subject their hands to frequently repeated motions, such as pianists, typists, violinists or others whose work entails use of the fingers and wrist for long periods of finely coordinated activity. Clinical observation does not seem to bear this out, since this tumor is seen in persons with all types of occupations. There was at one time especially among French observers an inclination to ascribe the condition to tuberculosis. However it seems likely that neither tuberculosis nor trauma is the real etiologic agent. Still another theory of its origin, and this was taught

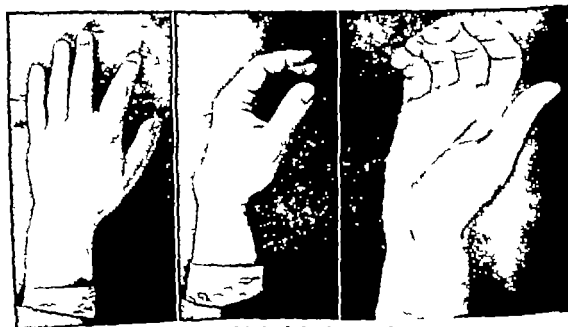


Figure 62 Ganglia of the hand—dorsal-carpal and volar on

for many years, was that it represented a herniation of the synovial sheath through a tiny defect in the fibrous capsule of a joint which explains why older surgeons always sought for the stalk when removing the tumor. The joint capsule is always opened when a ganglion is removed, but this is done because the tumor originates from those tissues which form its base and consequently removal entails excision of a portion of the joint capsule. Actual communication of the cystic cavity with joint space however cannot be demonstrated.

When ganglia are studied microscopically they are found to be made up of a basal tissue which is fibrous joint capsule or tendon sheath containing cellular areas within which may often be seen tiny cystic spaces. The cystic mass is continuous with this and consists of a thin-walled unilocular or multilocular cyst. It would appear that the large cysts come about by the enlargement and coalescence of the smaller cysts which originate in the basal tissue. It would appear also that the recurrence of ganglia is due to the fact that the basal tissue has been left behind in removal. This concept of origin likewise explains the frequent recurrence which follows simple rupture or aspiration.

Ganglia occur in a number of typical locations on the hand. Most frequent are the ones seen on the dorsal surface the so-called dorsal carpal ganglia, which take origin from the joint capsule at the base of the second metacarpal and which present themselves on the wrist between the tendons of the common

digital extensors and the radial extensors. Another favorite dorsal extension is in the anatomic snuff box where they originate from the joint capsule at the base of the thumb and from adjacent tendon sheaths. A volar-carpal ganglion is seen on the wrist at the radial side in connection with the joint capsule at the base of the thumb and the fibrous sheath tissue about the radial-carpal flexor. These latter two ganglia are in intimate association with the radial artery as it winds through the snuff box to the interval between the bases of the first and second metacarpals. The vessel is liable to injury during excision of the ganglia and must be visualized at all times.

Ganglia are also seen to arise from the fibrous tendon sheath of the flexor tendons over the proximal phalanges of the fingers usually the index, middle and ring fingers just distal to the proximal digital crease (Fig 63). Here they are thought by the uninitiated to be sesamoid bones although it should be realized that sesamoid bones lie over the joints which are considerably more proximal.

Occasionally cystic structures resembling ganglia are found within the substance of tendons and they may be so located as to cause the phenomenon of snapping tendon.

The symptoms of ganglia are mainly due to the presence of a hard mass in the tissues which causes either a lumpy cosmetic blemish or pain when the mass is pressed against underlying tissues. At times the development of the dorsal-carpal ganglion may be heralded by pain and tenderness at the base of the second metacarpal which may be present for weeks or months before the actual tumor appears. The recurrence of a ganglion after removal may be predicted by similar pain and tenderness.

Treatment of ganglion is surgical excision. Often excision of a ganglion is one of the first elective operations turned over to the tyro. However it is not the simple and easy procedure it would seem to be. The difficulty does not lie in removing the cyst intact although this is not at all easy but in removing the basal tissue from which the ganglion springs. This tissue is difficult to distinguish from the normal joint capsule and only the presence of tiny cysts within it gives evidence of its identity. The ganglion should be followed down to its base and then a generous segment of capsular tissue should be excised with the ganglion taking origin from its surface. It is imperative of course, that the procedure be carried out in a bloodless field. Local infiltration anesthesia usually suffices although there are certain advantages to re-

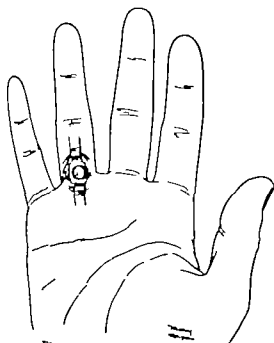


Figure 63. Volar digital ganglion.

removal with the patient under general or conduction anesthesia so as to avoid the obscuration of tissue detail by infiltration with fluid. The volar-digital ganglia (Fig. 63) which sit like pearls on the tendon sheath are excised in toto with the underlying square of tendon sheath. There is no clearer demonstration of the fact that ganglia are not herniations of tendon sheath than these tiny tumors.

Only too frequently the surgeon approaches ganglion through a longitudinal incision. This almost invariably leads to keloid scar over the dorsum of the wrist, to an unsightly scar on the volar surface and to a contracted scar on the finger. A transverse incision is always indicated. The incisions which run transversely across the lines of nerves blood vessels and tendons must be made with great care to avoid injury to these structures this is greatly facilitated by operating within a bloodless field. Following operation the wrist or finger is splinted in a functional position for about ten days to permit healing.

Other methods of treatment yield a high percentage of recurrences. Crushing the tumor by directing a heavy blow upon it with a book may sometimes effect a cure, although the surgeon who employs this treatment is not likely to know the final results. The procedure is quite painful. Injection or aspiration and injection likewise are unsatisfactory. The experience of others in the treatment with hydrocortisone seems to show that this method is also unsatisfactory.

The *mucous cyst* (Fig. 64) which occurs on the dorsum of the distal phalanges at or near the sides of the base of the nail resembles a ganglion in appearance and in the gelatinous character of its contents. As King has pointed out, however it is the result of mucoid changes in the subcutaneous tissue and does not originate from the joint capsule. The location over the base of the nail often leads to a depressed deformity of the nail. It recurs very promptly after removal, unless the overlying skin and cyst are excised together. This would seem to indicate that these tumors originate from the lower layers of the corium. Removal, therefore necessitates covering the defect with a tiny skin graft. These cysts must not be mistaken for Heberden's nodes.

Xanthomas or *giant cell xanthomatous tumors* occur as isolated tumor masses on the hand in contrast to the multiple xanthomatous processes which are seen in association with metabolic disturbance as multiple infiltration processes involving many tendons. There does not appear to be any cholesterol distur-

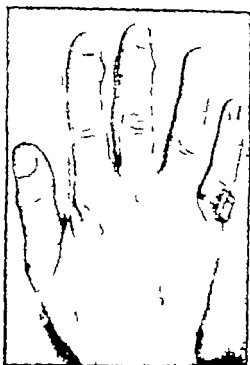


Figure 64 Mucous cyst of finger with compression of the nail bed.

ance in the instance of the isolated tumors and their exact nature is somewhat of a mystery. They are probably synovial tumors arising from tendon sheaths and joint capsules.

The histologic make-up of the tumors is quite variable and only by studying numerous sections from various areas of a single tumor can a composite picture be made of them. Study of a single section will not give a true picture of the histologic structure. The tumor is made up of four elemental areas intermixed in varying proportions from tumor to tumor. Foreign-body type giant cells are a striking characteristic. These have led many to diagnose the tumor as a giant cell sarcoma. Intermixed with the giant cells is a stroma of spindle shaped epithelioid cells. There are areas of variable size and shape made up of foamy cells with small nuclei and bubbly cytoplasm filled with cholesterol. Dissolved in the cholesterol is xanthine, which gives the tumor its yellow color; the cholesterol itself is colorless. Scattered throughout the tumor are hemosiderin deposits which some believe to represent hemorrhages indicative of trauma. In many of the tumors, peculiar spaces are found containing no blood cells, which have been thought to represent a tendency on the part of the tumor to form joint spaces and, hence, to indicate its derivation from synovial cells.

The tumor is benign and is amenable to local removal. This must, however, be very

thorough since the recurrence rate is quite high because the tumor takes origin from deeper tissues and the superficial mass may be only the surface manifestation of a long stalk which passes into the tendon sheath of a digit. If the stalk is not removed, the tumor re-forms. In other words recurrences are not true recurrences, but simply growth of the portion of the tumor mass which has been left behind. In rare instances following several removals, the tumor may assume malignant characteristics.

Xanthomas occur particularly on the digits (Fig. 65) where they may be confused with epidermoid cysts. These latter are usually unilobular in contrast to xanthomas which are usually multilobular. Either may be mistaken for the other. Xanthomas do not involve the overlying skin, but are firmly attached in the depths and, hence, are not movable. They are occasionally seen in the palm or the wrist, where they assume the characteristics of villous nodular synovitis. Few symptoms are caused except for cosmetic blemish or interference with use because of actual size and location.

Treatment is surgical removal. This must be carried out in a bloodless field, since every trace of the tumor must be removed to insure against recurrence. The digital nerves and vessels will be found coursing across the tumor mass, usually in a deep groove which separates one lobule from another. Unless particular pains are taken, the nerve and its associated vessel will be divided. Great care must also be taken to follow the tumor throughout its entire course and not be content with removing simply the large lobulated more superficial mass. A tail of the tumor will often be found to enter the tendon sheath and to run for a variable distance in it. This may be the origin of the tumor and must be removed. When the tumor presents itself at the wrist, it may be very extensive and involve the whole of the radial or ulnar bursa, or both, and require

as extensive a dissection as does tuberculous tenosynovitis. Here also one may occasionally find the tumor to have invaded the carpal bones.

The prognosis for xanthoma is good as far as malignancy and metastases are concerned, but the recurrence rate tends to be high unless special care is taken in the primary removal. In occasional instances the tumor may be so extensive on a digit as to replace almost entirely large areas of the digital fatty tissue and to compromise the blood supply so seriously that a finger may have to be amputated.

Synovioma. The tumors previously described are probably of synovial origin and should be classed as synoviomas. However the term "synovioma" has become attached to a particular malignant variety of these tumors which fortunately is rarely seen on the hand. These lesions occur near the joints as painless, slow-growing, rather indefinite masses suggestive of a rheumatic nodule. They form encapsulated or diffuse masses often butter yellow in color. They may be solid or fleshy and often are not easily distinguishable from the surrounding tissues. They appear to spring from the joint capsule and in early stages do not involve bone. Following simple removal, prompt recurrence is to be suspected and metastasis may be anticipated if amputation of the digit is not soon carried out.

Blood Vessel Tumors. All of the varieties of vascular tumors met with elsewhere on the body occur on the hand and forearm. Whether or not they are true tumors is often to be questioned. Reid, from an extensive experience with many such tumors concluded that most, if not all of them, represented some form of arteriovenous communication. It would seem logical to ascribe many of them to arrests in development of segments of the vascular tree. Study of the numerous contributions made by Sabin re-

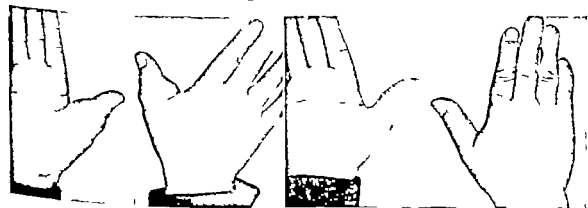


Figure 65 Xanthoma.

garding the growth of the blood vessels gives one a picture of the various phases of growth, in many of which the vessels resemble the vascular tumors as we know them. The only malignant blood vessel tumor resembles the angioblastic phase of vascular development. Other blood vessel tumors suggest certainly the stage of capillary plexus formation some the retiform stages; and still others arrest in the stage of stem formation. The diffuse arteriovenous aneurysms with multiple arteriovenous communications would certainly seem to represent arrests in intermediate stages of vascular tree development.

While arrest in development of blood vessels may represent the origin of the tumors their continued growth may be a manifestation of the effects of arteriovenous communications and the effect of the abnormal circulation upon neighboring vessels. A specific blood vessel tumor may not always be of a certain type but may contain various mixtures of capillary and cavernous and vessel types of angioma.

The *capillary* or *telangiectatic* type of angioma is present familiarly as the port wine stain or strawberry mark and has usually been manifest since the birth of the patient. It is usually elevated and soft, of bright color and nonpulsatile. It may occupy only the under layers of the skin, but there may be cavernous spaces beneath it. Occasionally it will disappear during the first year of life, but one cannot count on this. From a small beginning, the tumor may grow extending over larger and larger areas until its eradication presents a very difficult problem indeed. Hence early removal seems indicated if the mass is of any size, and especially if it shows any tendency to growth. It may be necessary to replace the skin area excised with skin grafts if primary closure cannot be accomplished otherwise.

The *cavernous angiomas* are occasionally seen on the hand, particularly in the palm, where they seem to take over the venous elements of the circulation (Fig. 66). When uncovered, there are discovered large thin-walled venous lakes which extend up and down about nerves and tendons into muscles and not infrequently from palm to dorsum. Very extensive involvement is almost the rule and, although the arteries are not involved per se, it is often impossible to remove the angioma without damaging the normal vessels. Hence, a staged operation is frequently required.

In not a few instances these cavernous



Figure 66 Venous angioma.

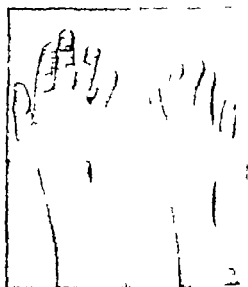


Figure 67 Arteriovenous aneurysm (congenital) involving the vessels of the middle finger

lakes involve the bones, usually of one or two digits a fact which is indicated by x ray examination.

The *congenital arteriovenous aneurysms*, as they occur on the hand, frequently affect the vessels of one area, e.g., one or two digits (Fig. 67). They are characterized by extensive pulsating vascular tumors, enlargement of the involved digits or of the whole hand, and venous dilatation over the hand and forearm. The arteriovenous communications in these instances are multiple, thus contrasting with the traumatic arteriovenous aneurysm and making for considerable difficulty in care. The tiny vessels in the skin become dilated the s

es thin or L

them and furious hemorrhage may occur following a trivial injury

Treatment of the angiomas consists in surgical eradication. Although the capillary type is sensitive to irradiation this must be given in such destructive doses to effect cure that the skin is left badly involved with irradiation dermatitis. When irradiation has been carried out, subsequent excision of the skin followed by grafting is often required. Such therapy seems contraindicated in youngsters since the epiphyses may be damaged and serious growth disturbances result. Injection of sclerosing agents although advised in the older literature is seldom practiced today.

Capillary angiomas may be excised and satisfactory results obtained. The need for skin replacement at the time of excision will depend upon the size of the area removed. Cavemous angioma may likewise, be excised, although it presents a more difficult problem than does the capillary type. As intimated above, the tumor may be so extensive that complete removal may be impossible and the surgeon must be content with partial excision of the most troublesome area. At times the procedure must be staged for fear of compromising the blood supply to one or more digits, if excision of the tumor involves the blood supply too seriously. These tumors only rarely develop arteriovenous communications hence, partial excision may be successful.

Congenital arteriovenous aneurysms present one of the most difficult problems in surgery of the hand. Theoretically one should be able to approach and ligate the arteriovenous communication. Practically this is seldom possible, except in a limited sense. The communications are multiple, often affecting vessels over a large area, on both volar and dorsal surfaces, and one usually finds that after supposedly adequate excision and ligation the tumor promptly grows again, often to a greater proportion than previously. If the vessels of a single digit are involved, amputation of that digit is indicated. Unfortunately the process is seldom confined to one finger. The surgeon may offer some chance of cure, or at least of amelioration, by local attack on the tumor plus ligation in stages of the blood vessels to the hand, allowing sufficient time between ligations to permit collateral circulation to develop.

Telangiectatic granuloma (pyogenic granuloma, Fig. 68) may or may not be a true tumor. It is known for a long time as false

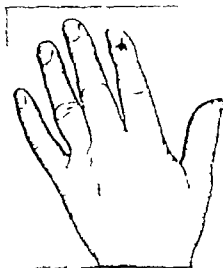


Figure 68 Telangiectatic or pyogenic granuloma.

botryomycosis since it resembled a tumor mass developing in horses following castration and was thought to be due to a fungus infection. While it is now known that it is not due to a fungus its true nature is still subject to controversy. It is most frequently called a pyogenic granuloma and the implication is that it is an infectious granuloma. It appears as a small mushroom-like growth frequently located on or near the finger tip but may be in any location on the hand. The stalk of the growth pushes out through a small opening in the skin which forms a thin collar about it. Microscopic study of the mass shows it to be made up of myriads of tiny capillary vessels, resembling very much a capillary angioma. Some think it is in fact, an angioma which has burst through the skin. It often disappears after x ray therapy occasionally even when it is treated by compression. It is quite amenable to simple excision with closure of the defect. This seems to be the preferable treatment. The tumor does not recur after removal.

Glomus tumor This tumor originates from the neuromyoarterial glomus, a structure associated with the autonomic nervous system and having to do with the heat-regulating system. The glomus is found only in warm-blooded animals and is especially developed in the feet of ducks. Glomera are essentially arteriovenous communications made up of fine arterioles connecting directly with venules forming the canals of Sucquet Hoyer. The vessels are surrounded by cells resembling epithelioid cells which are thought by many to be modified smooth muscle cells. No nerve cells are found in the glomus however there are many myelinated and nonmyelinated nerve fibrils in

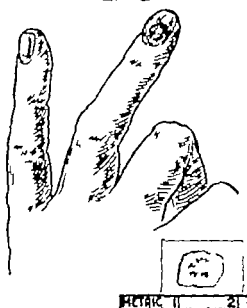


Figure 69 Subungual glomus tumor. Previous to operation it showed up as a dark purplish-red patch under the nail. The inset is a photograph of the tumor after excision.

it and these give evidence of its close association with the neurovegetative system. Glomera are found scattered all over the body and tumors arising from them have been reported in bone and other deeper structures. They are especially abundant in the palmar skin and in the nail bed (Fig. 69). Hence, the vast majority of glomus tumors are reported as occurring on the hand, although these lesions have been reported in practically every region.

The tumor is frequently very small (5 mm. or less in diameter) but may attain sizable proportions, measuring up to 1 inch in diameter. Multiple tumors have been reported.

Glomus tumors give rise to a rather typical pathognomonic chain of symptoms. Starting insidiously they develop into tiny painful nodules or painful areas which may not be palpable as nodules. If they occur in the nail bed, they may be visible as reddish or purplish tumors, a few millimeters in diameter which push up but do not perforate the overlying nail. They are visible at times invisible at others, and are more painful at times than at others. Sometimes they are especially painful when cold, in other instances when warm. They are always painful to touch and may be so exquisitely tender that the patient protects the part by placing his hand in his pocket. They often give rise to paroxysmal attacks of pain which radiate up the forearm and arm or even into the

neck. In a few of the patients, Horner's syndrome has been observed. The patient usually presents himself with a long history of an undiagnosed painful spot which has made life generally miserable.

Diagnosis is usually suggested by the history and the findings of a tender subcutaneous or subungual nodule. The tumor under the nail may be visible and the nail itself may be pushed up and rounded by the underlying mass. An x-ray examination of the finger tip may reveal a depression in the distal phalanx due to the pressure of the tumor. Similar tender spots may be caused by other tumors such as leiomyomas and neuromas, but these are rare and none gives the typical history of the glomus tumor.

Treatment consists in simple shelling out of the tumor. Because of the pain and apprehension on the patient's part, operation may often have to be accomplished under a general anesthetic. A bloodless field is essential since the tumor may suddenly constrict and what was a pink or red tumor mass may become invisible. This is especially liable to occur when the patient is under local anesthesia. Recurrence is rare unless the tumor is missed. These tumors do not become malignant.

Lymphangiomas. Lymphangiomas are extremely rare. They are seen as congenital lesions leading to enlargement of parts or all of the hand, forearm and arm. They may produce a type of gigantism. Excision is usually difficult because of their extent.

Bone Tumors. Enchondroma. Except for enchondromas tumors of the bones of the hand are rare. Enchondroma is however not at all infrequent. It is seen particularly in the shafts of the metacarpals and proximal phalanges but never in the carpus. It may be multiple and both hands should always be x rayed when a suggestive bony swelling is present. Occasionally the surgeon will find multiple tumors of both hands, but this is rare in our experience.

Enchondromas develop as slowly growing, hard tumor masses in the tubular bones, usually near the head of a metacarpal or base of a proximal phalanx (Fig. 70). Occasionally the patient will complain of dull pain, but more frequently there are no symptoms until a trivial injury leads to a fracture which prompts an x ray examination which discloses the tumor. Curiously enough, there is seldom gross displacement of the fracture, probably because the injury has been so trivial or because the solid tumor helps support the bone. The x ray shows a well

defined vesicular area of rarefaction within the shaft of bone which has expanded and thinned out the cortex but does not perforate it. The picture may resemble very closely that of a giant cell tumor. However the giant cell tumor is more like a soap bubble in appearance and in addition is of extreme rarity in the bones of the hand.

Treatment consists in unroofing of the tumor and thorough curettage of its granular pearly white contents. If the patient presents himself with a fracture the bone should be splinted and allowed to heal before the tumor is attacked. Healing of the fracture can be anticipated to take place satisfactorily.

At operation, the surgeon should expose the thinnest portion of the involved shaft and, after stripping back the periosteum, carefully remove the protruding cortex to give adequate access to the underlying tumor mass. The remainder of the shaft is undisturbed since it preserves continuity. The pearly white rather dry granular tumor is then scooped out with a small curet. A surprising amount of material will be removed and great care must be taken to secure all of it. Only the cortical shell should remain. It was at one time advised to fill the defect with bone chips; however this does not seem to be necessary. The stripped periosteum is allowed to drop back as well as it will and the soft tissues are closed. The cavity soon fills with a clot and the bone re-forms over it satisfactorily so that in time a practically normal shaft is present. The tumor will not

recur if it is adequately removed. It is not malignant.

Exostoses These tumors appear infrequently on the hand usually in the region of tendon insertions and are occasionally multiple. They are larger on palpation than the x-ray picture indicates since they are capped with cartilage, and are properly called cartilaginous exostoses. They may rarely develop in the nail bed as Dupuytren's exostoses. They push up and erode through the nail leading to a granulomatous lesion. These tumors are not malignant and respond to simple local removal.

Osteoid osteoma. This tumor was first separated as a definite entity by Jaffe, in 1940. It is seen with moderate frequency on the hand as indicated by the report of Carroll who collected the records of a number of cases from the literature and, with his own cases studied a series of twenty-eight of these tumors in the bones of the hand.

Osteoid osteomas are easily confused with other rarefying bone lesions such as osteomyelitis, bone abscess and syphilis and must be differentiated from them as well as from osteochondritis dissecans, xanthoma or even bone callus. The tumor appears roentgenologically as a small translucent area in the bone, either in spongy bone or the cortex, surrounded by an area of increased density or sclerosis. Pathologically it is made up of a center of osteoid tissue surrounded by sclerosed bone.

The clinical picture should be quite sug-

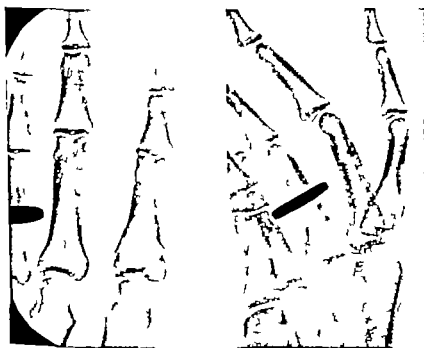


Figure 70. Enchondroma. The fracture led to x-ray examination which revealed the tumor.

gestive of the nature of the condition. The most prominent feature is pain—chronic, nagging in character rather than severe, and increasing especially at night. However the pain responds to mild sedation with aspirin. Study of the x ray film, with these facts in mind, makes the diagnosis certain.

Moberg has shown that the tumor runs a natural course over a period of several years, and eventually cures itself.

Treatment consists in removal of the tumor. Great care must be taken to remove the nidus or center of osteoid tissue since, if any of this remains, recurrence is certain.

Giant cell tumors. These are quite rarely seen on the hand. When they occur on the upper extremity they are usually seen in the radius near the wrist. They have, however, been reported in the metacarpals near the epiphysis. They are slow-growing tumors. Their presence is characterized by boring pain usually severe and worse at night. They may appear a great deal like enchondroma on x-ray examination but usually appear more like soap bubbles. The severe pain would tend to differentiate them from enchondroma which is painless.

Treatment of the giant cell tumor consists in its complete removal, which may be more difficult and more extensive than the procedure required for enchondroma. Frequently a segment of bone must be excised and a bone graft inserted although, occasionally thorough curettage of the currant jelly like contents may suffice.

Bone cysts myxomas osteomas fibromas hemangiomas and Ewing's tumors may occur in the bones of the hand. **Aneurysmal bone cyst** is rare in the bones of the hand. Only four or five have been reported. They resemble giant cell tumor and enchondroma on roentgen examination. Only microscopic study will determine the diagnosis. **Bone sarcomas** are also seen on the hand, but with extreme rarity only five cases have been reported. Treatment consists in amputation of the hand.

Tumors of Nerve. Tumors of the peripheral nerves, as they occur on the hand, are actually tumors of the sheath elements. They are nodular diffuse, a spindle-shaped enlargement of the nerve area or a plexiform thickening of all the nerves of an area—often of the entire hand—as in von Recklinghausen's disease.

The nodular types of neuromas, neurinomas or neurofibromas (Fig. 71) are usually subcutaneous nodules, single or occasionally multiple, in the subcutaneous tis-



Figure 71 Fibrosarcoma of the hand.

sues in connection with subcutaneous sensory nerves. They are derived from the connective tissue elements of the nerve sheath and present a typical histologic picture. Their removal occasions no disturbance in nerve function.

The fusiform neuromas develop along the course of the major nerves of the hand, usually the median, less often the ulnar nerve, and may extend over large distances of the trunk. We have seen several which involved the median nerve from a point several inches above the transverse carpal ligament down into the palm and along the terminal rami of the nerve to the base of the digits. Seddon claims that it is possible to strip the nerve fibers proper from the surface of the tumor about which they lie thinly spread out like the ribs of an umbrella. This has not been our experience with this tumor. In those which we have attempted to dissect free of nerve fibers, there has been such an interwoven complex of nerve fibers and tumor that separation has proved impossible. However the surgeon should always make this attempt before deciding to sacrifice the whole nerve trunk. The tumors are benign, but one must always suspect multiple occurrence in other parts of the body. Many of these may well be manifestations of von Recklinghausen's disease.

Von Recklinghausen's disease may involve the hand as it does other areas of the body. The significance lies in the fact that approxi-

nately 15 per cent of these tumors become malignant.

Plexiform neuroma. This condition is characterized by diffuse thickening of a nerve or group of nerves over a more or less extensive area. A single finger may be involved or several digits or even the whole hand. The thickening involves not only the main nerve trunk, but the fine terminal twigs and even the skin. Associated with this is general thickening of the skin and subcutaneous tissues the fingers become thick, soft and long. Grotesque enlargement may take place. When it is marked, it is known as elephantiasis neuromatosa. It would be impossible in many instances to remove all involved tissue without complete sacrifice of parts of the hand. However it is usually possible to improve the condition from a cosmetic standpoint by partial excision and skin grafts.

Neurosarcoma or neurofibrosarcoma. This tumor may appear as a nodular mass which grows back slowly after apparently adequate excision. The tumor in such instances may give all the appearance of being benign, even on microscopic section and only its prompt reappearance gives a clue as to its identity. Often its course may extend over many years eventually amputation of the extremity may be required. On the other hand, some of the neurosarcomas are extremely malignant, metastasizing early and leading to death in twelve to eighteen months. As with any sarcoma, the initial operation is the most important. It should be radical and extensive.

Metastatic tumors. The hand is occasionally the site of a metastatic tumor usually lodging in the bone. A fair number of such cases have been reported. In rare instances they may be the only manifestation of the primary tumor. The source is usually the lungs, prostate or testes.

CONGENITAL DEFORMITIES

Although congenital deformities of the hand tend to fall generally into certain patterns, these are only roughly definite compartments and there is not the general pattern of deformity such as is seen, for example, in harelip and cleft palate. Except in a general way no two cases are exactly alike and, except for such conditions as syndactylism, it is not possible to devise standard operative procedures, such as those that are employed for harelip. Many and various theories have been put forth in explanation of the origin of congenital deformities most

writers on the subject hark back to our prehistoric ancestors and to analogies with closely related present-day animal relatives to account for the various and curious malformations which occur. External influences and intrauterine accidents have been blamed. However in most instances it would seem that actual changes in the germ plasma, often of an inherited nature, are to blame. Experiments with irradiation indicate that this modality might lead to gonadal damage and the latter may cause the development of inheritable deformities in the offspring.

Despite difficulties in classifying these deformities better understanding is gained if one attempts to place them in certain groups, depending on certain general characteristics. Kanavel, who made an exhaustive study of congenital deformities concluded that deformities could be grouped into those showing hypoplasia and aplasia, disorientation of tissue and hyperplasia. Further he noted that malformations have a predilection for certain parts of the hand and forearm, e.g., radial and ulnar defects or combined radial and ulnar.

Kanaval was led to believe, on the basis of anatomic and functional studies and from certain evidence based on comparative anatomy and embryology that the hand and forearm may be divided into "ulnar and radial divisions separated by a fibrous partition attached to the middle metacarpal bone." The vascular and nerve supplies likewise suggest a close relation of the little, ring and middle fingers to the ulnar division, and a less close association of the index finger. The thumb is radial alone. Functionally the thumb acts alone the three ulnar digits together and the index finger in association with the ulnar digits. The fourth and fifth digits are frequently involved together and not infrequently the middle finger is involved with them. The index finger is seldom involved with the other digits, while the thumb is usually involved alone, occasionally with the index finger. In congenital absence of the ulna, the thumb and index are usually preserved, while in lobster claw hand the middle finger is usually the one absent while the thumb, the index, ring and little fingers are preserved.

In a study of congenital deformities of the hand, the surgeon is inclined to think only of the configuration of the bones as evidenced by x ray examinations, and the gross morphologic changes as evidenced by examination of the hand. One sees absence of

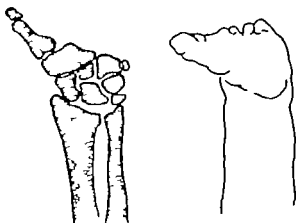


Figure 2. Congenital amputation. This was long thought to be due to amniotic bands and adhesions but is now recognized to be due to defects in the germ plasma (Kazavel, A. B. Arch. Surg., vol. 25)

bones, bones reduced in size, fused to each other or actually duplicated. On examination of the hand, the fusion of digits, webbing and rudimentary or extra digits are easily seen and recorded. What is not so often considered is the fact that these changes affect the whole fabric of the forearm and hand and may even be evidenced in tissues of the cord. There may be absence not only of the bones, but of nerves and tendons as well. Fusion may affect the deep soft tissues as well as the hard tissues and skin. Attempts to correct these deformities operatively must not be approached as simple problems of bone and skin surgery; attention to the nerve vascular and tendon supply is equally significant. It must be remembered also that, although certain types of deformities may be discussed as entities, they are seldom present alone. Thus, syndactylism or fusion of the fingers, is frequently combined with fusion or absence of joints in the digits, with shortening or absence of digits, with absence of a phalanx in a digit or even with an accessory digit. It will lead to less confusion, however if the various elements of deformity are discussed separately keeping in mind always that such elements are seldom, if ever the only feature of the deformity.

Congenital Amputations (Fig. 72) Amputations of digits, hand or arm and congenital constrictions involving these areas were for a long time thought to be due to bands of amniotic adhesions, actually constricting the part. This theory has been abandoned and these deformities are now thought to be due to actual factors in the germ plasma or to the effect of noxious factors, such as irradiation, on the developing embryo



Figure 3. Congenital absence of the radius (Kazavel, A. B. Arch. Surg., vol. 25)

When there is absence of a part, surgery can do little about it. Constricting bands, however can often be corrected or helped. Thus, deep constricting bands may seem almost ready to pinch off a digit at its base and it would hardly seem possible that there is space for blood vessels to enter. Such bands may often be opened up, usually by a Z-plasty and considerable improvement in appearance and function can be secured.

Hypoplasia and Aplasia. The group of deformities classed as hypoplastic and aplastic, while numerous and varied, have in common the element of tissue destruction ascribed to injury to the germinal anlage or to damage to the growing embryo. Parts of the extremity may vary in evidence of this destructive factor from simple diminution in size to complete absence or loss. In about 50 per cent of cases of congenital deformities of the hand, the most serious defects fall into this group. The severity varies from such simple anomalies as shortness of a digit to its complete absence from diminution in size of the hand to complete loss, from shortness of arm and forearm to their absence with the hand coming directly off the shoulder to entire absence of the whole upper extremity. Nor are those anomalies single; they are often multiple and are associated with other less severe defects due to disorientation of tissue. The hypoplastic and aplastic deformities show marked hereditary tendency to development of similar anomalies in succeeding generations.

Congenital absence of the radius (Fig. 73) is bilateral in almost one-half of reported instances the radius may be completely or partially absent. If it is partially absent, the upper end may be missing with a more or less rudimentary diaphysis; the lower end may be missing with varying amounts of diaphysis or the diaphysis may be absent with upper and lower

The ulna is usually thick, to the ra-

dal side or may rarely be double. Other deformities of the hand may be present, such as absence or atypical forms of the carpal bones, absence of the first metacarpal and absence of, or rudimentary thumb. The other digits are usually normal. The elbow may be ankylosed. The muscles and nerves of the forearm may be disoriented, atrophic, fibrotic and contracted. The hand is deviated to the radial side, thus producing the radiopalmal clubhand, and may be small, but is usually potentially functional.

Treatment consists in correcting the radial deviation by division of the shortened soft tissue or in favorable cases, by appropriate splinting and then holding the hand in proper position by replacement of the absent radius by a bone graft. It is occasionally necessary to fix the hand in the position of function by fusion at the wrist. If a bone graft is used, it should be fixed against bone at each end, otherwise, it will undergo resorption.

Absence of the ulna occurs much less frequently than does absence of the radius probably in the ratio of about 1 to 5. It is a complementary picture to that of radial absence in that the forearm is short, the radius is curved and there is ulnar deviation of the hand. The elbow however is usually dislocated and there are more likely to be deformities and destruction of the digits and carpal bones. The loss of digits often makes these hands less functional than does the radial type of deformity. The little, ring and middle fingers are the ones most usually absent. Loss, atrophy and fibrosis of muscles, nerves and vessels usually accompany the deformity in varying degrees. The defect is frequently bilateral. If the hand has not taken part in the process, function is good. If, however the hand is involved, the functional loss is often very great.

Hypoplasia affecting the hand produces a multiplicity of deformities which do not follow any definite pattern, such as the absence of radius or ulna does in the case of the forearm. The involvement may start at the wrist and the whole hand may be implicated. Only the medial digits may take part in the condition or the thumb bud may be the most affected. Individual bones may be shortened or lost. There is seldom hypoplasia alone—usually more than one extremity is affected and a hereditary tendency is very frequently noted.

Hypoplasia of the elements which make up the middle finger produces a striking picture quite out of proportion to the general

importance and, because of its redness lobster-claw hand has been accorded a prominent, easily remembered spot among the congenital deformities of the hand. In this condition, the middle finger with all or most of its metacarpals is missing and the hand is cleft down the center into two halves each bearing two digits, presumably the ring and little fingers to the ulnar side and the thumb and index finger to the radial side (Fig. 74). Since this condition is thought to resemble the foot of certain ancient mammals and since certain living mammals have cleft feet, the lobster-claw hand has been thought to be due to reversion of the human hand to ancestral human type. However this evidence is not especially convincing and does not explain the other types of hypoplasia of the hand and the numerous associated deformities which usually are found with this and other hypoplastic anomalies.

Treatment for median hypoplasia attempts to restore the digits to a functional and more cosmetic position. It must be remembered by the surgeon, however that attempts to restore cosmetic appearance should not interfere with the use of the hand which the patient already has or may develop. The deformed hand is the only hand the patient has known and he learns to use it remarkably well despite its bizarre appearance. In some cleft hands, it may be possible to unite the two halves of the hand after removal of remnants of the third metacarpal and to cor-

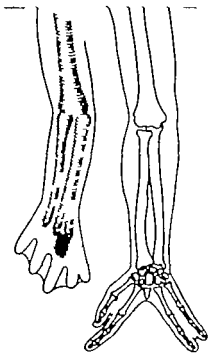


Figure 74 Lobster-claw hand (Kamuel, A. B : Arch. Surg., vol. 25)

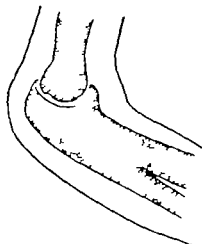


Figure 75. Radioulnar synostosis (Khanavel, A. B. Arch. Surg. vol. 25)

rect the syndactylism which obtains following it. Release of contracting bands physical therapy and traction splinting are often valuable in eventually improving function. However the patient himself, left to his own resources, will usually secure a most functional member from a seemingly hopeless situation.

Hypoplasia and aplasia of elements of the hand and wrist are many and varied. There may be shortening of digits due to short metacarpals or short or absent phalanges, in various combinations. There may be flexion deformities and deviations of the digits, occasionally with disorientation of epiphyses. The triphalangeal thumb is thought by some to be an atavistic affair by others to be a duplication of the index finger with absence of the thumb. The origin of the biphalaengeal thumb is thought by some to represent absorption or disappearance of the metacarpal with the present metacarpal being actually the proximal phalanx.

Fusion of Hand and Arm Elements. Fusion of the various parts of the hand, wrist and forearm may vary from simple webbing of the digits to actual fusion of bones in whole or part in all degrees. In the forearm, the picture of radioulnar synostosis is a fairly definite entity (Fig. 75). The union usually occurs at the proximal end of the bones and is often associated with anterior dislocation of the head of the radius. The forearm is usually in pronation. There is often little functional disturbance due to this lesion in itself, supination being carried out by movement of the shoulder. If the disability is severe, some motion may often be secured by osteotomy of the radius below the point of union.

Syndactylism. This is a fairly common condition, often part of other congenital de-

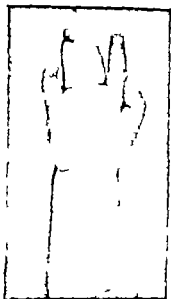


Figure 76 Syndactylism.

formities of the hand. It consists in the fusion of two or more digits (Fig. 76). The fusion may be complete from base to tip or may extend only a short distance down the web space. There may be only fusion or lack of separation of skin or from this relatively simple fusion there may be many degrees, ranging from fusion of distal phalanges on up to complete fusion of all joints of the digit. The soft tissues may likewise partake of this fusion, so that tendon, nerves and vessels may be reduced to only a single set between the fused bones. Any group of fingers may be involved, although the most frequent are the ring and middle fingers, less often fusion of other digits, including the thumb and the index finger.

Treatment of syndactylism has undergone many phases. The separation of fused fingers has intrigued surgeons for well over a century and many ingenious procedures have been proposed and carried out. Several factors are to be considered in the indications for operation and in the technique for carrying it out. As to whether operation is indicated at all, it must be remembered that, as far as use is concerned, the patient uses the hand usually quite well despite the cosmetic and unusual anatomy. In some subjects, the webbing may be so severe and the distortion of the digits so grotesque as to render the hand useless. Again the surgeon must study each hand most carefully to learn as much as possible about nerve and tendon involvement in the process and the condition of the bones and joints. When it would appear that only one tendon serves two fused digits, the wisdom of separating them, ex-

cept for cosmetic reasons would seem questionable. The age at which separation is indicated is important. Parents bring their children in soon after birth and the request for surgery is often quite urgent. However it must be remembered that these operations usually require skin grafting and splinting and much after-care: that, in the tiny child, splinting is most difficult; that the little hand can be immobilized only with difficulty; and that grafts are displaced by wiggling fingers. Unless there is gross distortion or the longer digits are being distorted by the shorter ones it is best to delay operation until the child has reached the age of four or five years, or even older.

Separation of the fingers is a plastic procedure based on a few definite principles. The first lesson the surgeon must learn is that it is seldom if ever possible to fashion flaps of skin from the webs to cover the defects left by separation of digits. The many ingenious flaps devised in the past look beautiful and most logical on paper. In practice they never work. There is never enough skin to cover the raw surfaces and suture lines under tension always spread and lead to raw surfaces which heal with scar and subsequent contracture.

Occasionally in a young child in whom digital distortion is occurring, early operation is justified to release digits which are being pulled into deviation, are being pulled down to the level of shorter digits or in which the tips of the fingers and thumb are fused. In the early operation, grafts must be used and since they do not keep pace with the growth of the hand, they must be replaced later.

In all other instances, the surgeon must start out by realizing that a free full thickness skin graft will be required. A tongue or flap of skin should be fashioned from the web, either anteriorly or posteriorly to fill the web space between the digits. This tongue of tissue is brought forward or dorsally and forms the base of the web. After it has been formed, the remainder of the web is divided in a wavy line and the digits are separated. This procedure leaves triangular defects on either side which are covered with free full thickness grafts.

An alternate procedure is to excise the whole of the web and fill it with a free full thickness skin graft shaped somewhat butterfly fashion. Care is taken that the lines of suture on the digits are wavy or zigzag, not straight, otherwise, contractile scars will develop.

Polydactylism or Duplication of Digits. This condition is often symmetrical and usually has a hereditary history. It is frequently associated with deformities of the feet. It ranges in manifestation from simple dichotomy of a digit to a so-called mirror hand. The digits most often involved are the little finger, the middle and ring fingers, the thumb or the index finger in that order of frequency.

In the case of the little finger the extra digit may be represented by a tiny flabby nubbin of skin and subcutaneous tissue or there may be complete separation of two complete digits. In the case of the middle and ring fingers the situation is usually complicated by syndactylism (Fig. 77) the polydactylism being unsuspected until x ray films are taken showing duplication of parts of the phalanges. Hyperphalangism, or polydactylism of the thumb is interesting from the speculative standpoint as to origin, since the thumb is the only digit coming from the radial bulb. Various degrees of polydactylism of the thumb are seen even as many as three thumbs on one hand have been recorded. There may be associated syndactylism, triphalangeal thumbs (Fig. 78) and extra metacarpals.

Double major bulbs produce the so-called mirror hand. In this condition there may be two ulnar elements with absence of the radial element. The sets of fingers are set at an angle to each other in such a way that they appear to be the mirror image of each other.

Treatment of polydactylism in its various forms may be simple or quite complex. The tiny flabby nubbins not infrequently seen in the newborn may be snipped off without any qualm however when the more serious deformities are met with, the problem becomes one of determining the muscle and tendon attachments of the apparently acces-

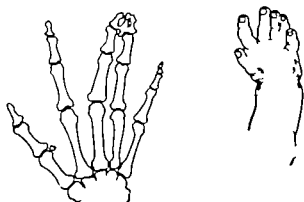


Figure 77 Polydactylism (Kanavel, A. B.: Arch. Surg., vol. 25)

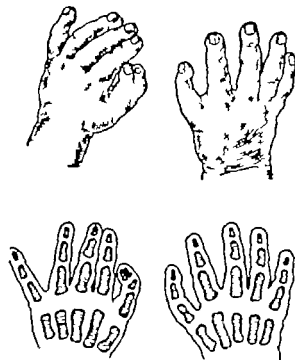


Figure 78 Tripalangeal thumb (modified from Müller: Die angeborenen Fehlbildungen der menschlichen Hand, Thieme.)

sory digits. It may be wise in some instances to leave the hand alone since function is more important than cosmetic appearance. Tendon transference may come into consideration. Bifid digits may often be improved in appearance if such an operation does not destroy function. Tendon transference has been described by Bilhaut-Cloquet for the bifid thumb.

Hypertrophy. Hypertrophy of a digit is rare and may be due to bone growth or to neurofibromatosis. When bone growth is the cause treatment is not indicated except for cosmetic reasons. If neurofibromatosis is present, the soft excess may be removed.

Arachnodactylism (Spider Fingers). This is seen as a definite entity in association with certain endocrine disturbances.

Multiple Cartilaginous Exostoses. These are occasionally found as congenital lesions on the hand as on other parts of the skeleton.

RECONSTRUCTIVE SURGERY

The problems which are encountered by the surgeon who carries out reconstructive surgery of the hand are many and varied. These range from the correction of scar contractures through nerve and tendon reconstruction, bone replacement, tendon transferences and digital reconstruction and transference.

The surgeon who undertakes this work must have first of all a concept of the func-

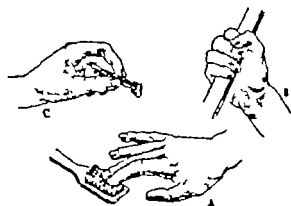


Figure 79 The hand possesses three basic functions: sensation (A), grasp (B) and pinch (C). Without sensation, a hand must be under constant surveillance of the eye. Grasp and pinch, even if limited, give considerable function to a hand, provided sensation is present.

tions (Fig. 79) of the hand and an appreciation of the basic motions. He must be able to visualize the anatomic structure of the part and have the diagnostic acumen to detect the basic loss and to recognize the potentialities of what is left. He must be able to plan a series of procedures, often requiring months or even years of effort and several stages, to restore even minimal use to a hand. He must appreciate that function and not cosmetic appearance is the end result to be secured.

It must be remembered that only in a certain percentage of cases is a single element of difficulty present, frequently more than one problem is present and these must usually be solved individually.

The *sine qua non* for any reconstructive surgery of the hand is intact, healthy pliable skin. As the result of burns, crushing wounds, infection and other causes, the skin may be lost or seriously damaged, so that when the patient presents himself for care there are serious contracted scars which interfere with the use of the part. Burn scars on the dorsum may pile up keloid tissue and prevent proper flexion of the digits. A similar scar between the thumb and second metacarpal may draw the thumb into extreme adduction. Scars on the volar surface may hold one or more or all digits in acute flexion.

These scars may be the only element leading to disability; the structures beneath them may be undamaged or relatively so. In such cases it will only be necessary to correct the cutaneous scar by some type of skin replacement to restore the hand to use (Fig. 80).

If, however, along with the damage to the skin there is deeper damage to nerves, ten-

doms and bones the skin replacement must be such that the surgeon can subsequently lift it up along one side and carry out needed surgical repairs beneath it. In such instances a free graft will not be satisfactory and a pedunculated flap of skin with subcutaneous fat beneath it and carrying its own blood supply will be necessary.

Also, if the excision of scar contracture of the skin entails uncovering of bones, joints, nerves and tendons it is usually necessary to apply a pedunculated flap to replace the skin excised. Determination of whether or not such a flap will be needed often requires very critical judgment and the surgeon must occasionally be prepared to use whichever seems indicated at the time of operation.

At times the functional loss may be due to loss or division of tendons and/or nerves (Fig. 81). Here, the first problem is to determine the extent of the damage—that is a diagnosis of the tendons and nerves involved must be made. The second problem will be to determine just how repair may be carried out.

Attention has been called to the fact that, when deep repair must be undertaken, the covering tissues must be supple and there must be adequate subcutaneous fatty tissues through which to work. Therefore, the first job will be to assure oneself that the skin through which surgery must be carried out is satisfactory. This may require, therefore, that before any nerve and tendon repair can be carried out a skin flap will have to be applied to the part.

Again, the joints which are to be moved by the newly repaired tendons must be mobile. Mobilization of joints may in itself require long periods of splinting, physical therapy and open operation before the tendon repair can be undertaken.

Lastly some decision must be reached as to the type of repair, i.e., tendon suture, ten-

don graft or tendon transference. The first problem is that of nerve and tendon division in the palm. The problem is not simple, but there are probably fewer factors to be considered than, for instance, in division of the digits. If the patient is seen quite early and there has been no infection and no unsuccessful attempts were made at repair at the time of injury the factor to be considered is the amount of tendon retraction. Under ideal circumstances, retraction in a few weeks will not have been very great and the surgeon can feel assured that in all probability a suture of all tendons can be carried out, certainly of the profundus tendons and that the nerves may likewise be repaired. The important factor is the amount of retraction which the proximal stumps have undergone. In the early case, this will be minimal.

If on the other hand, the tendon injury occurred six months or a year previously the forearm muscles will have retracted a good deal and, under such circumstances, it is quite likely that tendon grafts will be needed.

Another factor is introduced when one comes to consider repair of tendons in the digits. Whether immediate repair was either not carried out or was attempted and failed, there is only one answer to the problem and that is the introduction of a tendon graft. There is only one possible exception to this rule, which does not often obtain, and that is profundus tendon division alone. If division has occurred close to the distal joint, the amount of tendon retraction may be so minimal that a suture may occasionally be feasible. Usually however the proximal retraction will be so great that a tendon graft will be required.

Division of the flexor pollicis longus may or may not demand a tendon graft. The proximal stump frequently "snaps" up into the lower forearm, becomes bulbous and short

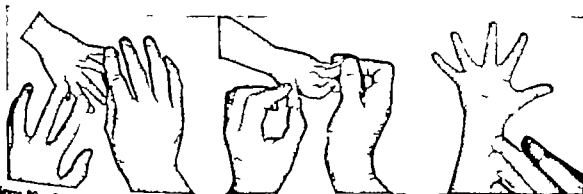


Figure 80. Scar contractures involving only the skin may be corrected by complete excision and replacement with a free full thickness skin graft.

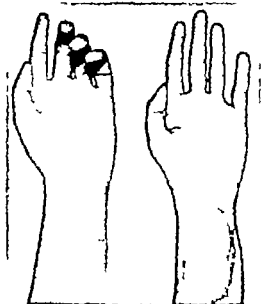


Figure 81 Deep scars involving tendons, particularly when nerve and tendon repairs are required, must be replaced by a pedunculated flap to furnish suitable coverage for subsequent surgery

ened and its distal end may degenerate and become friable and necrotic.

Division of the flexor tendons in the wrist and lower forearm, unless immediately repaired, presents one of the most unfortunate situations to correct. The median nerve is always divided and often the ulnar nerve also. A perfectly useless hand results which is not only without motion, but also without sensation. The forearm muscles retract with alarming rapidity the hand assumes a position which is difficult to prevent, even with splinting, and the most careful of repairs can promise but mediocre improvement. It is for this reason that primary suture of nerves and tendons should be undertaken in these patients whenever possible.

Secondary repair of the nerves and tendons above the wrist will eventually require tendon grafts not infrequently five grafts will be necessary one for each profundus tendon and for the flexor pollicis longus.

Division of tendons over the dorsum of the wrist presents a similar problem in that the proximal tendon stumps in synovial sheaths retract a good distance up the forearm and, within a short time, it is not possible to carry out suture. Tendon grafts are often required.

The extensor pollicis longus is usually best repaired by tendon transference. The course of the tendon through its tunnel on the dorsum of the wrist is so irregular that suture within it would be certain to end in failure. Therefore, a neighboring tendon stump

either the extensor carpi radialis brevis or, preferably the extensor indicis proprius is used.

Division over the dorsum of the metacarpus may usually be repaired by suture unless there has been actual loss of tendon substance. The paratendon through which the tendons pass in this portion of their course tends to prevent too great retraction.

Injuries of tendons over the dorsum of the fingers present complex problems varying from phalanx to phalanx. Division over the dorsum of the metacarpophalangeal joint is usually amenable to suture even when seen late. The attachment of tendon to joint capsule prevents too great separation and the gap between the divided capsule and tendon ends fills with tendon callus. It is usually possible to dissect out this callus, freshen up the tendon ends and capsule and carry out satisfactory suture.

Tendon transferences are frequently required when paralytic conditions of one type or another cannot be remedied by nerve repair. The most common condition met with requiring transferences is in patients having median and radial paralyses. In median nerve paralysis, the short muscles of the thumb may fail to recover after median nerve suture. This makes it impossible for the patient to rotate the thumb about into the position of function or apposition for grasping. The thumb lies back against the side of the hand and can grasp only against the side of the index finger. It is advantageous in this condition to transfer a tendon, frequently the sublimis flexor of the ring finger from the lower forearm subcutaneously across the thenar eminence to the thumb in the region of the metacarpophalangeal joint. This tendon will then pull the thumb out in abduction and apposition to face the other digits (Fig. 82).

In radial nerve paralysis, in which for some reason or other repair of the nerve is not possible it is customary to transfer one or both of the carpal flexors onto the dorsum of the wrist and insert it or them into the common digital extensors and into the extensor of the thumb.

One other transference which is occasionally carried out is valuable in ulnar paralysis in which, despite nerve repair or when repair is not possible, the interossei fail to regenerate. In this situation, the sublimis tendons of two fingers are divided at their insertions and slips are led around the sides of the finger to be inserted into the lateral slips of the extensor expansion. If transference is

successful, these slips will extend the interphalangeal joints.

Many other tendon transfers are practiced. In most instances, individual indications obtain in each case and the surgeon must determine what motions are most important and which motors are present to make those motions possible.

Problems with regard to the bony framework of the hand likewise occur. If they are present, they take precedence over every other repair except that of the skin which must be satisfactory to permit deep repairs on the bones. Correction of malunions, consolidation of nonunions and bone replacement by bone graft may be needed to restore satisfactory framework.

It may occasionally be obvious that active motion can never be secured in certain joints, and that it would be better to fuse them in a functional position. Thus it may be indicated to swing the thumb around into a position of abduction and apposition to face the pads of the other digits and fix it there by means of a bone block between the first and second metacarpals. Occasionally the interphalangeal joints may be so badly damaged that active motion is not likely to be restored. In these instances it may seem wise to fuse them by one means

or another in a slightly flexed position, thus permitting the digits to be flexed as a whole at the metacarpophalangeal joints.

The surgeon must have some conception of the basic functions of the hand before he can intelligently carry out any sort of reconstructive surgery. He must start out with the concept that the hand is fundamentally a grasping and sensory organ. From these two simplest mechanisms of the hand, the functions are built up gradually becoming more and more complex until the fully developed and trained human hand guided by a trained intelligence is reached. Following the function of grasping will come another simple function—that of pinching. These three functions, grasp pinch and feeling, should be the minimum which the surgeon should strive to restore. However this does not mean to imply that finer and more complicated actions should not be restored if the possibility exists. Thus it may be possible to restore the individual flexion and extension of the digits by various tendon transfers or tendon grafts. Abduction and adduction of the fingers so necessary in certain fine movements may be partially or completely restored by certain operative procedures.

The loss of the thumb which renders grasp impossible, is very serious and many procedures have been devised to compensate for this disability. In some patients it may be possible to restore grasp provided the metacarpal and a bit of the proximal phalanx are left, by deepening the cleft between the first and second metacarpals. This will render the thumb remnants more mobile and will often make a most satisfactory buttress against which the fingers can grasp objects. In instances in which a fair remnant of the thumb is left and the index finger is gone, the cleft may be further deepened by removal of the second metacarpal.

If there is no thumb remnant left, or if what little is left is too small to be serviceable, various procedures have been carried out to build up a new thumb. These have consisted essentially in the formation of a tubular graft of skin into which a bone or rib cartilage graft is inserted to give stability. While these thumbs occasionally function fairly well, often they do not. They are usually without sensation and are immobile, ugly appendages which patients would be glad to be rid of.

In a few instances surgeons have successfully reported the grafting of a toe to the hand to replace a lost thumb. The procedure

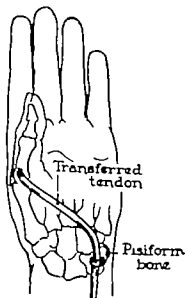


Figure 82. Rotation of the thumb may often be secured by transfer of a tendon, usually the sublimis flexor of the ring finger to the region of the metacarpophalangeal joint. The transferred tendon is led through a fascial loop about the flexor carpi ulnaris or may simply be passed beneath the flexor carpi ulnaris so that the line of pull is from the region of the pisiform bone. This is a very valuable procedure when the motor function of the median nerve has not returned and the small muscles of the thenar eminence are paralyzed.

would be applicable only in youngsters who could withstand the long period of awkward and tiring immobilization necessary for such a graft to take. Likewise, the tissues must be young and well vascularized to withstand such procedures.

A more ingenious procedure described by Hilgenfeldt, and later popularized by Littler. This consists in transferring another digit of the same hand to the location of the thumb. To be successful, it is necessary that some of the base of the metacarpal be present into which the metacarpal of the transferred digit is fixed. The digit to be transferred is isolated along with its nerve vascular supply and flexor tendons intact, and set onto the thumb metacarpal. A number of such successful cases have been reported.

Before procedures of this sort are to be carried out, the surgeon must make sure that the patient will benefit from them and that he cannot carry on satisfactorily without a new thumb. Hilgenfeldt has advised that patients be permitted to carry on for about a year with the hand before the operation is performed. Many times the patient will decide against the procedure.

While there is no doubt that the thumb is a most valuable digit, there may be some question as to the exalted position it has been given. Certainly the surgeon must make every effort to save all length possible and to restore to every possible function whatever remnants there are of the thumb. However when this digit is irretrievably gone, there is some question as to whether its loss is as great a handicap as we have been prone to think. Hilgenfeldt has given some good advice concerning replacement of the thumb. He has counseled waiting a year or at least a number of months before making the decision. During this time, the patient at tempts to get along with the hand, carries on his work and the general acts of daily life. If at the end of that time, the thumb loss is still a serious handicap operative restoration is undertaken. It is often surprising to see how well a patient can get along without a thumb. Many functions of the hand, awkward at first, are easily carried out and adequate substitute motions are developed.

If there is a good thumb on the opposite hand, the indications to restore the thumb are much less urgent than in instances in which the thumb has been lost bilaterally.

Age and occupation of the individual should be taken into account, as well as those other factors which the surgeon must con-

sider when starting off on a long series of operations or a course of treatments which will require the fullest cooperation of a willing and honest patient. Many patients are reduced to dependent loafers by apparently justifiable attempts to restore a moderate increase in function to an already fairly useful hand.

Finally, one must always keep in mind the surgical risk itself. There is always the possibility that the transferred digit may not survive—not great, to be sure, with skilled operative technique—but, if gangrene does develop the patient will not be just without a thumb, but also without another digit. If this has been a normally functioning finger the tragedy is great.

The surgeon who carries out reconstructive surgery of the hand must be skilled in peripheral nerve repair since it is part and parcel of hand surgery.

READING REFERENCES

- Allen, H. S., *Winger Injuries of the Upper Extremity*. Ann. Surg. 113:1101, 1941.
- Allen, H. S., *Flexor Tendon Grafting to the Hand*. Arch. Surg. 63:362, 1951.
- Allen, H. S., *Management of Lacerations of Flexor Tendon within the Digits*. S. Clin. North America 35:189, 1955.
- Allen, H. S. and Mason, M. L., *A Universal Splint for Immobilization of the Hand in the Position of Function*. Quart. Bull. Northwestern Univ. M. School 21:218, 1947.
- Banks, A. J., *Congenital Anomalies of the Hand*. J. Bone & Joint Surg. 33-A:35, 1951.
- Bell, C., *The Hand—Its Mechanism and Vital Endowments as Evincing Design*. London, William Pickering, 1833.
- Bickel, W. H., Kimbrough, R. F. and Dahlin, D. C., *Tuberculous Tenosynovitis*. J.A.M.A. 151:31, 1953.
- Blackberg, B., and Fec, J., *Syndrome of Median Nerve Compression in the Carpal Tunnel*. Acta orthop. scandinav. 26:120, 1956.
- Bohr, H. H., *Tendon Transposition in Paralysis of the Opposition of the Thumb*. Acta chir. scandinav. 163:45, 1953.
- Bolton, H., Fowler, P. J., and Jepson, R. P., *Natural History and Treatment of Pulp Space Infection and Osteomyelitis of the Terminal Phalanx*. J. Bone & Joint Surg. 31 B:499, 1949.
- Boyes, J. H., *Flexor Tendon Grafts on the Fingers and Thumb*. J. Bone & Joint Surg. 33-A:439, 1950.
- Boyes, J. H., *Dupuytren's Contracture: Notes on the Age at Onset and the Relationship to Handedness*. Am. J. Surg. 88:147, 1954.
- Bristow, W. R., *Injuries of Peripheral Nerves in Two World Wars*. Brit. J. Surg. 34:333, 1947.
- Brooks, B., *Experimental Study of Volkmann's Paralysis*. Arch. Surg. 15:188, 1922.
- Brown, J. B., McDowell, F., and Fryer, M. P., *Radiation Burns Including Vocational and Atomic Exposures, Treatment and Prevention of Chronic Lesions*. Ann. Surg. 130:593, 1949.

- Brown, J. B., McDowell, F., and Fryer M. P.: Surgical Treatment of Radiation Burns. *Surg. Gynec. & Obst.* 93:335, 1951.
- Bruser, J. M.: Incisions for Plastic and Reconstructive (Non-Septic) Surgery of the Hand. *Plast. & Reconstruct. Surg.* 4:48, 1951.
- Bruser, J. M.: Problems of Postoperative Position and Motion in Surgery of the Hand. *J. Bone & Joint Surg.* 35-A:355, 1953.
- Bonell, S.: Repair of Tendons in the Fingers. *Surg. Gynec. & Obst.* 35:88, 1922.
- Bonell, S.: Reconstructive Surgery of the Hand. *Surg. Gynec. & Obst.* 39:259, 1924.
- Bonell, S.: Injury of the Nerves of the Hand. *Surg. Gynec. & Obst.* 44:145, 1927.
- Bonell, S.: Surgery of the Hand, 3rd ed. Philadelphia, J. B. Lippincott Company 1936.
- Bonnan, M.: Stenosing Tendonitis of the Dorsal and Volar Compartments of the Wrist. *Arch. Surg.* 65:752, 1932.
- Carp, L.: The Distal Anterior Closed Space Infection. *Surg. Gynec. & Obst.* 46:484, 1928.
- Casley E. P., Whitmore C. W., and Wheeler C. E.: Mollus Nodules. *South. M. J.* 46:21, 1933.
- Cohen, R. R.: deQuervain's Disease. *J. Bone & Joint Surg.* 33-B:96, 1951.
- Conway H.: Dupuytren's Contracture. *Am. J. Surg.* 87:101, 1954.
- Cotler C. W., Jr.: The Hand—Its Disabilities and Diseases. Philadelphia, W. B. Saunders Company, 1942.
- Devis, L.: The Return of Sensation to Transplanted Skin. *Surg. Gynec. & Obst.* 59:533, 1934.
- Duperion, S. M.: An Evaluation of Skin Grafts for Hand Coverage. *J. Bone & Joint Surg.* 34-A:811, 1952.
- Duperion, S. M.: Skin Grafts in Reconstructive Surgery of the Hand. *Plast. & Reconstruct. Surg.* 12:107, 1953.
- Edgerton, M. T.: Immediate Reconstruction of the Injured Hand. *Surgery* 36:329, 1954.
- Eyles, D. and Markee, J. E.: The Anatomy and Function of the Intrinsic Musculature of the Fingers. *J. Bone & Joint Surg.* 36-A:1, 1954.
- Felander, M.: Tuberculous Tenosynovitis of the Hand Treated by Combined Surgery and Chemotherapy. *Acta chir. scandinav.* 111:142, 1956.
- Flynn, J. E.: Acute Suppurative Tenosynovitis of the Hand. *Surg. Gynec. & Obst.* 76:237, 1943.
- Flynn, J. E.: Open Wounds of the Hand. *New Eng. J. Med.* 250:605, 1954.
- Folsie, P. S.: Volkmann's Ischemic Contracture. *New England J. Med.* 226:671, 1942.
- Gall, B.: Dupuytren's Contracture. *Ann. Surg.* 107:122, 1938.
- Gordon, S.: Dupuytren's Contracture. The Significance of Various Factors in Its Etiology. *Ann. Surg.* 140:683, 1954.
- Goffin, D. L.: Volkmann's Ischemic Contracture. *Brit. J. Surg.* 28:239, 1940.
- Geib, H.: Über die Behandlung der Sehnenverletzungen der Hand. *Zschr. Orthop.* 88:121, 1956.
- Hambley-Jones, R. M.: Surgery of the Hand, 2nd ed. Baltimore, Williams & Williams Company 1948.
- Harris, C., and Riordan, D. C.: Intrinsic Contracture in the Hand and Its Surgical Treatment. *J. Bone & Joint Surg.* 36-A:10, 1954.
- Hoppe, M. F.: The Results of Tendon Suture of the Hand. *Acta orthop. scandinav.* 24:258, 1955.
- Horton B. T., and Ghormley R. K.: Congenital Arteriovenous Fistulae of the Extremities Visualized by Arteriography. *Surg. Gynec. & Obst.* 60:978, 1935.
- Hutchinson J.: Melanotic Whitlow. *Brit. M. J.* 1:49, 1886.
- Kanavel A. B.: Congenital Malformations of the Hands. *Arch. Surg.* 25:1, 282, 1932.
- Kanavel, A. B.: Infections of the Hand, 7th ed. Philadelphia, Lea & Febiger 1938.
- Kaplan, E. B.: Functional and Surgical Anatomy of the Hand. Philadelphia, J. B. Lippincott Company 1933.
- Kirklin, J. W. and Thomas, C. G.: Opponents Transplant: An Analysis of the Methods Employed and Results Obtained in Seventy Five Cases. *Surg. Gynec. & Obst.* 86:213, 1948.
- Kirkaldy, W. S.: Use of Flaps and Pedicles in the Repair of Hand and Arm Defects. *American Academy of Orthopedic Surgeons, Lectures, 1944* pp. 180-183.
- Koch S. L.: Immediate Treatment of Injuries of the Hand. *Surg. Gynec. & Obst.* 52:595, 1931.
- Koch, S. L.: Acute Rapidly Spreading Infections Following Typical Injuries of the Hand. *Surg. Gynec. & Obst.* 59:277, 1934.
- Koch, S. L.: Osteomyelitis of the Bones of the Hand. *Surg. Gynec. & Obst.* 64:1, 1937.
- Littler J. W.: The Hand and Wrist. In Howarth, M. B.: A Textbook of Orthopedics. Philadelphia, W. B. Saunders Company 1952.
- Marble H. C.: Purposeful Splinting Following Injuries to the Hand. *J.A.M.A.* 116:1373, 1941.
- Mason, M. L.: Rupture of Tendons of the Hand. *Surg. Gynec. & Obst.* 50:611, 1930.
- Mason, M. L.: Tuberculous Tenosynovitis of the Hand. *Surg. Gynec. & Obst.* 69:363, 1934.
- Mason, M. L.: Tumors of the Hand. *Surg. Gynec. & Obst.* 64:129, 1937.
- Mason, M. L. and Allen, H. S.: Rate of Healing of Tendons. *Ann. Surg.* 113:424, 1941.
- Mason, M. L., and Bell, J. L.: The Treatment of Open Injuries to the Hand. *S. Clin. North America* 36:1337, 1956.
- Mason, M. L., and Shearon, C. G.: The Process of Tendon Repair. *Arch. Surg.* 25:615, 1932.
- May H.: Tendon Transplantation in the Hand. *Surg. Gynec. & Obst.* 83:631, 1946.
- Meleny F. L., and Johnson, B. A.: The Clinical Significance of the Increasing Resistance of Organisms to the Antibiotics. *Surg. Gynec. & Obst.* 97:267, 1953.
- Meyerding, H. W., Black, J. R., and Broders, A. C.: Etiology and Pathology of Dupuytren's Contracture. *Surg. Gynec. & Obst.* 72:582, 1941.
- Miller H., and Winfield, J. M.: Human Bites of the Hand. *Surg. Gynec. & Obst.* 74:153, 1942.
- Nichols, H. M.: Manual of Hand Injuries. Chicago, Year Book Publishers, 1953.
- Parkes, A.: The Treatment of Established Volkmann's Contracture by Tendon Transplantation. *J. Bone & Joint Surg.* 31-B:359, 1951.
- Pimm, L. H., and Waugh, W.: Tuberculous Tenosynovitis. *J. Bone & Joint Surg.* 39-B:91, 1957.
- Poehj, J. L.: Injuries of the Hand. *S. Clin. North America* 33:1081, 1953.
- Poehj, J. L.: Primary Tenorrhaphies and Tendon Grafting Procedures in Hand Injuries. *Arch. Surg.* 73:609, 1956.

- Poeh, J. L., and Weller, C. N. Mangle and Severe Wringer Injuries of the Hand in Children. *J. Bone & Joint Surg.* 36-A:57, 1954.
- Pratt, D. R., Bunnell, S., and Howard, L. D. Mallet Finger. *Am. J. Surg.* 93:573, 1957.
- Pulvertaft, R. G. Repair of Tendon Injuries of the Hand. Hunterian Lecture. *Ann. Roy. Coll. Surg. Great Britain* 3:3, 1948.
- Reed, J. V., and Harcourt, A. K. Immediate Full-Thickness Grafts to Finger Tips. *Surg. Gynec. & Obst.* 68:625, 1939.
- Riordan, D. C. Tendon Transplantation in Median-Nerve and Ulnar Nerve Paralysis. *J. Bone & Joint Surg.* 35-A:312, 1953.
- Rivers, M., and Pack, G. T. The Glomus Tumor. *Ann. Surg.* 133:394, 1951.
- Siler, V. E. Primary Tenorrhaphy of the Flexor Tendons of the Hand. *J. Bone & Joint Surg.* 32-A:318, 1950.
- Skoog, T. Dupuytren's Contracture with Special Reference to Etiology and Improved Surgical Treatment, Its Occurrence in Epileptics; Note on Knuckle-Pads. *Acta chir. scandinav. (suppl.)* 130:98, 1948.
- Steenrod, E. J., Ghormley, R. L., and Craig, W. M. Injuries of the Hands Due to Shattered Porcelain Handles of Water Faucets. *Surg. Gynec. & Obst.* 64:650, 1937.
- Tanzer, R. C. Dupuytren's Contracture. *New England J. Med.* 246:807, 1952.
- Teloh, H. A., Mason, M. L., and Wheelock, M. C. Histopathological Study of Radiation Injuries of the Skin. *Surg. Gynec. & Obst.* 90:335, 1950.
- Tempest, M. N. The Emergency Treatment of Digital Injuries. *Brit. J. Plast. Surg.* 7:153, 1954.
- Verdan, C. Chirurgie des Lésions Traumatiques Recentes de la Main. *Helvet. chir. acta* 23:411, 1956.
- Webster, G. V. Late Repair of Tendons of the Hand. *Am. J. Surg.* 72:171, 1946.
- Webster, G. V., and Roland, W. D. Skin-Grafting the Burned Dorsum of the Hand. *Ann. Surg.* 124:449, 1946.
- Wood-Jones, F. W. The Principles of Anatomy as Seen in the Hand. London, J. & A. Churchill, 1930.

THE FOOT

By WILLIAM A LARMON M D

WILLIAM ALEXANDER LARMON born in California received his collegiate and medical education at Northwestern University where he is now an Assistant Professor of Orthopedic Surgery Dr Larmen has been responsible for writing and producing a series of teaching color motion pictures for the Veterans Administration on the principles of the treatment of fractures His teaching abilities are reflected in his contribution to this volume

The surgeon must be mindful of the complex relationships of the foot to the rest of the body. The foot is the connecting link with our terrestrial environment, balancing the body in the upright posture and propelling it about. Proper function of the foot depends upon the integrity of the musculoskeletal and nervous systems for balance and movement, the circulatory system for nourishment and the skin and subcutaneous tissues for protection and covering. Pathologic changes in any of these systems may affect the foot and alter or limit our ability to stand, walk or run.

FUNCTIONAL ANATOMY

Motion and Positions of the Foot. To treat successfully pathologic conditions of the foot, the surgeon must understand the normal anatomy and physiology of the lower extremity.

Movements and positions of the foot are traditionally described with the foot off the ground and unencumbered. When the dorsum of the foot is brought toward the anterior surface of the leg, the movement is dorsiflexion and the position of the foot is termed calcaneus. The opposite movement is plantar flexion and the foot is in the equinus position. These motions take place at the tibiotalar joint. When the heel is in-

clined toward the midline of the body it is inverted and in varus position. The opposite is eversion and valgus position. This motion takes place in the joint between the talus and the os calcis or subtalar joint. When the forefoot is brought toward the midline of the body, it is adducted. The opposite movement and position place the forefoot in abduction. These motions take place in the calcaneocuboid and talonavicular joints as well as in the joints of the other tarsal bones.

The subtalar calcaneocuboid and talonavicular joints work as a unit producing supination of the foot when the plantar aspect is directed toward the body midline. Pronation of the foot is found when the sole of the foot is directed away from the mid plane of the body. Varus and valgus positions of the foot are synonymous with supination and pronation.

When the toes are brought toward the plantar surface of the foot, they are flexed, and when in the opposite position, they are extended.

The cardinal positions of the foot are calcaneus, equinus, varus and valgus. Various combinations of these terms describe the positional deformities that occur in the foot. Thus, talipes equinovarus points out the abnormal position of clubfoot.

The Arches of the Foot. The act of stand

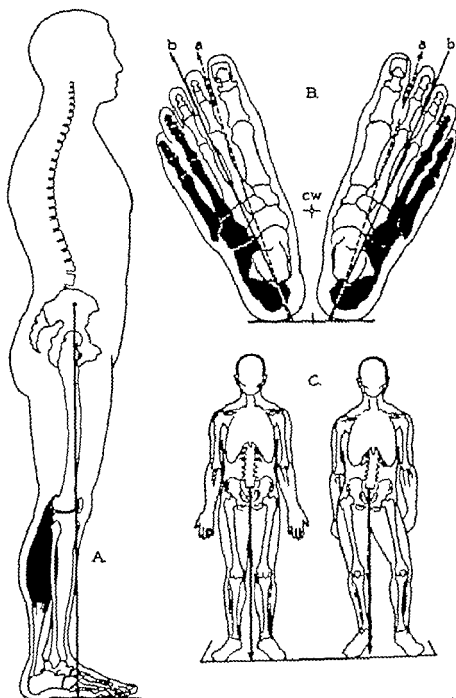


Figure 1 The center of gravity of the body is located at the second sacral vertebrae (A) Weight of the body falls between the feet at the level of the navicular bone CW. When standing the body sways forward periodically. Contraction of the triceps surae (A) pulls the tibia posteriorly on the foot to maintain balance. Ligamentous strain in the foot is relieved by shifting body weight from one foot to the other frequently (C). The medial half of the foot is mobile (B) while the lateral half is a stable platform. The axis of leverage in walking is line a. The axis of medial and lateral balance is line b (redrawn from Morton, D. J. *The Human Foot*, Columbia University Press)

ing demands stability and balance in the structures of the foot and walking and running require mobile, elastic, shock absorbing properties. The architecture of the foot fulfills all of these functional requirements.

The hind foot, composed of the talus and the massive os calcis, is adapted to support body weight. The midtarsal bones, the meta-

tarsal bones and the phalanges are more mobile and are adapted to the function of balance and propulsion.

The medial and lateral halves of the foot are also designed for special purposes (Fig. 1B). The os calcis, cuboid and fourth and fifth metatarsal bones with their phalanges form a stable platform in contact with the

ground. The medial half of the foot composed of the talus, navicular, cuneiform bones, and the first, second and third metatarsal bones and phalanges form an arched, elastic and mobile structure necessary for balance and propulsion.

The arches of the foot have been likened to a stone bridge, the fit and shape of the bones of the foot maintaining a rigid arch. Such is not the case (Fig. 2A). The medial longitudinal arch is composed of bones with curved articulations which in no way resemble the keystone shape of the stones in a bridge. These curved joints held together and supported by strong ligaments, tendons and muscles, provide elasticity and mobility necessary for the function of the foot. The longitudinal arch of the foot is more accurately described as a bow or spring. The plantar structures composed of fascia, ligaments, tendons and muscles are the bow strings.

The transverse arch of the foot exists at the metatarsal cuneiform joints but gradually flattens as the heads of the metatarsal bones are approached (Fig. 2B-D). All of the metatarsal heads in the normal foot make equal contact with the ground when weight is borne and no true arch exists at this point. The transverse arch is stable at the metatarsal cuneiform joints as these

bones more nearly resemble the keystones of the stone bridge. The configuration of these joints provides stability.

The Ligaments. The bones in the foot are joined by ligaments which are thickened areas in the joint capsules. In addition to the ligaments of the individual joints there are two major ligaments and the plantar aponeurosis which act as bow strings to aid in maintaining the arches (Fig. 3A). One of these—the calcaneonavicular ligament—arises near the sustentaculum tali on the calcaneus and inserts into the plantar aspect of the navicular bone. This ligament passes under the head of the talus as a sling, providing support but not restricting motion of the talonavicular joint. It prevents the talus from excessive plantar flexion at the midtarsal joints thus preserving the longitudinal arch.

The second important ligament is the long plantar arising near the tuberosities of the calcaneus and inserting into the cuboid and the base of the metatarsal bones (Fig. 3B). This ligament supports the midportion of the longitudinal arch, approximating the metatarsal bases to the calcaneus, and is a second string in the bow.

The third structure—the plantar aponeurosis—arises at the tuberosities of the os calcis and inserts into the toes on the plantar as-

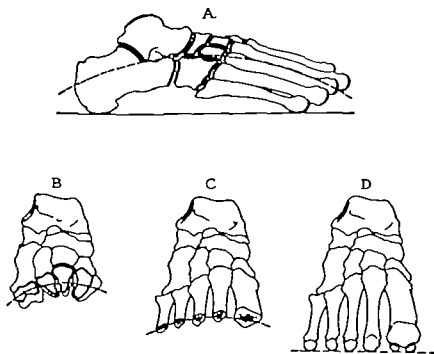


Figure 2. A, The longitudinal arch of the foot is made up of bones with curved articular surfaces adapted for complex movements. Ligaments must maintain the relationships of these surfaces to preserve the arch. B, The transverse arch is well defined at the base of the metatarsal bones. The articulations are wedge shaped and stable. C, The transverse arch flattens in the midmetatarsal area. D No arch is present at the head of the metatarsal bone. Body weight is carried by all of the metatarsal bones. When one bone becomes prominent in the sole of the foot, more weight is borne by the involved bone and pressure symptoms occur.

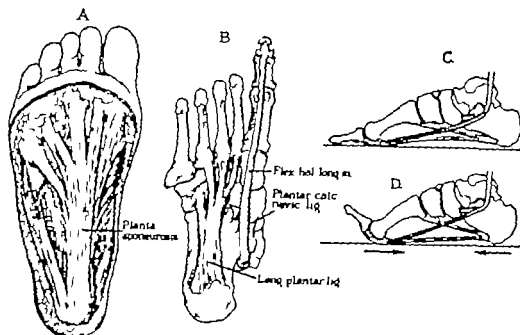


Figure 3 A, The plantar aponeurosis aids in support of the longitudinal arch. B, The long plantar and the calcaneonavicular ligaments support the midpoint of the arch. The calcaneonavicular ligament acts as a sling under the head of the talus. The pulley like arrangement of the tendons on the plantar aspect of the foot running beneath the malleolus produces active support for the arch. C and D, The heads of the metatarsal bones act as a pulley for the plantar aponeurosis when the toes are dorsiflexed. This shortens distance between the metatarsal heads and the condyles of the os calcis and raises the arch. This mechanism supports the arch at the point of "push-off" in walking (C and D redrawn from Hicks, *J. H. J. Anat.*, vol. 153)

pect (Fig. 3C, D) When the toes are extended, the plantar aponeurosis passing over the rounded metatarsal heads tightens. This mechanism shortens the plantar aponeurosis, approximating the metatarsal heads toward the heel, maintaining the longitudinal arch. This action can be likened to the mechanics of the windlass. The toes are the handle and the metatarsal heads the drum of the windlass; the plantar fascia is the rope pulled over the drum, shortening the distance between metatarsal heads and the heel, raising the longitudinal arch. This takes place when most needed at the point of push-off in walking when maximum strain is thrown on the arch of the foot.

The Muscles. The intrinsic muscles of the foot lend active support to the ligaments in standing and walking. They are arranged to aid in supporting the elastic arches (Fig. 3C and D). Medially the abductor hallucis arising on the os calcis and inserting into the great toe adds another string to the bow. The central muscle group composed of the quadratus plantae, flexor brevis of the toes and the lumbricales, maintains the central portion of the arch.

The long muscles arising in the leg provide the motor power for the gross move-

ments of the foot necessary for balance and locomotion.

The triceps surae or calf group of muscles, composed of the gastrocnemius and soleus muscles, is the prime lifter of the body in walking, through plantar flexion of the foot at the tibiotalar joint. In addition to this action this muscle group inverts the heel at the subtalar joint in plantar flexion (Fig. 4A). This inverting action is prevented by the balanced action of the peroneus longus muscle which contracts synchronously in walking (Fig. 4B). The pulley action of the peroneus longus tendon, passing from the lateral side of the foot at the cuboid bone to the base of the first metatarsal bone, everts the foot in plantar flexion. It also depresses the distal end of the first metatarsal against the ground in walking and standing, thus elevating the arches of the foot and aiding in propulsion.

The dorsiflexors of the foot are the strong tibialis anticus and the weaker extensor digitorum longus. The tibialis anticus inserting into the medial aspect of the foot inverts and supinates the foot, elevating the arch, while the extensor digitorum longus passing lateral to the axis of balance of the foot, tends to evert the foot in dorsiflexion (Fig. 5)

The tibialis posterior passing beneath the pulley of the medial malleolus and inserting into the plantar aspect of the navicular bone inverts the foot and adducts the forefoot. The peroneus brevis, passing beneath the lateral malleolus and inserting into the base of the fifth metatarsal balances this action of the tibialis posterior and produces

eversion of the foot and abduction of the forefoot.

The action of these muscles can be likened to three sets of reins attached to a bridle in providing medial and lateral stability for the foot. The triceps surae and peroneus longus balance each other in plantar flexion. The tibialis posterior and peroneus brevis balance

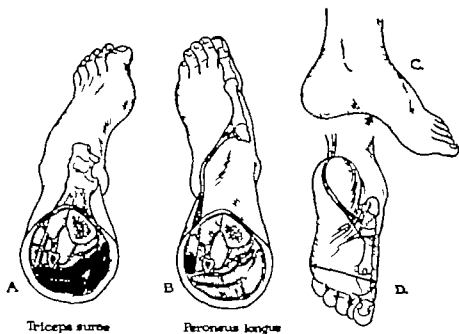


Figure 4 A, The triceps surae muscles produce plantar flexion and inversion, equinovarus position. B, The peroneus longus counteracts the inversion by the triceps surae in plantar flexion. C and D Acting alone it everts the foot in plantar flexion, valgus position and depresses the distal end of the first metatarsal bone, raising the arch and fixing the bone to the ground in walking during the push-off phase.

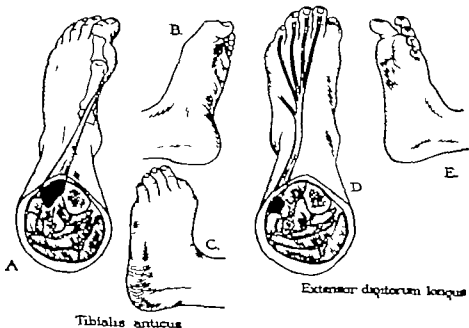


Figure 5 A, B, C, The tibialis anterior dorsiflexes and inverts the foot and elevates the first metatarsal bone, calcaneovarus position. D and E, The extensor digitorum longus is a weak dorsiflexor of the foot as well as the toes. The tendons pass lateral to the axis of balance of the foot and produce eversion of the foot in dorsiflexion, calcaneovalgus position. The two muscles balance each other in pure dorsiflexion of the foot.

the foot in neutral position and the tibialis anticus and the extensor digitorum longus balance in dorsiflexion.

Any disturbance in the function of these muscles and tendons will result in foot imbalance and produce static and dynamic deformities and disturbances in walking.

Standing and Balancing When we stand in the normal position the feet are moderately separated with the toes pointing outward. In this position the body weight is equally divided between the feet. The center of gravity of the body may be assumed to be at the center of the second sacral vertebra (Fig. 1A and B). A plumb line dropped from the center of gravity falls midway between the feet at the level of the navicular bone. Thus the body is supported by two pillars with the center of gravity falling between them. These two pillars are supported on the lever arms of the foot which extend forward of this center of gravity as the forefoot and backward as the heel. Thus, stability is insured through these lever arms forward, backward and from side to side.

Balance is maintained by a minimum expenditure of muscle power. The muscles contract periodically to correct swaying of the body but the majority of the time they are at rest (Fig. 1A). There is a rhythmic forward sway of the body which is counteracted by periodic contraction of the triceps surae muscles and the toe flexors. This forces the forefoot lever against the ground. Since the foot is fixed to the ground, the effect of this force is to pull the tibia backward, bringing the center of gravity back to the normal position. The act of balancing can be clarified if we consider the foot as fixed to the ground. The muscles then act on the leg to maintain its position over the foot. Attempts to plantar flex the foot pull the tibia backward. When one attempts dorsiflexion, the tibia is pulled forward. Inversion force pulls the leg medially and eversion force on the foot pulls the leg laterally.

While this balancing is taking place, the weight passing through the feet is supported by the ligaments. However these ligaments are not strained because we shift position frequently (Fig. 1C). It has been found that the standing person shifts his position about every 30 seconds from one foot to the other and forward and backward on both feet. Seldom does a person stand still for a full minute. This economy of muscle power and shifting of body weight to various ligaments

accounts for our ability to stand for long periods without fatigue.

Weight transmission through the feet while standing has been determined by many investigators. When the center of gravity falls opposite the navicular bone, equal weight is supported by the forefoot and heel. The heel bears half of the body weight, the metatarsal bones bear the other half. The division of weight through the metatarsal heads, however is not equal. The first metatarsal bone supports one-third of the weight borne by the forefoot and the lateral four metatarsal bones share equally the remaining two-thirds. Thus it is apparent that there is no transverse arch on weight bearing at the level of the metatarsal heads. Any disturbance in transmission of this load through these structures will produce static problems in the foot. The typical callus on the plantar aspect of the foot over the distal end of a metatarsal bone signifies the disturbance in weight bearing and indicates overloading of that metatarsal.

This distribution of weight in the long axis of the foot has been termed the *axis of leverage* by Morton (Fig. 1B). However in balancing, the functional axis of the foot falls between the second and third metatarsal bones each half bearing equal weight. This has been termed the *axis of balance*.

Walking The act of walking is accomplished by shifting the center of gravity of the body forward. This is accomplished by inclining the body forward and propelling it upward by plantar flexion of the foot (Fig. 6). Gravity pulls the center of gravity forward and downward. The forward-swinging opposite leg and foot catch the falling body. The momentum of body weight carries the trunk over the leg. The process is then repeated, the center of gravity is raised and propelled forward as the triceps surae contracts, forcing the foot into plantar flexion. Thus muscle contractions raise the center of gravity and incline it forward, gravity then supplies the acceleration necessary to carry the body over the advanced leg. This mechanism conserves muscle power. The muscles in the leg and foot act only for short periods in the swing and stance phase of walking; gravity supplies part of the force necessary for locomotion.

Other muscles in the leg and foot contract briefly at various phases of the step. Following the forward propulsive force of the triceps surae and toe flexor muscles, the quadriceps muscle group contracts briefly to

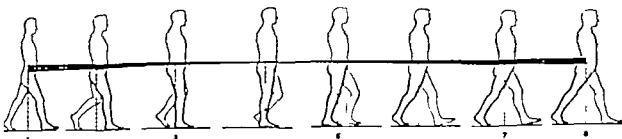


Figure 6. In walking the center of gravity falls forward and is caught by the advancing foot (1) The forward momentum carries the body over the foot, raising the center of gravity (2-3-4) During the push-off phase, plantar flexion of the foot inclines the center of gravity forward and propels it forward (5) The center of gravity then falls forward, advancing the body to be caught by the opposite foot (6-7-8) (adopted from Morton, D. J., *The Human Foot*. Columbia University Press)

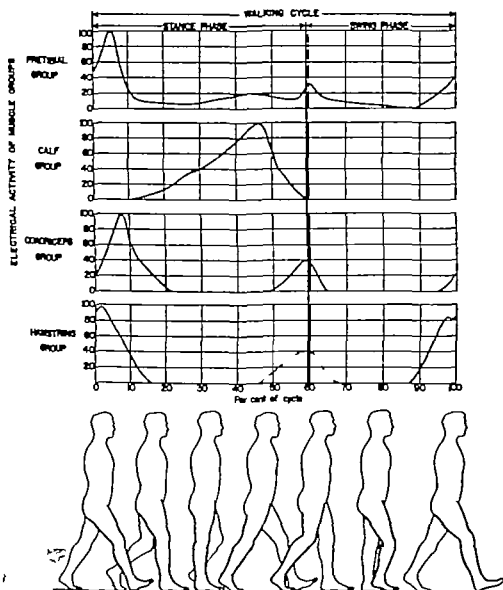


Figure 7. Electromyographic tracings of muscle activity during the stance and swing phase of walking indicate the economy of muscle power. As the heel strikes the ground, the pretibial muscles (i.e., the dorsiflexors of the foot and toes) contract to prevent the forefoot from slapping the ground. The quadriceps muscles contract briefly to straighten the knee. The hamstring muscles have already contracted to slow the forward swing of the leg at the knee acting as a brake. As the body advances over the foot, there is little muscle activity in the leg until the push-off phase when the calf muscles contract forcefully to propel the body forward. During the swing phase of walking, the pretibial muscles contract to dorsiflex the foot enough to clear the ground. Just before the heel strikes the ground, the hamstring muscles contract to slow the forward swinging leg. (Redrawn by permission from *Human Limbs and Their Substitutes* by P. E. Klopsteg and P. D. Wilson. Copyright, 1934. McGraw-Hill Book Company, Inc.)

straighten the knee (Fig. 7) As the leg swings forward, the hamstring muscles contract for a short period just before the leg is completely extended. This action of the hamstring muscles prevents the leg from snapping into extension at the knee. The dorsal flexor muscles contract as the heel strikes the ground to prevent the foot from sudden plantar flexion. These various muscle contractions supply no forward propulsion but control the forward-swinging leg and make for a smooth rhythmic gait.

Disturbances of the muscle control of the leg and foot in the swing and stance phase of walking produce dynamic changes in the foot, a source of limping and abnormal strain.

The joints of the foot and leg not only are concerned with the up-and-down oscillations of the body but take part in other actions. Because of the configuration of the hip and knee joints, the femur internally rotates at the hip and the tibia at the knee as the lower extremity swings forward and the knee is extended. As the heel strikes the ground and weight is borne on the foot, the tibia and femur externally rotate. Since the foot is fixed to the ground, the external rotation of these bones is taken up in the subtalar joint. This produces torque to the foot similar to that applied in skating, aiding in forward propulsion.

When there are disturbances in the alignment of these joints, normal function is compromised and abnormal strains are imposed on the ligaments and joint surfaces.

The weight borne by the foot during the stance phase passes through the center of the talus to the os calcis as the heel strikes the ground. As the body swings over the foot, the weight progresses into the midtarsal region. A part of this is transmitted to the navicular and cuneiform bones to the first two metatarsal bones and part through the cuboid and lateral three metatarsal bones. The weight with the foot flat to the ground is thus divided between the medial and lateral halves of the foot along the axis of balance between the second and third metatarsals (Fig. 1B). As the push-off phase of the step is reached, the weight of the body is transferred to the leverage axis of the foot between the first and second metatarsal bones. Strong plantar flexion of the great toe at this point adds further forward propulsive force.

Abnormalities of this weight bearing transmission through the foot will produce abnormal strains on bones, joints, ligaments, tendons and muscles and abnormal pressure

on the protecting covering of skin and subcutaneous tissue.

DISEASES OF THE FOOT

Diseases of the foot may be classified as congenital and acquired. Congenital deformities include skeletal defects absent or supernumerary parts defects in muscles and tendons, defective joints, circulatory and lymphatic system defects and defects of the nervous system producing abnormalities of the leg and foot.

Acquired defects include postural strains, static and dynamic deformities, diseases of the nervous system producing paralysis or spasticity of muscles circulatory diseases, tumors, infections injuries and generalized systemic disease.

CONGENITAL DISEASES OF THE FOOT

Congenital Talipes Equinovarus. Congenital defects of the foot are many and varied. Some of them have a tendency to transmission to succeeding generations.

Congenital talipes equinovarus, or clubfoot, is a serious deformity occurring once in every 700 to 1000 births (Fig. 8). The defect in varying degrees encompasses 75 per cent of the congenital anomalies of the foot. Frequently it is bilateral and is found twice as often in boys as in girls.

Clubfoot is a combination of three distinct deformities in the foot and ankle. The forefoot is adducted and supinated, the heel inverted and the foot is in equinus at the ankle. The degree of deformity varies from a mild flexible one to a severe, rigid defect. There is no bone deformity at birth, but the relationship of the joints is abnormal. The metatarsal bones are adducted at the metatarsal-tarsal joints. The first metatarsal bone may be shorter than normal. The cuboid and navicular bone shift medialward, producing widening laterally at the calcaneocuboid joint. The navicular bone lies on the medial surface of the head and neck of the talus.

The normal relationship of the talus and



Figure 8. Talipes equinovarus or clubfoot in infancy

the calcaneus is disturbed. When viewed from above, the long axis of the talus is directed forward and medially while the axis of the calcaneus is directed forward and laterally (Fig. 9). The intersection of these axes forms a V with the opening forward. The clubfoot heel is inverted and the os calcis appears to lie directly under the talus with their long axes parallel.

The soft tissues are contracted on the medial and plantar aspect of the foot, accompanying the varus deformity.

The calf muscles are short, holding the foot in equinus position, and the insertion of the Achilles tendon appears to lie more medially on the os calcis.

The etiology of clubfoot is not clear. Several theories are applied to explain the deformity. Arrest or anomalous development of the germ plasm concerned with develop-

ment of the foot in the first trimester of pregnancy may explain the defect in some instances.

During fetal development, the limb bud first is in the equinovarus position, but gradually the limb rotates to the normal position. Failure of rotation may account for clubfoot.

Biopsy material has shown degenerative changes in the muscles and there may be anomalous insertion of the tendons in the foot.

In infancy since the deformity is produced by soft tissue contractures stretching of these structures is begun as soon as possible, often within the first week of birth, and continued until complete overcorrection is obtained.

Three definite steps are followed to correct the deformity. Adduction and supination of the forefoot are gently changed into ab-

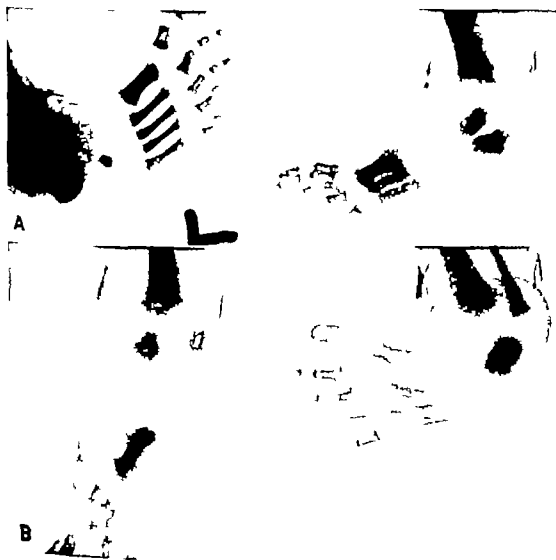
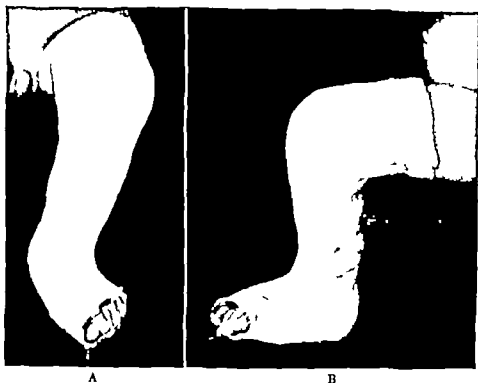


Figure 9. A, Relationship of bones in the normal foot. B, Abnormal position of bones in clubfoot. The adduction of the forefoot is marked. The talus lies directly over the os calcis in the anteroposterior view and the foot is plantar flexed at the ankle.



A

B

Figure 10 A, Corrective clubfoot cast extends above the bent knee and holds the foot in abduction and eversion. B Correction of equinus by dorsiflexion is started only after adduction of the forefoot and varus of the heel have been fully corrected.

duction and pronation. The inverted heel is everted and the equinus is corrected by dorsiflexion of the foot.

The overcorrection must be maintained until the affected muscles recover which often may require many months.

There is frequently a tendency for recurrence of the deformity and the child must be observed until growth is completed and any recurrence treated immediately.

To correct the deformity the foot is gently stretched manually by the surgeon and a well padded cast applied from the toes to above the flexed knee with the forefoot abducted (Fig. 10A and B). The cast is changed every two weeks. When full abduction of the forefoot is obtained, the heel is gradually everted. Dorsiflexion of the foot must not be attempted until this has been accomplished.

Failure to fully correct the adduction of the forefoot, the talonavicular joint and the inverted heel before dorsiflexing the foot will result in a rocker-bottom foot. The foot dorsiflexes through the midtarsal joints; the talus and calcaneus remain in equinus. This deformity may be as disabling as clubfoot. When this occurs, the entire procedure must be started over.

When clubfoot is fully corrected, the surgeon should be able to approximate the lit



Figure 11 The test for full correction of equinovarus deformity approximates the fifth toe to the anterolateral aspect of the leg.

tle toe to the lateral side of the leg (Fig. 11).

Following cast treatment, to maintain the correct position, the baby wears prewalker clubfoot shoes and a Denis Browne splint until walking is begun.

When the foot is rigid or treatment is started late, wedged casts may be necessary (Fig. 12). Occasionally soft tissue contractures on the medial side of the foot are released surgically. T capsules are

incised to allow correction of the forefoot adduction and heel inversion. Excessive stripping of the structures from the bone is avoided, as the scar tissue which forms is inelastic and may cause recurrence.

The Achilles tendon may be lengthened when there is persistent equinus in order to prevent a flat-top talus. The talus held in prolonged equinus develops a flat tibial articular surface which blocks dorsiflexion. However some weakness of the leg results from lengthening of the Achilles tendon and the procedure should be reserved for resistant clubfeet which fail to respond to stretching.

When the peroneal muscles function poorly or do not develop, even a well-corrected clubfoot will recur. When the child walks the peroneal muscles fail to contract and the foot swings into inversion. Transferring the tibialis anticus to the cuboid bone tends to balance the foot and prevent the deformity.

Older children who have not been treated or who have been allowed to develop recurrent clubfoot may have abnormally shaped bones in the foot. If wedged casts fail to correct the deformity resection of the deformed bones may be necessary. This is most conveniently done by removing appropriate wedges of bone from the subtalar talonavicular

and calcaneocuboid joints and fixing these joints in the proper position by arthrodesis (Fig. 13). The operation must not be done before the age of ten years as disturbance of growth of the foot follows.

Congenital Metatarsus Varus or Adductus. Two types of congenital metatarsus varus are found in the newborn. The first type exhibits adduction and inversion of the metatarsal bones with the heel in the valgus position. This foot deformity is rigid and difficult to correct and recurrence is the rule. Fortunately the condition is rare.

The second type is flexible: the forefoot is adducted and the heel is in neutral or slight varus position (Fig. 14A and B). This type has been increasing in frequency in recent years. Fortunately this deformity is easily corrected and recurrence is uncommon. The forefoot is adducted and inverted and the longitudinal arch is higher than normal through the action of the tibialis anticus muscle. The lateral aspect of the foot at the base of the fifth metatarsal bone is prominent. The hind foot is normal and the foot can be readily dorsiflexed.

In mild deformities stretching the forefoot in abduction and eversion is done by the mother each time the baby's diaper is changed. This may be the only treatment

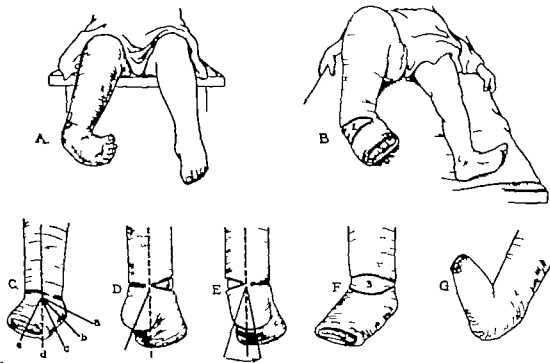


Figure 12. A, Recurrent and resistant clubfeet may require wedged casts to correct the deformity. B, A wedge of plaster is removed laterally to allow gradual stretching of the adducted forefoot. C, The wedge closed and the forefoot abducted. D, The heel viewed posteriorly is forced into eversion by removing a wedge of plaster laterally just above the subtalar joint. E, The wedge closed and the heel everted. F, The equinus deformity is corrected by removing a plaster wedge from the cast at the level of the ankle joint. G, The fully corrected position of the foot in the plaster cast.

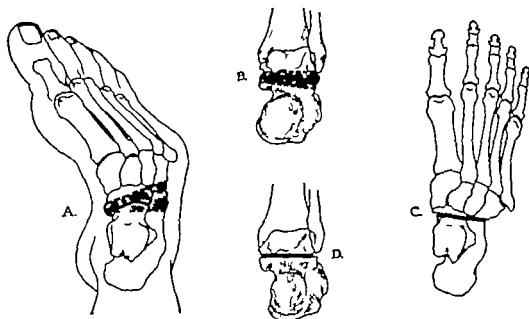


Figure 13. A, Bony deformity of clubfoot demands resection of bone to place the foot in the normal position. Arthrodesis of the talonavicular and calcaneocuboid joints is done to prevent recurrence of the adduction deformity (redrawn from Speed, J. S., and Smith, H.: *Campbell's Operative Orthopaedics*, 2nd ed. C. V. Mosby Company).

B A wedge of bone is removed from the subtalar joint to place the heel in slight eversion. C and D, The corrected foot.



Figure 14. A, Metatarsus varus deformity. B The deformity is increased and perpetuated if the infant sleeps with the legs internally rotated.

necessary. Prewalker clubfoot shoes attached to the Denis Browne splint in external rotation may correct more severe deformity. Occasionally casts are used as in the first part of the correction for clubfoot.

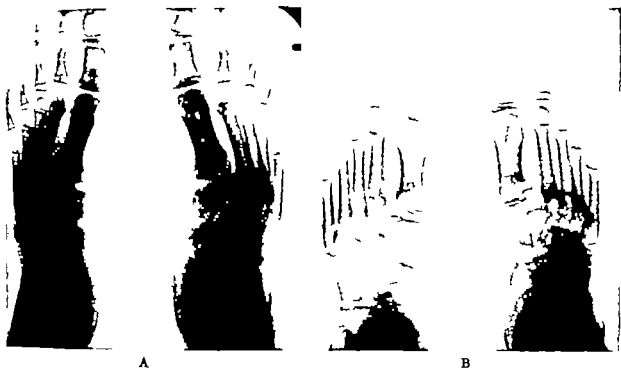
Untreated, the deformity may persist and increase in severity. Older children who fail to respond to use of a wedged cast require osteotomy of the base of the metatarsal bones (Fig. 15A and B). Following union of the bones, outflare shoes are worn for several months.

Congenital Calcaneovalgus. The congenital calcaneovalgus foot is the direct opposite

of talipes equinovarus. The etiology is thought to be abnormal intrauterine position of the foot.

The bones of the foot are normal. The foot is in dorsiflexion, the heel points downward in valgus position and the forefoot is abducted and dorsiflexed. The fifth toe may be easily approximated to the anterolateral aspect of the leg. Usually the foot is quite flexible and may be partially plantar flexed and inverted without difficulty.

The deformity is easily corrected by repeated gentle stretching and there is little tendency toward recurrence. The mother is



A

B

Figure 15. A, X-rays of bilateral metatarsus varus. B, Healing after osteotomy near the base of the metatarsal bones correcting the varus deformities.

instructed to stretch the foot in plantar flexion, inversion and adduction each time the baby's diaper is changed. Denis Browne splints may be used if the deformity is resistant to stretching. The feet are internally rotated and inverted by the splint. Rarely is cast correction indicated.

When the child begins to walk, the foot may tend to assume a valgus position on weight bearing. The child should be fitted with a $\frac{1}{4}$ inch inner heel wedge on the shoe during this period.

Syndactyly of Toes. Syndactyly may occur in two or more toes. The deformity tends to be a familial one. The toes may be webbed completely or partially from the base to the tip. The soft tissues only may be affected or the deformity may have a bony bridge between the phalanges. Little if any functional disability results from the defect. Separation of the toes by surgery is seldom necessary as the foot functions well and shoe-fitting problems are unusual.

If, for cosmetic reasons the toes are separated surgically x-rays must first be made to determine the degree of bone involvement. When the bones are joined near the epiphyseal plates of the phalanges, the operation should be deferred until spontaneous closure of the epiphyses has taken place. If the operation is done too early unilateral closure of a portion of the epiphyseal plate will cause angulation and deformity of the toes with growth.

Polydactyly Polydactylism in the foot is characterized by the presence of supernumerary toes. The deformity is usually familial. The extra digit may be completely formed, normal in appearance and function, or it may be misshapen, rudimentary and functionless. Reduplication of a part of the toe, such as the distal phalanx, is a variation of the deformity. Shoe fitting problems are frequently encountered, demanding amputation of the extra toe or plastic reconstruction of reduplicated phalanges. Amputation of a complete toe can be done at any age; however resection of portions of duplicate phalanges must not interfere with the epi-



Figure 16. Reduplication of the distal phalanx of the great toe. The epiphysis is involved in the defect.



Figure 17 Agnasia, syndactyly and deformity of the toes. Congenital constriction band is present in the right forefoot.



Figure 18 Os trigonum. Thin cortex about the entire bone differentiates it from fracture of the posterior talus.

physal plate. X-rays should be taken to determine the degree of bone deformity and involvement of the epiphysis (Fig. 16).

Agnasia. Agnasia of part or all of the foot may result from faulty limb bud development and be the source of profound disability. The forefoot is most frequently the site of deformity. Parts of toes and metatarsals may be absent. Abnormal insertion of tendons and absence of bones may produce considerable distortion of the foot (Fig. 17). Occasionally tendon transference, plastic operations or stabilization of the joints of the foot is necessary to correct deformity.

Macroductyly. Occasionally a foot is encountered which exhibits one or more giant toes. The overgrowth of the part may be due to vascular or neurotropic lesions or to some defect in the growth capacity of the toe. Amputation of the toe or reduction of the size of the toe by plastic surgical procedures is usually necessary to permit the wearing of matched shoes.

Accessory Bones of the Foot. Accessory bones of the foot occur in many locations but most do not produce symptoms and are found incidental to x ray examination of the foot. The recognition of these bones as accessory bones and not fractures is of medicolegal import. The accessory bone has a complete cortex surrounding the bone while the recent fracture has an absence of cortex at the fracture line.

The most common accessory bones in the foot are the os trigonum, found near the posterior aspect of the talus, and the os vesallianum, at the base of the fifth metatarsal bone

(Fig. 18). These bones do not produce symptoms or weaken the foot but are often confused with fractures.

The accessory navicular bone located at the medial aspect of the parent bone produces a prominent mass. A bursa and painful callus may form over the medial aspect of the foot at this site from shoe pressure. The longitudinal arch of the foot may be weak and flat because the tibialis posterior tendon is abnormally inserted.

Symptoms are usually pain and swelling of the bursa over the bony prominence and fatigue and aching in the midtarsal region because of faulty support for the longitudinal arch.

When the symptoms cannot be relieved by supports to the arch of the foot, surgical correction of the deformity is necessary. The accessory navicular bone is removed and the tibialis posterior tendon is inserted under the navicular bone.

Pes Planus or Flatfoot. Pes planus may be acquired or occur as a result of a congenital deformity (Fig. 19A-C). Flatfoot associated with congenital calcaneovalgus deformity and the accessory navicular bone has been described. Other congenital defects may be etiologic factors in the development of flatfoot. Hypermobile flatfoot with a short tendo achilles and coalition of the tarsal bones producing peroneal spastic flatfoot are examples. Pes planus may be acquired as a result of excessive strain to the osseous with weak ligaments, neurologic lesions affecting the muscles and ligaments,

trauma or infection producing bone, joint or muscle abnormalities, and disease of muscle.

Not all flatfeet are painful. Like the shape of noses, the height of the arches of the foot varies and may follow familial patterns. The small child beginning to walk may appear to have no arch at all. A fat pad occupies the plantar area at this age and may obscure the arch which is readily revealed when weight bearing x rays are taken.

Pain develops in flatfeet when the ligaments are subjected to abnormal strain. Con-

tinued use loads to synovitis of the joints of the foot and later traumatic arthritis. In the growing child, the tarsal bones develop abnormal shapes. The patient suffering from painful flatfoot rotates the foot and leg externally while walking. In this position the anterior lever of the foot is shortened the push-off is from the medial side of the first metatarsal head and great toe. The stride is shortened, producing a shuffling gait.

The calcaneus is in valgus position at the subtalar joint. The talus points more medialward and the head of the bone tips down-



Figure 19 A, Pes planus. Flat medial arch with abduction of the forefoot. B Complete flattening of the longitudinal arch. C, Valgus position of the heels



Figure 20 Abduction of the forefoot with separation of the anterior relationship of the talus and os calcis—adult flatfoot.

ward. The longitudinal arch is flattened by angulation plantarward at the talonavicular or cuneiform-navicular joints, or at both joints. These bones rotate medialward and down, carrying the base of the metatarsal toward the plantar surface of the foot. The metatarsal bones abduct and the joints on the medial aspect of the foot open and compress on the lateral side (Fig. 20). The calcaneonavicular and long plantar ligaments are stretched and relaxed.

Short Triceps Surae Muscles. When the tendo achillis and triceps surae muscles are short, dorsiflexion of the foot is limited and increased strain is placed on the anterior lever arm of the foot. If the supporting ligaments stretch with weight bearing, the longitudinal arch flattens. The talus and calcaneus remain in equinus and the forefoot dorsiflexes through the midtarsal joints. The condition may occur as congenital shortening of the muscle group or secondary to the contractions of spastic paralysis or other neuromuscular afflictions.

Treatment is directed toward lengthening the tendo achillis and calf muscles and supporting the arch in the normal position until the muscles and ligaments recover. The triceps surae muscles in the child are stretched with wedged casts. The foot must be inverted and the forefoot adducted during this procedure to prevent further stretching of the medial plantar ligaments and increasing the deformity.

When the calf muscles cannot be stretched, the Achilles tendon or the musculotendinous junction can be lengthened surgically. The foot and leg are immobilized in a plaster cast with the foot inverted and dorsiflexed for three to six weeks. A supporting shoe with an extended rigid counter, Thomas heel and medial heel wedge is used postoperatively to support the repositioned arch.

Hypermobile Flatfoot. Hypermobile flat foot with short tendo achillis occurs secondary to a congenital defect in the relationship of the talus to the os calcis. Characteristically the foot is flat only on weight bearing, assuming a normal appearing arch when weight is removed. A history is obtained usually of asymptomatic flatfeet during childhood followed by aching, easily fatigued feet in adolescence or early adult life. Occasionally pain develops in childhood. Symptoms vary in intensity.

The primary etiologic factor is inadequate support for the head and neck of the talus by the os calcis. The triceps surae muscles are short, but this finding is secondary to the

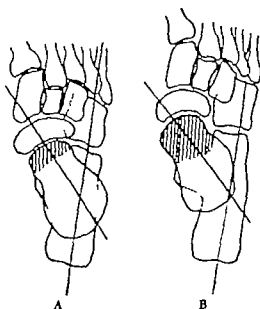


Figure 21 A, The normal relationship of the bones is shown for comparison. B Relationship of the talus to the os calcis in hypermobile flatfoot. The talus is displaced medially and the long axis of the bones diverges anteriorly. The head and neck of the talus project farther forward and the sustentaculum tali is narrow, offering less support for the talus. (Redrawn by permission from *Hypermobile Flat Foot with Short Tendo Achilles*, by R. I. Harris and T. Beath, *J. Bone & Joint Surg.*, vol. 30-A, pp. 116-140 Jan., 1948.)

bone anomaly. The head and neck of the talus are displaced medially and the bone forms a more divergent angle with the long axis of the calcaneus (Fig. 21). The talus appears to project farther forward on the os calcis and the sustentaculum tali is underdeveloped, slanting and more posterior on the calcaneus than normal. This lack of bony support under the head of the talus allows it to plantar flex. Coupled with this defect, the midtarsal joints are more mobile than normal, allowing the forefoot to abduct and dorsiflex to a greater degree. The calcaneus assumes a valgus position on weight bearing and, since the calf muscles are short, the forefoot dorsiflexes and abducts to produce the flat foot.

Treatment is directed toward support for the talus and relieving the pull of the short triceps surae. Since the degree of anatomic abnormality and the severity of symptoms vary, so does the treatment. In childhood it may be necessary only to support the foot with adequate shoes and arch supports. A heel lift may be used to lessen the pull of the calf muscles. This same treatment may continue into adult life if the condition is mild and the symptoms easily controlled.

When the symptom Anatomical deficit

are severe, the subtalar and midtarsal bones must be stabilized by arthrodesis in the correct weight-bearing position. This may be accomplished by triple arthrodesis after growth of the foot is complete (Figs 22 and 23)

Peroneal Spastic Flatfoot. Peroneal spastic flatfoot is a painful rigid foot associated with spasm and contracture of the peroneal muscles. It is due to congenital coalition of the tarsal bones. Complete or partial bony

bridges form between one or more tarsal bones. This coalition of tarsal bones limits motion between the involved bones. Since the subtalar talonavicular and calcaneocuboid joints act as a unit, limitation of motion in any joint throws additional strain on the remaining mobile ones. This strain eventually produces irritation in the joints and ligaments. The irritation in turn, produces reflex spasm of the peroneal muscles which forces the foot into eversion. As the irritation

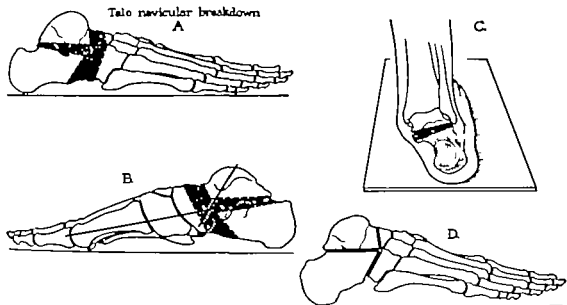


Figure 22 A and B Triple arthrodesis of the subtalar talonavicular and calcaneocuboid joint is performed to correct and stabilize the hypermobile flatfoot when the major angulation is at the talonavicular joint. The procedure may be used in any type of flatfoot. C, A wedge of bone is removed from the subtalar joint with the base medial to correct the valgus of the heel.

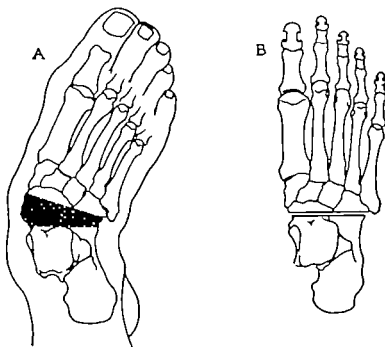


Figure 23 A and B The forefoot abduction is corrected by removing a wedge of bone with the base medial, stabilizing the talonavicular and calcaneocuboid joints.

O. L. Miller operation for flat foot

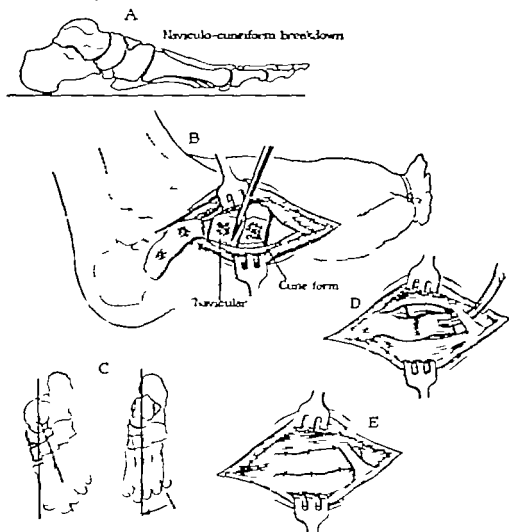


Figure 24 A, Painful flatfoot with the major plantar angulation at the naviculocuneiform joint is stabilized by arthrodesis of the naviculocuneiform and cuneiform-metatarsal joints. B A flap of joint capsule and bone is raised from the navicular and cuneiform joints. The cartilage and subchondral bone are removed. C, The abduction and eversion of the forefoot are corrected. D and E, The flap of ligament and bone is sutured under the tibialis anterior tendon and reefed to hold the new position of the arch. While fusion takes place the foot is immobilized in inversion with the arch molded by a plaster cast. (Redrawn by permission from A Plastic Flat Foot Operation by O. L. Miller M. D. J Bone & Joint Surg., vol. 9 p. 84 January 1927)

continues with weight bearing, pain develops in the midtarsal and subtalar joint areas, often beneath the lateral malleolus.

The patient cannot voluntarily invert the foot and attempts by the surgeon to do this produce immediate pain and the peroneal muscles involuntarily contract. A similar clinical picture may occur when irritation and limitation of motion are present in the damaged subtalar joint following fracture of the articular surface of the os calcis.

The most frequent coalition is between the anterior os calcis and the navicular bone. The bones fuse by either a complete or partial bridge of bone at the medial anterior portion of the os calcis and the lateral inferior portion of the navicular bone. This bridge of

bone limits inversion and eversion in the subtalar joint. Routine anteroposterior and lateral x-ray views fail to reveal the abnormality. Oblique x-ray views are necessary to show the coalition.

Other bony or cartilaginous bridges form between the os calcis and talus. One may occur posteriorly through the os trigonum, the other in the region of the sustentaculum tali. The latter can only be visualized by special x-ray technique. To obtain the x-ray the patient is placed on the x-ray table with the knees flexed. The x-ray beam is directed from the posterior downward and forward between 35 and 45 degrees to correspond to the angulation of the articular surface of the sustentaculum.

The calcaneus may coalesce with the cuboid bone through a bridge of bone through the os peroneum. Other fusions of bone take place between the cuboid and navicular bones and the talus and navicular bone. Rarely massive fusion of several tarsal bones is found.

Injury may precipitate the original pain in the ligaments followed by the syndrome of peroneal spastic flatfoot.

Treatment is directed toward relief of irritation and muscle spasm. Rest, elevation and heat are prescribed followed by gentle manipulation of the foot to the inverted position. The foot and leg are immobilized in a plaster cast with the foot inverted for six weeks. Weight bearing in a walking cast is resumed at the end of three weeks. If the symptoms subside, the patient is fitted with an orthopedic shoe to support the longitudinal arch and hold the heel slightly inverted.

Recurrence of the symptoms and deformity is the rule and surgical correction is often necessary. Arthrodesis of the tarsal joints eliminates motion and the irritation which produces the peroneal muscle spasm. Following this procedure inversion and eversion of the foot are lost, but the foot is stable in the proper weight-bearing position and is painless.

Triple arthrodesis of the subtalar, calcaneocuboid and talonavicular joints is useful in correcting flatfoot deformities which do not respond to conservative supportive measures (Figs. 22 and 23). The operation is indicated in peroneal spastic flatfoot, hypermobile flatfoot, arthritic flatfeet and in flatfeet when the main deformity occurs at the talonavicular joint.

Some flatfeet exhibit the primary plantar angulation at the navicular-cuneiform joint. Children or young adults with symptomatic flatfeet that do not respond to conservative measures may require correction and fusion of this joint with the first metatarsal-cuneiform joint (Fig. 24). Following the operation, the foot is immobilized in inversion and plantar flexion to raise the arch until arthrodesis occurs.

Congenital Vertical Talus. The deformity of congenital vertical talus produces a rigid rocker-bottom foot. The longitudinal arch is nonexistent or reversed. Although the etiology is considered congenital, the high incidence of this deformity associated with arthrogryposis suggests a definite relationship to this disease.

The forefoot is dorsiflexed, the heel is in equinus and deep creases may be present

over the anterior ankle. Eversion of the heel and abduction of the forefoot are not present at birth but develop later. Rigidity of the foot is a characteristic finding. The posterior os calcis does not touch the ground when standing. The first metatarsal is dorsiflexed and a flexion deformity may be present at the metatarsal phalangeal joint of the great toe. The calcaneus is narrow and beak shaped anteriorly and the talus resembles an hour glass. The navicular bone dislocates dorsally and laterally and articulates with the talus at the neck of the bone.

The disability is profound. Shoes are difficult to fit and pain develops in the foot. The rigidity of the foot defeats conservative corrective measures. Triple arthrodesis at an appropriate age may place the foot in a better weight-bearing position, but the result is never perfect.

Talipes Cavus. Pes cavus, or clawfoot, is the opposite deformity of flatfoot. The longitudinal arch is higher than normal. Usually the hind foot is near normal, the os calcis may be slightly inverted. The high arch is due to plantar flexion of the forefoot through the tarsal and metatarsal joints (Fig. 25). Mild deformities may not affect the toes and are asymptomatic. With more severe deformity the toes become flexed at the interphalangeal joints and hyperextended or dorsally dislocated at the metatarsal phalangeal joints. The plantar fascia becomes shortened and the metatarsal heads are prominent in the ball of the foot. Calluses develop over the metatarsal heads and corns appear over the proximal interphalangeal joints of the toes. The foot may be in moderate equinus



Figure 25 The arch is elevated in the cavus foot. The dorsum of the foot is prominent. The deformity is primarily due to plantar flexion of the forefoot with the heel in the normal position. In this case the toes are contracted into the clawfoot deformity.



Figures 26. A, The high arch of cavus deformity is lowered by resecting a wedge of bone through the talonavicular joints and the body of the cuboid bone. The triple joints of the foot—the talonavicular, calcaneocuboid and subtalar joints—are preserved, retaining normal motion. B, When the wedge defect is closed, the forefoot is displaced plantarward at the osteotomy site and dorsiflexed to lower the longitudinal arch. Dorsal wedge section is useful when the heel is in neutral position.

position associated with contracted calf muscles.

The deformity may be idiopathic or secondary to diseases of the nervous system or muscles. It frequently follows poliomyelitis, myelodysplasia, spina bifida and muscular dystrophy. The intrinsic muscles of the foot are insufficient and may be contracted. Failure of these muscles to maintain extension of the interphalangeal joints of the toes creates imbalance. The unopposed long extensor muscles produce hyperextension at the metatarsal-phalangeal joints and the long flexor muscles promote flexion of the interphalangeal joints.

Conservative treatment in the mild condition consists in elevation of the depressed heads of the metatarsal bones and stretching of the tight plantar structures. This may be done with a transverse metatarsal bar placed on the sole of the shoe just proximal to the metatarsal heads. A similar method may be used in which a metatarsal arch support is placed in the shoe. This raises the metatarsal heads and relieves pressure on the plantar calluses by transferring the weight more proximally in the foot. The elevation of the metatarsal bones tends to straighten the contracted toes.

When more severe deformity is found, operative correction may be necessary to relieve the symptoms. Since the plantar aponeurosis is short, it may be lengthened by section of the attachment to the os calcis. The foot is stretched and immobilized in the corrected position in a plaster cast for three weeks. When the toes are in the claw position, the metatarsal heads are depressed and the toes do not function. Arthrodesis of the interphalangeal joints so they are straight permits the long flexor muscles of the toes to act at the metatarsal-phalangeal joints,

holding the toes in proper weight bearing position.

If the deformity is progressive or the foot is rigid more extensive operative procedures are demanded. In addition to arthrodesis of the toe joints the extensor tendons of the toes are resected with the dorsal joint capsules. The dorsally dislocated toes can then be reduced. The extensor digitorum longus tendons are transferred and inserted into the neck of the metatarsal bones. This aids in elevating the distal metatarsal bones, reducing the forefoot drop and prominent metatarsal heads.

Wedge resection of the navicular-cuneiform joints and the cuboid bone, coupled with foregoing procedures, may be necessary to correct severe deformity. The base of the wedge of bone that is removed is dorsal (Fig. 26). When the osteotomy site is closed and the base of the metatarsal bones is displaced plantarward, the longitudinal arch is lowered. This is feasible only when the relationship in the hind foot is normal. If the heel is inverted and the forefoot adducted, triple arthrodesis with removal of appropriate wedges of bone from the subtalar, talonavicular and calcaneocuboid joints is necessary to correct the deformity. These operations on bone should be deferred until the foot is well developed. Before the age of ten, the ossification of the bones is incomplete and growth of the foot is retarded or arrested when the operation is done. The operative procedures on the soft tissues can be done before this time and may help prevent more serious deformity.

AFFLICTIONS OF THE FOREFOOT AND TOES

Hallux Valgus and Bunion. One of the most common and distressing deformities of the forefoot and toes is the bunion and bun-

ion (Fig. 27) The great toe points laterally producing a prominence on the medial aspect of the head of the first metatarsal bone. Shoes create pressure on the bony prominence and a painful bursa develops. When the deformity is pronounced, the great toe overlaps or underlaps the second toe forcing this toe dorsally or plantarward. The second toe may dislocate (Fig. 28A and B)

The cause is usually an abnormal varus angulation of the first metatarsal bone. Occasionally the condition occurs with metatarsus latus or splayfoot which produces a similar angulation of the first metatarsal bone. The space between the distal end of the first



Figure 27. Hallux valgus and bunion. The great toe is deviated laterally the first metatarsal bone is angulated medially. The prominent medial aspect of the head of the metatarsal bone is the site of a painful bursa.

and second metatarsal bones and toes is exaggerated. The adductor of the great toe deviates the toe laterally and the extensor hallucis longus tendon subluxates laterally. The flexor hallucis brevis with the lateral sesamoid bone and the flexor hallucis longus become similarly displaced. The abductor hallucis subluxates plantarward and the corrective influence of the muscle is lost. The tendons in the laterally displaced position increase the angulation of the great toe. The condition is usually slowly progressive.

When the deformity is mild and inflammation of the bursa periodic, a wide shoe made of soft kidskin is worn to minimize pressure on the bursa. The longitudinal arch of the foot and first metatarsal bone is supported with pads or a corrective shoe. To prevent increasing splayfoot, the metatarsal arch is elevated with a pad.

Since the deformity is progressive, the metatarsal head becomes more prominent. The inflamed bursa stimulates the bone and an exostosis forms medially beneath the bursa and joint capsule. Osteoarthritis is prone to develop in the metatarsal-phalangeal joint. The push-off action of the great toe is lost as the deformity progresses.

Surgical treatment is indicated when conservative measures fail. Numerous operations have been devised to relieve the distress of bunions. The objective is to correct the valgus position of the great toe, remove the prominence of the head of the metatarsal bone and correct the deforming pull of the muscles.

The metatarsus primus varus or metatarsus latus can be corrected in young people to prevent recurrence. The presence of osteoarthritis in the joint demands remodeling of the joint surfaces.

Three general types of operative procedures are available to the surgeon. The choice

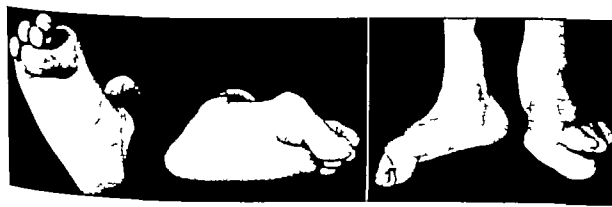
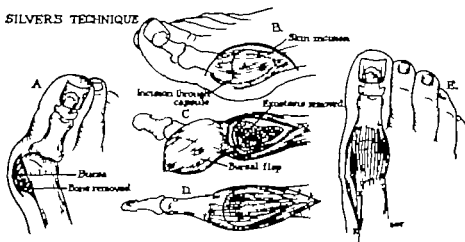
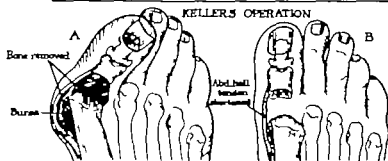


Figure 28. A, Severe hallux valgus with plantar flexion forces the small toes dorsally. B, The second toe is dislocated dorsally at the metatarsal-phalangeal joint.

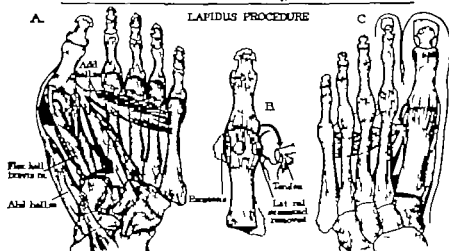
SILVER'S TECHNIQUE



KELLER'S OPERATION



LAPIDUS PROCEDURE



Figures 29 Upper: A, Mild hallux valgus may be corrected by Silver's technique. B, The joint capsule is reflected, exposing the exostosis of the head of the metatarsal bone. C, The excess bone is removed. D and E, The toe is straightened by shortening the joint capsule when it is sutured. Occasionally the lateral joint capsule is incised to relax the joint and allow correction (redrawn with permission from *The Operative Treatment of Hallux Valgus* by D. Silver, J. Bone & Joint Surg., vol. 5, 1923).

Center: The Keller operation is used when the deformity is pronounced or the metatarsal-phalangeal joint is arthritic. A, The exostosis is removed from the metatarsal head and the proximal third of the phalanx of the great toe is resected. B, The toe corrects easily and a painless pseudarthrosis forms. The toe is shortened but functions well. (Redrawn from Keller, W. L., *New York M. J.*, vol. 95.)

Lower: The Lapidus procedure corrects the metatarsus varus and hallux valgus. A, The exostosis is removed from the metatarsal head. The cuneiform-metatarsal joint is resected to correct the metatarsus varus. B, The adductor tendon is resected from the proximal phalanx and sutured to the first metatarsal. C, The deformity is corrected and the base of the first metatarsal bone is stabilized to the cuneiform bone and the second metatarsal bone to prevent recurrence. (Redrawn from Lapidus, P. W., *Operative Correction of the Metatarsus Varus Primus in Hallux Valgus*, *Surg. Gynec. & Obst.*, vol. 58.)

of operation depends upon the patient's age, circulation, degree of deformity and the presence or absence of arthritis in the joint. Mild deformities require only removal of the exostosis and the prominent bone (Fig. 29). Correction of the toe is obtained by shortening the medial joint capsule and, in some cases, sectioning the lateral joint capsule and adductor tendon. When the deformity is greater the adductor tendon may be reinserted into the metatarsal bone to remove the deforming influence and to help correct the metatarsus primus varus. The abductor tendon may be repositioned over the medial aspect of the joint.

If osteoarthritis is found, arthroplasty or remodeling of the joint must be done in order to prevent pain and stiffness (Fig. 29). This is most simply done by excision of the proximal third of the proximal phalanx of the great toe. The malposition of the toe is easily corrected as relaxation of soft tissues follows removal of the segment of bone. Pseudoarthrosis develops at the metatarsophalangeal joint which is pain free and mobile.

Young adults or older patients who have excessive metatarsus varus may require more extensive surgery to correct the deformity and to prevent its recurrence (Fig. 29). Adequate circulation of the foot is a prerequisite. The objective is to correct the metatarsus varus as well as the hallux valgus. Osteotomy or wedge resection, at or near the base of the first metatarsal bone, is performed and the bone realigned. Arthrodesis of the first metatarsal base to the second as well as the metatarsal-cuneiform joint may be necessary to maintain the position.

Metatarsus latus or splayfoot, may be corrected by a similar procedure combined with the creation of a plantar sling of tendon from the fifth to the first metatarsal heads pulling the metatarsal bones together.

Hallux Rugosus or Hallux Nonextensus. Hallux rigidus, as the name implies, is characterized by painful limitation of extension at the great toe joint.

The etiology is not always clear, however an exostosis develops over the dorsal articular margin of the metatarsal phalangeal joint. Extension of the toe is blocked to varying degrees and osteoarthritis frequently accompanies this change. A bursa may develop over the dorsum of the joint. The foot is turned outward when walking to avoid dorsiflexion of the toe. A callus may form on the plantar aspect of the interphalangeal joint secondary to hyperextension of this joint.

Conservative measures are limited to the

use of a stiff-soled shoe to prevent dorsal flexion force on the toe and removal of pressure on the bursa when it is present.

The dorsal exostosis may be removed surgically and the proximal third of the phalanx of the toe resected. Following surgery the patient must be taught to walk with the foot pointing forward to lessen arch strain.

Digitus Quinti Varus or Bunionette. Varus position of the fifth toe with bunion formation is less common than hallux valgus. The bursa forms over an exostosis or prominence of the head of the fifth metatarsal bone on the lateral aspect of the foot. The little toe may exhibit little or marked varus deformity, angulating toward the medial side of the foot. It may overlap the fourth toe or lie beneath it.

Conservative treatment is designed, as for hallux valgus, to lessen pressure on the bursa and to straighten the fifth toe. Stretching the shoe over the bursa and placing a felt pad between the fourth and fifth toes often is all that is necessary to relieve the symptoms.

Resection of the exostosis combined with shortening or reefing the lateral capsule of the metatarsal phalangeal joint straightens the toe and removes the irritating prominence. When the fifth toe is in extreme varus position over or underlapping the fourth toe, osteotomy of the base of the first phalanx will correct the position of the toe.

When the fifth toe is held in dorsiflexion and varus by a contracted extensor digiti quinti longus tendon, the tendon is sectioned at the ankle. The tendon is withdrawn through a second incision at the base of the toe. A tunnel is created by blunt dissection medially and plantarward about the base of the phalanx and the tendon passed through the tunnel and terminated laterally. It is sutured into the abductor tendon and muscle of the fifth toe, converting the tendon to a flexor abductor pulling the toe down and out.

Hammer Toe. Hammer toes are similar in appearance to claw toes but lack the extreme hyperextension of the metatarsal phalangeal joints. The proximal interphalangeal joint is flexed and the distal joint flexed or extended.

Symptoms are produced when the toe rubs in the shoe. A callus is formed over the plantar pad of the distal phalanx and a corn is found over the dorsum of the flexed interphalangeal joint. Shoes with a boxlike toe may give the toes room and relieve the pressure symptoms. Padding beneath the metatarsals may tend to straighten the toes when they are flexible.

Straightening the toes by resection or ar

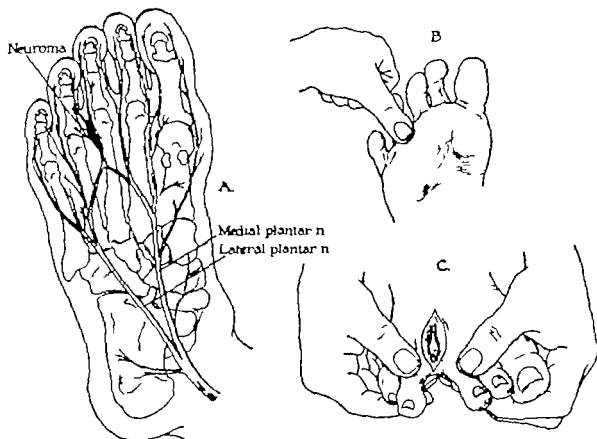


Figure 30 A, The medial and lateral plantar nerves join between the third and fourth metatarsal bones. The common digital nerve is fixed at this point. The most frequent site of interdigital neuroma is between the third and fourth toes. B, The typical symptoms of Morton's toe are produced by pressure between the heads of the metatarsal bones. C, A web-splitting incision and blunt dissection expose the tumor mass between the metatarsal bones. The entire mass is excised.

throdesis of the proximal interphalangeal joint produces a comfortable foot, eliminating the points of pressure and friction.

Plantar Callosities and Prominent Metatarsal Heads. The heads of the metatarsal bones may be depressed into the sole of the foot by a variety of abnormalities. The involved bone then carries more weight. Friction and pressure lead to hyperkeratotic skin changes. Pain on weight bearing is the usual complaint.

Treatment is directed toward correction of associated foot deformity and redistribution of weight through the metatarsal bones. Transverse metatarsal bars and arch supports elevate the metatarsal bone and relieve the abnormal pressure. With relief of excessive pressure, the callus disappears.

Plantar warts, thought to be virus infections, may be found in the callus. These lesions may disappear when excessive pressure is relieved. Some plantar warts, however, are persistent or recurrent. Salicylic acid adhesive plaster is applied to the area for forty-eight hours. The callus and wart may be carefully trimmed after the skin softens.

X ray therapy supervised by a roentgenologist is warranted when plantar warts do not respond to the above treatment. Care must be exercised to prevent excessive radiation.

Occasionally pressure of a prominent metatarsal head cannot be relieved with arch supports. After the other associated defects in the foot are corrected, one or two metatarsal heads, with the exception of the first, may be resected without weakening the foot. Removal of the head of the first metatarsal bone produces a serious defect in the weight bearing mechanism of the foot.

Interdigital Neuroma. Neuroma of the common digital branch of the plantar nerves produces characteristic symptoms which have been called Morton's toe (Fig. 30A). Lancing pain in the region of the third and fourth toes and metatarsal bones occurs periodically while walking. Pain may radiate to the anterior surface of the leg, and the toes may be drawn into the claw position. Relief is obtained by removing the shoe and massaging the foot.

Examination may reveal normal appear

ing foot or the common forefoot deformities may accompany the condition (Fig. 30B). Pressure exerted by the examining finger between the base of the third and fourth toes will often reproduce the typical attack of pain. Occasionally a firm tender mass can be palpated between the metatarsal bones. The sensation of pain and temperature is usually diminished on the adjacent surfaces of the toes.

The medial and lateral branches of the plantar nerve form an arch in the forefoot joining at the fourth common digital nerve. Traction or pressure on the nerve at this fixed point is thought to be the etiologic factor producing the neuroma. Inflammatory reaction about the nerve stimulates the formation of fibrous tissue in the nerve, forming the tumor mass. Occasionally a small bursa filled with clear yellow fluid surrounds the neuroma.

During the early stages of the syndrome, symptoms may be relieved by a transverse metatarsal bar on the sole of the shoe to minimize pressure on the third and fourth metatarsal bones. If the symptoms recur or persist, the area about the tumor is injected with local anesthetic and 25 mg. of corn pound F.

Symptoms which have been present sev-



Figure 31. The ingrowing toenail is abnormally curved, predisposing to encroachment on the soft tissues at the margin of the nail.

eral months and do not improve with conservative measures usually require excision of the tumor. This is done through a web-splitting incision between the toes (Fig. 30C). Identification of the tumor mass just proximal to the bifurcation of the proper digital nerves is easily accomplished by blunt dissection. The common digital nerve is sectioned proximal to the mass and the tumor removed.

The same lesion is found less frequently in the other toes but may occur in any of the digital nerves.

Ingrowing Toenails. Ingrowing toenails (Fig. 31) develop secondary to injury, infection or pressure. The edge of the nail cuts into the adjacent soft tissue, injuring the skin. Bacteria invade the area and an abscess or chronic granuloma results. Periodic recurrent episodes of pain and swelling occur as the nail grows. Injury to the nail bed or a fungus infection may deform the nail or pressure of the shoe on the soft tissues about the nail margin can produce the condition.

During the acute infection, the principles of rest, heat and the establishment of adequate drainage are applied. The offending portion of the nail is removed and the nail margin packed away from the skin with cotton. Packing with cotton is continued to form a gutter through which the nail may grow without pressure on the skin. Frequent changes of cotton, application of fungicides and removal of keratotic skin are carried out until the nail has grown past the distal skin margin of the toe. The toenail is then trimmed straight across.

Recurrent episodes of infection are treated by resection of the margin of the nail and destroying the nail bed from which it grows (Fig. 32).

Clavus or Corns of the Toes. Pressure and friction by shoes on the toes produces localized keratotic lesions in the skin called clavus or corns. The lesions occur typically at the flexed interphalangeal joint with claw-toe

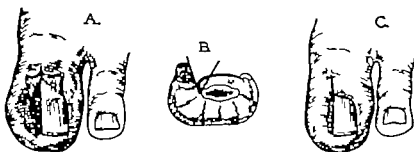


Figure 32. Resection of ingrowing toenail. A, Eponychium incised and reflected to expose the base of the nail. B, Wedge-shaped resection of the nail and toenail bed. C, The eponychium is closed and the remainder of the wound packed with petrolatum gauze.

deformities. The lateral dorsal surface of the fifth toe is the most frequent site. When pressure and friction are excessive, a small painful bursa develops beneath the layer of keratotic skin. Eliminating pressure with a soft leather low heel shoe with adequate room for the toes may relieve the symptoms. The toes can be straightened if they are clawed. The corn over the lateral aspect of the fifth toe is due to prominence of the lateral condyle of the proximal phalanx. Excision of the bony prominence along with the bursa is usually successful.

HIND FOOT PROBLEMS

Afflictions of the hind foot involve primarily the heel and adjacent structures. Bursitis of the os calcis on the plantar aspect and Achilles tendon posteriorly is a frequent source of pain in the heel. Spurs about the origin of the plantar aponeurosis were at one time thought to be a source of pain. Removal of these spurs seldom gave relief and the procedure is now done infrequently.

Symptoms of bursitis in the plantar surface of the os calcis are localized to that area. Tenderness to pressure and weight bearing is confined to the condyles of the os calcis over which the bursa forms. Plantar fasciitis producing similar symptoms is more diffuse and follows the course of the plantar aponeurosis into the foot.

Bursitis about the attachment of the Achilles tendon may be deep or superficial. The deep bursa is located between the upper margin of the os calcis and the tendon. The superficial one lies between the subcutaneous tissue and the insertion of the Achilles tendon. Irritation of these bursae results from pressure of the shoe coupled with a prominent posterior border of the os calcis. Hyperkeratotic skin forms over the protruding bone or bursae. Cracks or blisters in this skin lead

to infection. Reduction of pressure at the inflamed bursa is necessary to bring about relief of symptoms. On the plantar surface, sponge rubber pads are placed in the heel of the shoe to cushion the bursa. Posteriorly felt pads are placed on each side of the heel to relieve pressure on the Achilles tendon insertion. It is sometimes necessary to cut out the counter of the shoe.

Injection of local anesthetic and compound F into the bursae frequently relieves the inflammation. Surgery is seldom necessary. Excision of the plantar bursa of the os calcis is often followed by the formation of painful scar tissue. Resection of the prominence of bone about the Achilles tendon insertion narrows the os calcis and relieves the pressure.

PARALYTIC DEFORMITIES

Polio-myelitis and other neuromuscular afflictions are the etiologic factors in many disabling deformities of the foot. Since these diseases frequently occur in childhood during the growth period, muscle imbalance and contractures produce profound changes in the foot. Growth disturbance in the paralyzed leg may result in unequal leg length and foot size.

Obviously countless combinations and grades of muscle weakness and paralysis can take place following polio-myelitis. The major deformities however follow the pattern of the cardinal positions of the foot—equinus, calcaneus, varus or valgus (Fig. 33). Combined deformities are usual. The high arch, or cavus, or flatfoot may accompany these deformities. Frequently cavus and equinus or cavus and calcaneus deformities are combined. Pes planus and valgus or cavus and varus occur in combination.

These deformities result from muscle imbalance. The development of deformity follows a pattern. First, dynamic muscle imbal-

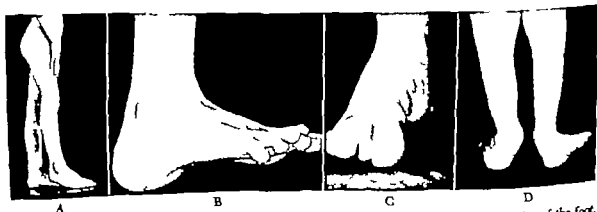


Figure 33. A, Equinus position of the foot. B, Calcaneus position of the foot. C, Varus position of the foot. D, Valgus position of the foot.

once appears which is exaggerated by use of the part. Static deformities appear accompanied by contractures. Alteration of bone configuration occurs with growth. These changes lead eventually to fixed deformity.

The deformities of the foot may be classified according to the loss of power in the major groups, dorsiflexors, evertors, invertors, intrinsic, and plantar flexor muscles.

As in all paralytic disorders prevention of deformity by preventing contractures and developing maximum muscle power in the remaining muscle fibers is essential to rehabilitation. Physical medical measures consisting of active exercise, stretching of contracted muscles, supporting braces and corrective shoes are aids in the prevention of deformity. However when gross muscle imbalance is present, deformities will develop in the growing child and to a lesser degree in the adult. Restoration of muscle balance and power is necessary to correct the deformity and provide maximum function. Often it is impossible to obtain sufficient power from other muscles to effectively substitute for those paralyzed. Stabilization of foot joints to provide a firm, stable base for walking and standing is then necessary. When deformities are established, the deformity must first be corrected, then the foot balanced and stabilized. This may demand stretching of contractures with wedged casts, surgical length-

ening of soft tissues or resection of deformed bones to re-establish proper weight bearing.

Paralysis of Dorsiflexor Muscles. When all of the dorsiflexors of the foot are functionless, i.e., the tibialis anterior and long toe extensors the foot assumes the equinus position because of the unopposed power of the tri-

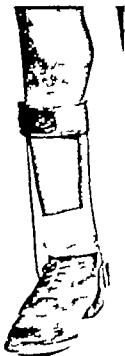
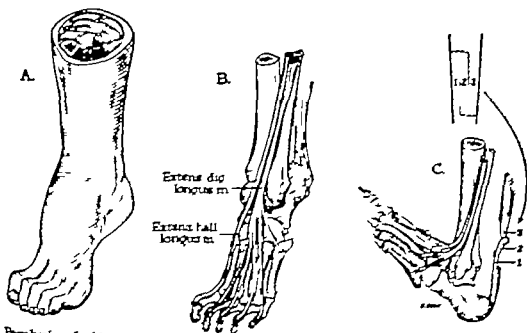


Figure 34 Drop-foot brace for paralysis of the dorsiflexors of the foot.



Paralysis of tibialis anterior

Figure 35 A and B Paralysis of the tibialis anterior muscle with the remaining muscles balanced often produces equinus deformity by contraction of the calf muscles. The extensor longus tendons of the toes produce claw toes and equinus deformity may occur. C. To convert the long extensor muscles of the toes to dorsiflexors of the foot, the Achilles tendon is lengthened or stretched. The extensor hallucis longus is transferred from the first metatarsal and the extensor digitorum longus to the base of the middle metatarsal.

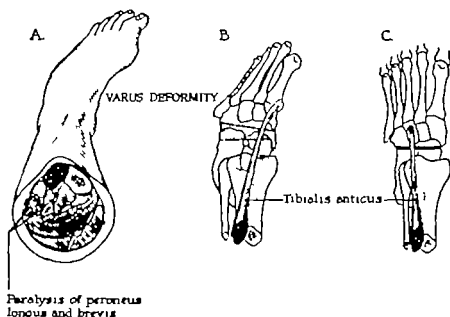


Figure 36 A, Paralysis of the peroneus longus and brevis allows the tibialis anticus (B) to invert the foot and elevate the first metatarsal bone. B, Triple arthrodesis to stabilize the foot is combined with (C) transference of the tibialis anticus to the middle of the tarsal region.

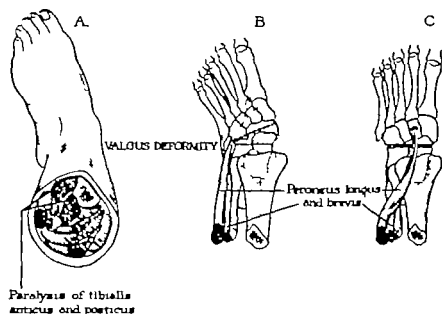


Figure 37 A, Paralysis of the invertors—the tibialis anticus and posticus—produces valgus deformity by the action of the peroneal and toe extensor muscles. B, Stabilization of the foot by triple arthrodesis, followed by (C) transfer of the peroneal muscles to the middle tarsal region.

ceps surae. If the peroneal and tibialis posticus are normal, the foot is usually balanced medially and laterally. If the calf muscle group is regularly stretched in walking, fixed equinus may not occur and little deformity is present. In walking, it is necessary to raise the leg higher to clear the ground when swinging the planter flexed foot forward. A lightweight footdrop brace may be all that is necessary to provide a practically normal gait (Fig. 34). If the calf muscle group has been allowed to contract so that a fixed equinus

position of the foot is present, the muscle may be stretched with wedged casts, forcing the foot into dorsiflexion. In exceptional instances it may be necessary to lengthen the Achilles tendon, however this should be avoided if possible as inevitably some weakness of plantar flexion and push-off in walking results (Fig. 35).

Paralysis of the Evertor Muscles. When paralysis of the peroneus longus and brevis muscles occurs, the primary evertors are lost except for the rather weak evertor action of

the long extensors of the toes. The peroneus longus acts as a depressor of the first metatarsal bone as well as an evertor. The unopposed dorsiflexor invertor action of the tibialis anticus elevates the first metatarsal and inverts the foot. The tibialis posterior inverts and adducts the forefoot. The weak action of the long toe extensors acting as evertors is insufficient to overcome this tendency and the foot gradually assumes the position of varus of the heel adduction of the forefoot and elevation of the first metatarsal. The great toe flexes in an attempt to contact the floor. With growth, the bones adapt to this position and the deformity becomes fixed (Fig. 36A).

The foot may be balanced by transferring the tibialis anticus tendon from the normal insertion on the cuneiform first metatarsal to a more lateral position on the dorsum of the foot in the middle or lateral cuneiform or the second or third metatarsal. The strong invertor action is thus eliminated and when done early may be all that is required. If deformity of the bones and marked contraction of the soft tissue on the medial side of the foot are present wedge resection of the bone with triple arthrodesis of the talonavicular, calcaneocuboid joints and the subtalar joints may be necessary reshaping the foot to a normal weight bearing position (Fig. 36B and C).

Paralysis of the Invertor Muscles. Paralysis of the tibialis anticus and posticus mus-

cles allows overaction of the peroneals, forcing the foot into valgus or eversion (Fig. 37A) with complete loss of the longitudinal arch and a completely flat foot with an everted heel (Fig. 33D). If the calf muscle is allowed to contract an equinovalgus deformity develops. If the long extensor tendons of the toes are lost dorsiflexion of the foot is lost as well. The peroneus longus and brevis may be transferred to the dorsum of the foot to remove the deforming evertor action and to produce dorsiflexion of the foot (Fig. 37B and C). Since medial and lateral stability is lost, triple arthrodesis is necessary to stabilize the foot or to correct fixed deformity. If the foot is in equinus, this must first be corrected by wedged casts or Achilles tendon lengthening.

Paralysis of the tibialis anticus with normal posterior tibial and peroneals and strong long toe extensors may result in a cavovarus deformity. In this instance, the tibialis posterior may prevent the valgus deformity of the heel. The peroneus longus depresses the first metatarsal and the extensor hallucis longus forces the toe into cock up position further depressing the first metatarsal. A paradoxical situation arises dorsiflexion of the foot causes the foot to evert through the action of the long extensors of the toes. However on weight bearing the fixed depression of the first metatarsal head forces the foot into varus position.

Transferring the long extensor of the great



Figure 33. Prolonged equinus position of the foot either secondary to paralysis or clubfoot produces a flat tibiotalar articulation. This deformity of the bone prevents dorsiflexion.

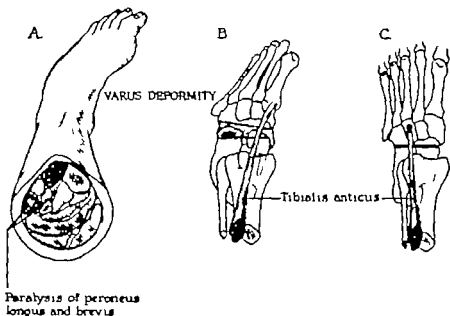


Figure 36 A Paralysis of the peroneus longus and brevis allows the tibialis anticus (B) to invert the foot and elevate the first metatarsal bone B Triple arthrodesis to stabilize the foot is combined with (C) transference of the tibialis anticus to the middle of the tarsal region.

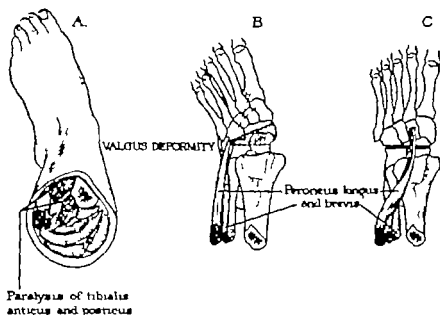


Figure 37 A, Paralysis of the invertors—the tibialis anticus and posticus—produces valgus deformity by the action of the peroneal and toe extensor muscles. B Stabilization of the foot by triple arthrodesis, followed by (C) transfer of the peroneal muscles to the middle tarsal region.

ceps surae. If the peronei and tibialis posticus are normal, the foot is usually balanced medially and laterally. If the calf muscle group is regularly stretched in walking, fixed equinus may not occur and little deformity is present. In walking, it is necessary to raise the leg higher to clear the ground when swinging the planter flexed foot forward. A lightweight footdrop brace may be all that is necessary to provide a practically normal gait (Fig. 34). If the calf muscle group has been allowed to contract so that a fixed equinus

position of the foot is present, the muscle may be stretched with wedged casts, forcing the foot into dorsiflexion. In exceptional instances it may be necessary to lengthen the Achilles tendon, however this should be avoided if possible as inevitably some weakness of plantar flexion and push-off in walking results (Fig. 35).

Paralysis of the Evertor Muscles. When paralysis of the peroneus longus and brevis muscles occurs the primary evertors are lost except for the rather weak evertor action of

the long extensors of the toes. The peroneus longus acts as a depressor of the first metatarsal bone as well as an evertor. The unopposed dorsiflexor/invertor action of the tibialis anticus elevates the first metatarsal and inverts the foot. The tibialis posterior everts and adducts the forefoot. The weak action of the long toe extensors acting as evertors is insufficient to overcome this tendency and the foot gradually assumes the position of varus of the heel, adduction of the forefoot and elevation of the first metatarsal. The great toe flexes in an attempt to contact the floor. With growth, the bones adapt to this position and the deformity becomes fixed (Fig. 38A).

The foot may be balanced by transferring the tibialis anticus tendon from the normal insertion on the cuneiform first metatarsal to a more lateral position on the dorsum of the foot in the middle or lateral cuneiform or the second or third metatarsal. The strong invertor action is thus eliminated and when done early may be all that is required. If deformity of the bones and marked contraction of the soft tissue on the medial side of the foot are present wedge resection of the bone with triple arthrodesis of the talonavicular, calcaneocuboid joints and the subtalar joints may be necessary, reshaping the foot to a normal weight bearing position (Fig. 38B and C).

Paralysis of the Invertor Muscles. Paralysis of the tibialis anticus and posticus mus-

cles allows overaction of the peroneals forcing the foot into valgus or eversion (Fig. 37A) with complete loss of the longitudinal arch and a completely flat foot with an everted heel (Fig. 33D). If the calf muscle is allowed to contract an equinovalgus deformity develops. If the long extensor tendons of the toes are lost dorsiflexion of the foot is lost as well. The peroneus longus and brevis may be transferred to the dorsum of the foot to remove the deforming evertor action and to produce dorsiflexion of the foot (Fig. 37B and C). Since medial and lateral stability is lost, triple arthrodesis is necessary to stabilize the foot or to correct fixed deformity. If the foot is in equinus this must first be corrected by wedged casts or Achilles tendon lengthening.

Paralysis of the tibialis anticus with normal posterior tibial and peroneals and strong long toe extensors may result in a cavovarus deformity. In this instance the tibialis posterior may prevent the valgus deformity of the heel. The peroneus longus depresses the first metatarsal and the extensor hallucis longus forces the toe into cock up position further depressing the first metatarsal. A paradoxical situation arises dorsiflexion of the foot causes the foot to evert through the action of the long extensors of the toes. However on weight bearing the fixed depression of the first metatarsal head forces the foot into varus position.

Transferring the long extensor of the great



Figure 38. Prolonged equinus position of the foot either secondary to paralysis or clubfoot produces a flat tibiotalar articulation. This deformity of the bone prevents dorsiflexion.

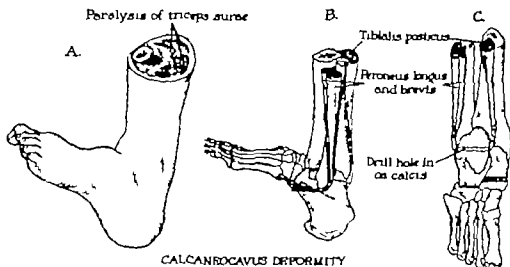


Figure 39 A, Paralysis of the triceps surae muscles prevents active plantar flexion. Unopposed dorsiflexor muscles pull the foot into dorsiflexion. The long and short muscles of the plantar aspect contract to compensate for the triceps surae in walking and produce cavus deformity. B, The deformity of the os calcis and metatarsal bones is corrected and the foot stabilized by triple arthrodesis. C, The tibialis posterior tendon and the peroneal tendons are transferred to the os calcis to provide limited plantar flexion and counteract the deforming pull of the plantar muscles.

toe to the neck of the first metatarsal helps to elevate the metatarsal and eliminates the dorsiflexion of the toe. Transfer of the peroneus longus tendon to the dorsum of the foot in the region of the base of the second metatarsal eliminates the depressor effect on the first metatarsal and aids in dorsiflexion.

Intrinsic Muscle Paralysis of the Foot. Cavus deformity with claw toes may be produced by intrinsic paralysis of the muscles of the foot. Loss of the lumbricales and interossei and short flexors of the toes which normally produce extension of the interphalangeal joints allows hyperextension of the toes at the metatarsal phalangeal joints and the long flexors of the toes produce flexion of the interphalangeal joints. This deformity depresses the metatarsal heads and, coupled with soft tissue contracture of the plantar structures, cavus deformity results.

Before fixed bony deformity develops, transfer of the long extensor tendons to the necks of the metatarsals eliminates the dorsiflexion of the toes and aids in elevation of the metatarsal heads. Interphalangeal joint fusion of the great toe is usually necessary to prevent excessive plantar flexion at this joint. Plantar fasciotomy may be necessary to correct a mild cavus deformity. Often coupled with this defect is insufficiency of the tibialis anticus, producing an equinovarus deformity. The calf muscles or Achilles tendon is first stretched or lengthened to correct the equinus deformity (Fig. 38). The above procedure is then done on the extensor digitorum



Figure 40 Weight bearing x-ray of calcanocavus foot. The os calcis is vertical and the forefoot relatively plantar flexed. Toes are contracted.

longus is transferred to the base of the third metatarsal to produce greater dorsiflexion power.

When fixed cavus deformity has occurred, dorsal wedge section through the cuneiform-navicular joints and through the body of the cuboid is demanded. The high arch is further depressed by plantar displacement of the distal lever of the foot.

Paralysis of the Plantar Flexor Muscles. Paralysis of the triceps surae, or calf muscle group produces one of the most serious disabilities in the growing child—the calcaneus or calcanocavus foot (Fig. 39A). In bilateral paralysis which may occur in poliomyelitis or spina bifida with myelodysplasia, the disability may be previously

pointed out the forward sway of the body in balancing while standing is counteracted by the strong calf muscles. When paralysis of this muscle group takes place this effect is lost and these people cannot balance well without external support. When walking, the normal push-off is lost and the foot is forced into dorsiflexion. When the pull of the triceps surae is lost on the growing calcaneus the unopposed intrinsic muscles of the foot along with the plantar aponeurosis and the flexors of the toes tend to approximate the heel toward the metatarsal heads (Fig. 40). The os calcis grows vertically producing an abnormally high arch. The weight bearing surface of the heel becomes a peg and provides less bearing surface. Valgus or varus of the heel may also be coupled with the deformity when there is weakness of the invertors or evertors.

Since the calf muscle group is more powerful than all of the other long muscles of the foot combined it is almost impossible to reproduce normal plantar flexion power by tendon transference to the heel. However, every effort should be made early in the development of the deformity to prevent progressive changes.

Long leg braces should be used to prevent hyperextension at the knees and a checkrein at the ankle should be incorporated in the brace to prevent excessive dorsiflexion. Plantar fasciotomy may lessen the pull on the metatarsals and os calcis and lessen the bowstring effect. Early transfer of available active muscles to the os calcis is essential. When the peroneals and posterior tibial are normal, they may be transferred to the os calcis and the foot will remain balanced medially and laterally and the forefoot depression is lessened (Fig. 39B and C). If maximum power is required the tibialis anticus may be transplanted posteriorly into the heel through the interosseous membrane. If the calf muscle group is completely paralyzed the transferred muscles will not produce enough power to allow tiptoe walking, however they tend to counteract the vertical growth of the os calcis and eliminate some of the excessive dorsiflexion power. Practically all feet affected with this deformity require some bone correction, either dorsal wedge section to correct the forefoot drop or triple arthrodesis with removal of appropriate bone wedges to correct the deformity and stabilize the foot. The bone procedures must await the time when the foot is more mature and are seldom done before the patient is ten or twelve years of age.

When the foot is completely flail there is little tendency to progressive or fixed deformity other than flatfoot. To secure a stable base for walking, or standing, it may be necessary to arthrodesis the ankle joint along with the subtalar and calcaneocuboid and talonavicular joints.

Spastic Paralytic Deformities. Spastic paralysis of the lower extremity may produce changes in the foot similar to those enumerated above. However the most frequently encountered deformity is equinus due to spasticity and contracture of the calf muscle group. Tendon transference in spastic paralysis is much less useful than in flaccid paralysis as normal muscles are usually not available to transfer and the deformities result from overactive abnormal muscles.

Lengthening of contracted muscles and soft tissues is useful either by stretching casts or by surgical lengthening of tendons or section of fascia and soft tissue. Occasionally a deforming muscle pull may be weakened by partial denervation of the muscle. The often encountered equinus deformity tends to be recurrent particularly during periods of rapid growth. The use of splints worn at night is helpful in preventing the deformity. These splints hold the foot in dorsiflexion preventing contracture of the calf muscle. Gait training is essential. The child is taught the importance of making the heel contact the ground in walking.

If the deformity progresses, wedged and stretching casts are useful. The stretching cast is applied below the flexed knee with the foot in maximum dorsiflexion. An anterior heel is incorporated in the cast. When the knee is straightened in walking, the gastrocnemius muscle originating on the femoral condyles is stretched. If the soleus muscle is not excessively contracted, this method of treatment will re-establish dorsiflexion in walking. However the procedure must be repeated as the child grows. If excessive spasticity of the calf muscle group prevents maintenance of the corrected equinus, neurectomy of the motor nerves to the gastrocnemius muscle is done in the popliteal space. This lessens the power of the muscle group and may prevent recurrence of the equinus. When the equinus recurs after this procedure the musculotendinous junction of the triceps surae is lengthened. This procedure consists in making chevron like incisions on alternate medial and lateral aspects of the junction of the Achilles tendon and the muscle, thus lengthening the muscle as well as the tendon.

If the Achilles tendon alone is lengthened,

a small misshapen calf muscle results and muscle function is lessened.

Flatfoot are frequently encountered with the short contracted calf muscle group. Or thopedic shoes with rigid extended counters tend to preserve the arch after overcoming the contractures. Rarely triple arthrodesis may be required to correct excessively deformed flatfoot.

OSTEOCHONDRITIS

Osteochondritis of the growing foot occurs usually in three locations—the second metatarsal head, the navicular bone and the apophysis of the calcaneus. The etiology of the condition is not clear but the progressive changes noted in x ray examinations of the bones suggest interference with the blood supply of the bone with necrosis followed by substitution of new bone. Local metabolic, toxic or traumatic factors may have a place in the etiology of the disturbance. The osteochondritic lesions occur during the periods of rapid growth between four and thirteen years of age in the case of the calcaneal apophysis and navicular bone, and near the age of closure of the epiphysis at eighteen years in the second metatarsal bone.

The symptoms are pain on weight bearing, swelling and local tenderness to pressure over the involved bone. The onset of symptoms may be acute or gradual and the disease is self limited but may be symptomatic for weeks to months.

X ray findings are irregular density of bone, fragmentation and at times abnormal growth of bone.

When the condition occurs in the region of the second or third metatarsal head, it is called Freiberg's infraction. The symptoms are pain and swelling about the involved joint, particularly at the push-off phase of the step. Enlargement of the head and distal shaft of the bone, flattening and widening of the joint surface, irregularity of the epiphyseal plate and increased joint space between the proximal phalanx of the toe and the metatarsal head characterize the lesion. Degenerative arthritic changes in the joint occur later and may produce symptoms in adult life.

During the acute, painful stage of the disease, weight bearing should be relieved with crutches. Hot packs to the foot may be applied several times daily. Usually the acute distress is of short duration—a week or two. Following relief of the acute pain and swelling, the foot should be fitted to a stiff shank, low-heel shoe with a stiff leather sole to pro-

tect the metatarsal head. A felt pad is placed in the shoe just back of the metatarsal head to relieve pressure on the head and joint and a transverse metatarsal bar is placed on the sole of the shoe.

Alteration of growth of the metatarsal joint surface may lead to chronic degenerative arthritic changes in adult life which, if not relieved by pads and arch supports, may require resection of the proximal third of the phalanx of the toe to form a painless pseudarthrosis. Excess bone about the head of the metatarsal may be removed at the same time if the bone is prominent.

When the condition occurs in the navicular bone, it is termed Köhler's disease and is found between the ages of four and ten years. Pain and swelling about the navicular bone on weight bearing are the usual symptoms. Non weight bearing during the acute phase may be necessary however the symptoms are usually mild and padding the shoe with felt may relieve the strain on the longitudinal arch.

X ray studies reveal a progressive narrowing of the navicular bone between the talus and cuneiform bones. The bone increases in density and may project dorsally above the level of the adjacent bones. Substitution of bone takes place slowly but in several years the navicular bone is usually completely restored with little, if any residual deformity.

Calcaneal apophysitis occurs in the apophysis of the os calcis (Fig. 41). Pain is present at the prominence of the heel on weight bearing and about the attachment of the Achilles tendon. The symptoms frequently appear between the ages of seven and thirteen years. X-rays show increased density of the calcaneal apophysis with fragmentation and irregularity of the epiphyseal plate. Non weight bearing with crutches may



Figure 41 Osteochondritis apophysitis of the

be necessary for several weeks followed by limited weight bearing with a built up heel and sponge rubber pad in the shoe to lessen the pull of the Achilles tendon and to cushion the heel. Healing takes place over several months with no residual deformity.

ARTHRITIS

Rheumatoid arthritis, gout and osteoarthritis may affect the foot in varying degrees. Rheumatoid arthritis and gout affect peripheral joints characteristically. The toes and metatarsophalangeal joints are frequently affected however any and all parts of the foot may be involved. Osteoarthritis may be found in the first metatarsophalangeal joint secondary to trauma or hallux valgus but the disease frequently involves the metatarsal area and the subtalar joint. During the acute phase of rheumatoid arthritis the inflammatory process may be confined to the interphalangeal joints or may progressively affect the metatarsophalangeal joints, the metatarsal, subtalar and ankle joints. The tissues swell and there is increased local heat. Weight bearing is often impossible. Frequently the heel pad becomes painful over the plantar aspect and about the margin of the heel. The plantar fascia may become tender as may the Achilles tendon. Rheumatoid nodules may form over the dorsum of the foot in the bunion areas about the heel and occasionally on the plantar aspect of the foot. Bursae filled with fibrin flecked yellow, cloudy fluid may form over the metatarsal heads. Later in the course of the disease debris and fibrin may form cottage cheese like deposits in these bursae. As the disease progresses the joint surfaces are destroyed by a synovial pannus and fibrous ankylosis may take place in the joints of the foot and toes. Contractures of the toes may develop early. The usual deformity is one of dorsal dislocation and hyperextension of the proximal phalanx and flexion of the interphalangeal joint. All of the toes may deviate laterally. As the subtalar joint is destroyed, lateral motion is lost and the foot becomes more rigid. Destruction of the joint capsules and loss of elastic tissue in the ligaments may lead to flatfoot deformity. When the toes contract, the metatarsal heads become prominent in the sole of the foot and the fat pad in the ball of the foot atrophies. Weight bearing then results in callous formation over these areas of pressure.

During the acute phase of the disease, the patient is placed at bed rest and no weight bearing is allowed. Since contractures de-

velop early, equinus deformity is prevented by placing a padded footboard at the bottom of the bed. The weight of the bed clothing is removed from the feet and the feet are kept in neutral position with the sole of the foot supported by the board. Often the heels are tender and pain is produced when they are resting on the mattress. A small pillow under the calf of the leg lifts the heel off the mattress and relieves the pressure. Hot moist packs applied several times a day for one hour help relieve the pain and prepare the feet for mild active exercise. Muscles atrophy rapidly in rheumatoid arthritis and joints tend to stiffen quickly. To minimize this the patient is instructed in active exercise of the foot without weight bearing. The toes should be flexed and extended, the foot inverted and everted and plantar flexed and dorsiflexed several times following use of the hot packs. Vigorous massage, whirlpool baths and stretching aggravate the condition and should not be employed during the acute phase of the disease. Administration of steroid and salicylate drugs and the general supportive measures for the treatment of rheumatoid arthritis should be started.

When the pain, heat and swelling subside, weight bearing may be started on a very restricted schedule walking only a few minutes of each hour or several times a day. A low heel, well fitted Oxford must be used, not a soft bedroom slipper as the weakened ligaments demand support. Usually rigid arch supports and metatarsal bars are not tolerated. Felt pads placed in the shoe and occasionally a foam-rubber insole help to support the arches and cushion the weight bearing surface of the foot.

Rheumatoid nodules and bursae may be excised if they persist and are subject to pressure. Injection of compound F into joints and bursae has been disappointing.

When deformities have occurred, they usually involve the forefoot. If relief of contracted toes and plantar calluses is not obtained with arch supports, operative correction becomes necessary. Since the joint surfaces are destroyed, the extensor tendons of the toes contracted and the metatarsal heads prominent, the proximal phalanx of the lateral four toes is resected. This allows the toes to fall into a more normal position and shoes can be fitted more comfortably. The Keller type resection of the proximal third of the proximal phalanx will relieve pain in the metatarsal phalangeal joint. The prominent metatarsal heads are shaved flat, parallel to the plantar surface of the foot. This

converts the rounded marble-like head into a flat spatula. Adjacent remaining fat tends to cover the partially resected bone and a fairly effective cushion at the ball of the foot is re-established. Arthrodesis of other painful joints may be done. Following operative correction of deformities, an adequate supporting low-heel Oxford with transverse and long flexible arch supports should be used permanently.

OSTEOARTHRITIS

Osteoarthritis frequently follows trauma to the joints of the foot. The trauma may be massive, producing fractured joint surfaces or it may be minimal and often repeated occurring with abnormalities of weight bearing. The degeneration of cartilage and the production of osteophytes about the margins of the joint is a slow process and symptoms may not develop in the foot until the changes are far advanced or the foot is suddenly traumatized or strained by unusual activity. Pain is often confined to one joint. The subtalar joint following fracture of the os calcis is frequently the site of painful osteoarthritis. The great toe joint degenerates in time when hallux valgus or rigidus is present. Pain usually is produced by chronic synovitis secondary to the destruction of joint surfaces. When acute synovitis is present, rest, heat and elevation of the foot are necessary and weight bearing should be eliminated. Usually the symptoms subside quickly. The foot is then fitted with a supporting Oxford with appropriate arch supports. If the foot is flat and fairly rigid because of the joint changes, radical raising of the arch should not be attempted. Fixed joints do not tolerate extensive position changes. Pads should be built up gradually in the transverse and long arches until comfortable weight bearing is obtained.

Occasionally injection of compound F into the osteoarthritic joints will give relief of pain for varying periods. Salicylate drugs are helpful.

Occasionally arthrodesis of the subtalar joint is necessary in the younger patient with adequate circulation. Extensive operative procedures in the elderly are not warranted. The Keller bunionectomy is useful for relief of the damaged metatarsophalangeal joint of the great toe.

GOUT

Gout is a metabolic disturbance of uric acid metabolism. Greater amounts of uric acid are formed in the body periodically. Attacks of acute arthritis develop. The metatarsophalangeal joint of the great toe is a characteristic area for the lesion. The sudden pain, heat, redness and swelling resemble cellulitis, and fever and leukocytosis may add to the confusion. The serum uric acid level should be determined to confirm the diagnosis. The pain is intense and weight bearing is impossible. The maximum area of tenderness is usually along the medial side of the joint. The discoloration has a cyanotic tone rather than the brighter red of infection. Symptoms subside rapidly with use of colchicine and uricosuric agents and little, if any, residual change remains. Ten per cent of patients who have gout have deposits of urates in the skin, subcutaneous tissue, tendons, ligaments, bones and joints. These tophi are destructive. Severe crippling of the foot can take place if the disease is inadequately treated. Proper therapy to prevent destruction by tophi demands the routine continued use of uricosuric agents. These drugs increase excretion of uric acid by blocking tubular resorption in the kidney. The most effective agent at present is probenecid although salicylates have long been used. Colchicine is an ancient drug specific for relief of pain in gout, however it has no effect on the serum uric acid level. The two drugs should be combined in the management of gout.

When tophi form in the foot, they should be removed surgically when they cause pressure, interfere with tendon movement, ulcerate or destroy bones, joints and tendons (Fig. 42). Because the deposits of urate are infiltrative, special surgical techniques are required to preserve vital structures.

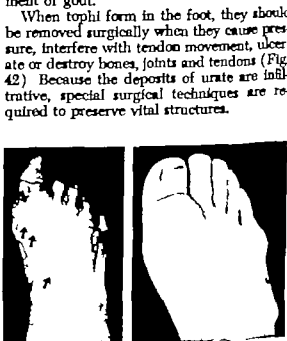


Figure 42. Chronic tophaceous gout. Massive deposits of urates destroy bones and joints and produce deformity of the foot. (Reproduced by permission from *The Surgical Management of Chronic Tophaceous Gout*, by W. R. Larmon and J. F. Kurtz, *J. Bone & Joint Surg.* Vol. 40-A, pp. 743-772, July 1958.)

LEDDERHOSES DISEASE

Dupuytren's contracture of the hand is a familiar affliction. Contracture of the palmar fascia with the formation of nodules and cords producing fixed flexion of the fingers is well known. Microscopically the fascia is more cellular than normal with increased collagenous elements. A similar affliction occurs in the plantar fascia of the foot and may be associated with the Dupuytren's disease of the hand. The condition in the foot is called Ledderhoses disease. Painful nodules develop in the plantar fascia of the foot. The nodules slowly increase in size and pain on weight bearing is complicated by burning pain at rest, particularly at night. The disease must not be confused with Volkmann's ischemic contracture of the foot or the fibrous contractures which follow cast immobilization for fractures of the lower extremities. These conditions may produce tight plantar fasciae and deformed toes but the nodules typical of Ledderhoses disease do not develop.

The plantar fascia should be resected when the painful masses do not spontaneously regress. The fascia may be excised through a medial plantar incision avoiding the weight bearing areas of the sole of the foot. Weight bearing must be delayed until the wound is completely healed to avoid excessive scar formation—usually three weeks.

TUMORS OF THE FOOT

Tumors of the foot are uncommon lesions when compared to the many other lesions which affect the foot. The classification of tumors of the hand applies equally well to the foot with the exception of irradiation carcinoma. This lesion in the foot usually follows excessive radiation in the therapy of plantar warts but is of rare occurrence. Tumors of the foot usually demand attention when they occur on the weight bearing surface of the foot and produce pain. When they occur on the dorsum of the foot or produce enlargement of the foot, shoe fitting is difficult.

The principles of treatment are similar to those in the hand. However there are several differences. The plantar or weight bearing skin of the foot must be avoided in the excision of tumors of the foot, to prevent painful scars. If radical excisions are necessary which involve supporting ligaments, tendons or bone, stabilization of the foot by appropriate arthrodesis may be necessary.

Partial amputations for tumors of the foot must preserve adequate balance and painless weight bearing. If this cannot be done

amputation at a higher level in the leg is mandatory.

INFECTIONS

Infections in the foot fortunately are usually superficial and may be caused by Strep. *Staphylococcus* fungus or other inhabitants of the skin. Deeper infections of the fascial spaces, tendon sheaths and bones and joints are uncommon. The plantar skin is thick and affords good protection for the deeper structures except in the case of penetrating wounds or from extension of infection from the toes.

The most common source of infection is from blisters, bursae, corns or ingrowing toenails. Blisters usually occur over the heel near the insertion of the Achilles tendon or at the dorsum of the toes from friction of shoes or tight stockings.

Bursae over the first and fifth metatarsal heads secondary to corns may become infected through cracks in keratotic skin. Usually the infections are localized however infection in the toes may drain through the lymphatics of the superficial dorsal tissues of the foot creating spreading cellulitis and lymphangitis. Infections about the heel drain through lymphatics on the posterior aspect of the leg to the popliteal lymph nodes. The cardinal signs of infection—heat, redness, swelling and pain—are present. Since the skin is loosely applied to the dorsum of the foot any infection in the foot may produce swelling over the dorsum.

Weight bearing must be eliminated immediately. The foot should be elevated, and warm moist, sterile packs applied. The foot should be splinted with a well padded, molded metal splint. Excessive heat should not be applied particularly in the elderly when vascular impairment may be present. In a leg and foot with considerable arterial insufficiency excessive heat may precipitate gangrene. If a wound or lesion is open, a culture should be obtained and sensitivity tests made for the various antibiotic drugs. If the infection is spreading after the culture is obtained, broad spectrum antibiotics may be administered until the specific antibiotic is determined by the sensitivity tests. The wounds should be dressed as often as necessary to maintain clean care and remove toxic bacterial products.

When the infection originates in a paronychia (Fig. 43) or ingrowing toenail, adequate drainage may be instituted by incision through the eponychium and removal of the offending portion of nail.

If an abscess develops on the dorsum of



Figure 43 Paronychia of great toe.

the foot, it should be incised through a longitudinal incision and the wound packed open with moist gauze, and the warm moist, sterile saline packs and splinting continued. When the drainage has stopped and necrotic tissue separated, the wound may be allowed to close.

Occasionally the deeper tissues may be involved. Infection introduced through puncture wounds in the sole of the foot or spreading along the tendons from the toes involves the fascial spaces of the foot. Pus may accumulate beneath the plantar aponeurosis or in the deep median, central or lateral plantar fascial spaces located between the short plantar muscle groups. Incision and drainage of this area of the foot are done either medially or laterally at the junction of plantar and dorsal skin. Incisions should never be made through the plantar skin or from the dorsum of the foot for drainage of the deep spaces. Incisions through plantar skin usually leave a painful scar in a weight-bearing area and adequate drainage cannot be secured through the dorsal approach, thus resulting in a chronic draining sinus.

Occasionally a collar button like abscess originates in a plantar callus or wart. The infection forms a localized collection of pus beneath the adjacent metatarsal head or may burrow between the metatarsal heads to produce swelling on the dorsum. Pyogenic arthritis involving the metatarsophalangeal joint or osteomyelitis invading the metatarsal shaft and head may occur. This is a serious complication which usually demands surgical drainage and occasionally excision of the

affected joint and metatarsal head. The approach is through the web space where blunt dissection is carried out until the abscess is evacuated. In chronic draining osteomyelitis or pyogenic arthritis the metatarsal head and joint may be excised by extending the incision onto the dorsum of the foot. The wound may be partially closed and a drain inserted in the web space for seventy-two hours. The original wart or callus is treated locally.

Osteomyelitis may be of pyogenic, fungus or tuberculous origin. Whenever possible, the organism must be identified. Pyogenic osteomyelitis is treated as elsewhere in the body. Rest and splinting of the part and massive doses of appropriate antibiotics are used. Saucerization of involved bones may be necessary when draining sinuses develop, along with excision of sinus tracts. Often joints are destroyed and spontaneous bony or fibrous fusion takes place. Therefore it is essential to maintain the foot in weight-bearing position while treating the infection. This may be done with splints or casts.

Tuberculous arthritis and osteomyelitis involving the foot are extremely serious, as are the fungus infections. Before the advent of antibiotic therapy and chemotherapy appropriate for these infections, amputation was the usual result. The outlook is now more hopeful with the availability of these drugs. However the old principle of immobilization in tuberculosis still applies. Cast fixation may be necessary with non-weight bearing for many months and surgical fusion of infected joints may be necessary to permanently arrest the lesion.

Superficial skin infections in the foot are commonly caused by fungus or yeast organisms. The condition is known as dermatophytosis (Fig. 44). The common causative organisms are the *Epidermophyton*, *Trichophyton* or *Candida albicans*. These organisms are normal inhabitants on the skin. They require moisture, warmth and an alkaline medium for growth. In the presence of these conditions and lowered resistance of the skin, infection can take place. A frequent area of invasion is between the toes, particularly the fourth and fifth toes which fit closely together and the plantar surface of the foot and toes. The lesions produce maceration between the toes in which cracks may occur allowing secondary invading bacteria to cause spreading cellulitis. Lesions on the plantar aspect may resemble the lesions of ringworm or they may be vesicular filled with clear or milky fluid. Occasionally nu-



Figure 44 Epidermophytosis of the feet: lesions about the toes and plantar aspect of the foot with deformity of the toenail

merous small lesions coalesce or large bullous lesions develop. The bullae may rupture, followed by crusting and peeling of the superficial layers of the skin. The disease tends to be chronic or recurrent.

Treatment is directed at cleanly care of the feet, reduction of moisture, conversion of the alkaline to an acid medium, and the use of fungicides.

The feet should be washed daily and dried thoroughly. Application of undecylenic acid compounds night and morning changes the skin to an acid medium. Nystatin, a potent fungicide, may be applied daily in resistant infections. Allowing the shoes to dry thoroughly by changing shoes daily reduces the moisture content. Clean stockings should be used daily.

VASCULAR DISEASE OF THE FOOT

When peripheral vascular disease is known to be present, prophylaxis of complicating lesions in the foot should be the concern of the surgeon as well as the patient. Since the foot is the farthest point in the area of the peripheral circulation, manifestations of failure or impairment may occur early or be precipitated by acute failure.

The primary objective of prophylactic measures is the prevention of pressure and friction, exceeding the circulatory capacity, avoidance of excessive heat or cold. All of these factors may precipitate circulatory failure. Trauma is an important factor. Trim-

ming toenails or calluses to the point where the skin is damaged may permit bacteria to invade the tissues, increasing circulatory demands through infection and thus precipitating gangrene.

Toenails must be kept free from debris under the margins. Lubricating the skin with lanolin prevents the accumulation of keratotic skin which may crack or cause pressure on the underlying skin with necrosis. Careful removal of this material with an orangewood stick is helpful. Trimming the nails so that they do not grow in is important. Calluses and corns must be carefully trimmed. When the skin is broken by excessive or careless trimming, infection frequently supervenes in the devitalized tissues. Pumice stone or an emery board may be used carefully to reduce the thickness of calluses and corns. Salicylic acid should not be used as the devitalizing effect on already impaired tissues may allow infection to develop. The shoes and stockings should be carefully fitted to prevent friction and pressure. Excessive moisture and drying should be counteracted by appropriate measures.

READING REFERENCES

- Bechtol, C. O., and Mossman, H. W.: Club-Foot. An Embryological Study of Associated Muscle Abnormalities. *J. Bone & Joint Surg.* 32 A:827 1950.
 Betts, L. O.: Morton's Metatarsalgia. Neuritis of the Fourth Digital Nerve. *J. Australia* 1:514 1940.
 Bohm, M.: The Embryologic Origin of Club-Foot. *J. Bone & Joint Surg.* 11:229 1929.

- Demetralopoulos, N. J., and Mason, M. L.: Ledderhose's Disease. *Quart. Bull. Northwestern Univ. M. School*, 31:333, 1937.
- Duchenne G. B.: *Physiology of Motion*. Philadelphia, W. B. Saunders Company, 1939 (translated and edited by E. B. Kaplan).
- Ellisman, H. A., and Munster, J. T.: The Evolution of the Human Foot with Especial Reference to the Joints. *J. Anat.* 70:56, 1935.
- Freiberg, A. H.: The So-Called Infracture of the Second Metatarsal Bone. *J. Bone & Joint Surg.* 8:257, 1926.
- Grodinsky, M.: A Study of the Fascial Spaces of the Foot and Their Bearing on Infections. *Surg. Gynec. & Obst.* 49:737, 1929.
- Grodinsky, M.: Study of Tendon Sheaths of Foot and Their Relation to Infection. *Surg. Gynec. & Obst.* 51:400, 1930.
- Haines, R. W., and McDougall, A.: *The Anatomy of Hallux Valgus*. *J. Bone & Joint Surg.* 36-B:272, 1954.
- Hammer, E. D. W.: *Diseases of the Foot*. Philadelphia, W. B. Saunders Company, 1950.
- Jones, F. W.: *Structure and Function as Seen in the Foot*. Baltimore, Williams & Williams Company, 1944.
- Jones, R.: The Soldiers' Foot and the Treatment of Common Deformities of the Foot. III Hammer Toe. *Brit. Med. J.* 1:78, 1916.
- Keller, W. L.: Further Observations on the Surgical Treatment of Hallux Valgus and Bunions. *New York M. J.* 95:606, 1912.
- Klopsteg, P. E., and Wilson, P. D.: *Human Limbs and Their Substitutes*. New York, McGraw-Hill Book Company, 1954.
- Lake, N. C.: *The Foot*, 4th ed. Baltimore, Williams & Williams Company, 1952.
- Larson, W. A.: Surgical Treatment of Deformities of Rheumatoid Arthritis of the Forefoot and Toes. *Quart. Bull. Northwestern Univ. M. School* 35:59, 1931.
- Larson, W. A., and Kurtz, J. F.: The Surgical Management of Chronic Tophaceous Gout. *J. Bone & Joint Surg.* 40-A:743, 1958.
- Mayer, L.: The Physiological Method of Tendon Transplants Reviewed after Forty Years. *Instructional Courses of the American Academy of Orthopaedic Surgeons*, vol. 13. Ann Arbor, Mich., J. W. Edwards, 1956.
- McCarroll, H. R.: Foot Deformities Resulting from Irreparable Nerve Lesions. *American Academy of Orthopaedic Surgeons, Reconstructive Surgery of the Extremities*. Ann Arbor, Mich., J. W. Edwards, 1944.
- Morton, D. J.: Mechanism of the Normal Foot and of Flat Foot. *J. Bone & Joint Surg.* 6:365, 1924.
- Murphy, D. P.: *Congenital Malformations, A Study of Parental Characteristics with Special Reference to the Reproductive Process*, 2nd ed. Philadelphia, J. B. Lippincott Company, 1947.
- Shands, A. H., and Raney, R. B.: *Handbook of Orthopaedic Surgery*, 5th ed. St. Louis, C. V. Mosby Company, 1957.
- Willis, T. A.: *Orthopaedic Anatomy of the Foot and Ankle, Regional Orthopaedic Surgery and Fundamental Orthopaedic Problems, Instructional Courses of The American Academy of Orthopaedic Surgeons*, vol. 1. Ann Arbor, Mich., J. W. Edwards, 1947.

PHYSICAL MEDICINE AND REHABILITATION

By LOUIS B. NEWMAN, M.D.

LOUIS BENJAMIN NEWMAN was interested in electrical currents as a high school student when he was sure he wished to be a doctor. Graduating from the Illinois Institute of Technology in Mechanical Engineering, he spent several years in research upon high frequency currents. He received his doctor of medicine degree from Rush Medical College and has devoted himself to the development of the relatively new specialty of physical medicine. He has had wide experience and has made many contributions to the development of his chosen field.

INTRODUCTION

Manpower is our greatest resource and must be well conserved. Physical medicine and rehabilitation, as part of total medical management, is dedicated toward the objective of maximum rehabilitation of all those disabled from disease or injury. All methods and means of helping man help himself must be used to bring him back to maximum usefulness whenever a disability exists. Physical medicine and rehabilitation properly integrated and coordinated with all of the available medical and other professional services can, through effective diagnostic and therapeutic procedures combined with clinical and basic research, reduce to a minimum the handicap associated with disability. All trained individuals are needed to aid in this tremendous job—a job having the many physical and mental complexities associated with illness. No final word has been or will be

written regarding either diagnosis or treatment.

The scope and need for rehabilitation is broad and ever expanding because of the increase in disease and injury associated with a longer life span and through accidents related to home, industry, various means of transportation and others. There must be a clear realistic awareness and understanding on everyone's part of the value and benefits derived from rehabilitation, including overcoming the psychologic and emotional problems always associated with disease or injury and with hospitalization. The philosophy of the specialty of physical medicine and rehabilitation of early intensive medically prescribed therapy for a sufficient time to meet the specific needs of the sick is basic.

The success of the rehabilitation program which endeavors to return the maximum number of disabled individuals to productive

living in industry business and the home depends to a great extent on selective job placement. The patients over all physical and mental abilities must be carefully matched with the over all physical and mental job requirements. In this way he returns to his community as a useful productive member with a satisfactory measure of happiness and contentment.

DEFINITION AND SCOPE

Physical medicine and rehabilitation is part of the total medical care of the sick and includes the diagnosis of prescribing for and treatment of disease, defect or injury by the use of physical means and restoration to the fullest physical mental social, vocational and economic usefulness possible.

All activities are medically prescribed by the physiatrist (physician specializing in physical medicine and rehabilitation). In order to achieve maximum benefits for the patient, a program closely integrated and coordinated with all of the other medical and paramedical services is essential. Full cooperation and planning among the physiatrist internist, neurologist, neurologic surgeon, orthopedist, urologist, plastic surgeon, psychiatrist, the various therapists, nurse psychologist, social worker vocational counselor recreational technician and others are fundamental. The Physical Medicine and Rehabilitation Service, as established in the Veterans Administration, consists of the physiatrist, rehabilitation coordinator physical therapist, corrective therapist, occupational therapist, manual arts therapist, education therapist, speech therapist, and blind rehabilitation therapist. The Armed Forces as well as some of the civilian rehabilitation institutions have somewhat similar organizations.

The general objective of all treatment of physical and mental disabilities resulting from disease or injury is restitution of maximum function and an achievement of realistic vocational goal.

Physical therapy Medically proscribed diagnostic and therapeutic procedures utilizing heat, cold, hydrotherapy ultraviolet and ultrasound radiation, various forms of electricity and therapeutic exercise

Corrective therapy The application for therapeutic purposes of medically prescribed activities of an exercise and self-care nature in all types of disabilities

Occupational therapy Medically prescribed kinetic, metric, tonic and psychiatric therapy utilizing primarily hand and foot operated tools in such activities as ceramics,

lapidary jewelry making and leather tooling.

Manual arts therapy Therapeutic and exploratory medically prescribed activities through the use of hand and power-driven tools and equipment in woodworking, mechanical drawing, radio and electronics, photography etc.

Educational therapy Medically prescribed treatment to aid in exploring patients interests promote resocialization improve the educational levels and increase work capacity by the utilization of academic commercial and technical studies.

Speech therapy Medically prescribed therapy for disorders in language and communication, including esophageal voice training following laryngectomy the training of aphasic patients, such as those seen after a cerebrovascular accident, for recovery of receptive and expressive language capacities and for the improvement of impairments of abstract ability concept formation, calculation ability reading ability and memory patterns

Blind rehabilitation therapy Medically prescribed treatment for the blinded to aid in reorganizing and adjusting their lives toward maximum usefulness through such activities as eating, walking, grooming, reading and writing braille, the use of office and industrial machines and many other daily living accomplishments

The disabled persons residual potential abilities must be recognized early and through physical medicine and rehabilitation enhanced to the fullest capacity. As soon as it is medically feasible, a patient should receive physical medicine and rehabilitation judiciously prescribed to meet his specific needs which must include psychological and social aid in adjusting to his disability and hospitalization.

Physical medicine and rehabilitation can be considered to consist of three phases or stages not entirely distinct from each other but overlapping to a degree and having continuity. These phases are (1) preventive, (2) definitive and (3) maintenance.

1. Preventive rehabilitation. As long as there is no medical contraindication preventive rehabilitation can be started even in an unconscious patient by instituting proper positioning in bed, the use of splints and passive movements of the extremity through full range of joint motion in order to prevent contractures and deformities and to aid circulation.

2. Definitive rehabilitation specific medi-

cally prescribed rehabilitation procedures are instituted to enhance function so the patient can perform the maximum possible number of self-care activities. It is no longer necessary to wait until the patient can be taken out of his bed. Treatment can be accomplished at the bedside and later continued in the clinic.

3. Maintenance rehabilitation. When maximum rehabilitation has been attained certain therapeutic procedures should be continued so that the patient can at least maintain his accomplishments and not regress. For example if the patient is able to feed himself and ambulate, then these activities should be continued. Frequently continuation of maintenance activities by the patient is impossible in certain progressive disorders.

The various physical and other effective properties of ultraviolet and radiant energy, heat, cold, water, electricity, ultrasound radiation, massage, therapeutic exercise and mechanical devices are employed for both diagnosis and treatment. A rehabilitation objective must be established including both hospital and posthospital planning. Post-hospital follow-up will usually enhance and prolong the beneficial effects of the physical medicine and rehabilitation program. It will serve as a preventive measure in some conditions to reduce the incidence of recurrences and hospital readmissions and also will result in important economic benefits.

Those patients who present special rehabilitation problems can be best served by the integrated action of their physician, physiotherapist, rehabilitation coordinator and other physical medicine and rehabilitation personnel, social worker, vocational counselor, nurse, dietitian, psychiatrist, psychologist and other medical specialists when indicated. The patient should also participate in the planning to the extent of his abilities. The physical, social, emotional and vocational needs of the patient should be discussed and joint planning and concerted action toward achieving a definite rehabilitation goal should result.

BASIC AND GENERAL PRINCIPLES

Even though there are almost countless varieties of disabilities, both as to type and extent, there are certain general and fundamental physical medicine and rehabilitation principles that can be applied as an approach to the problem. There are usually psychologic and emotional problems, to a lesser or greater degree, whenever an individual suffers illness or injury. It is through

the combined integrated efforts and skills of the medical and paramedical specialists that the patient is motivated to participate in the prescribed activities and thereby is aided in overcoming these unhappy complications. He must be helped to accept his disability as well as the rehabilitation program.

Fundamental Objectives. The objectives for total rehabilitation are: To aid the patient in his psychologic, emotional and social adjustment to his disease and hospitalization to relieve the boredom so frequently associated with the slow passage of time during illness and to enhance motivation. Effective use should be made of the hospitalization period. Selective activity participation helps to allay anxiety and fear.

To institute as early as medically feasible, intensive physical medicine and rehabilitation for a sufficient period to relieve pain and secure a minimum degree of handicap.

To prevent general body deconditioning and deformities with their resulting detrimental effects both physical and mental in order to maintain the greatest possible degree of physical fitness and function for the performance of the maximum possible number of self-care and daily living activities, including ambulation.

To furnish when indicated, and to thoroughly train the disabled person in the use of crutches, braces, canes, artificial extremities, wheel chairs and other types of assistive devices for maximum usefulness.

To establish a realistic vocational goal leading to selective job placement and gainful employment for those who because of their disability cannot return to their former type of work. The hospital program should include posthospital planning for full or part time employment and for some individuals who require sheltered or home work.

The maximum number of self-care activities which the patient has the ability to perform must be the foundation for his total rehabilitation; the objectives upon which to build a life of usefulness. These activities are daily accomplishments for the normal person. The Veterans Administration "Self-Care Activities—Functional Evaluation" form lists 154 activities, which have been divided into a number of groups, such as those of eating (drink from cup or glass, eat with fork and spoon, cut with knife), communication (write type, use telephone, open envelope, remove letter from envelope), hygiene (shave, wash hands and face, brush teeth, brush or comb hair, turn faucet on and off,

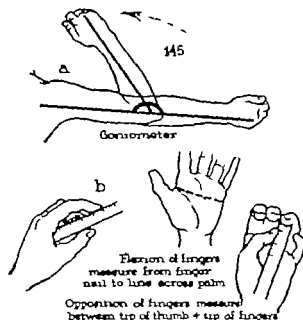


Figure 1 Measurement of range of joint motion. a, Measurement of elbow extension-flexion with goniometer (normal range 0 to 145 degrees) b, Measurement of finger motion with ruler

use toilet, get into and out of bath or shower) dressing (put on braces or prosthesis use zippers, hooks and eyes put on and remove clothing, necktie, hose, shoes put in and remove objects from pocket) locomotion (get in and out of bed, use wheel chair stand, get down and up from floor walk up and down stairs, get into and out of and drive an automobile get into and out of church pew or theater seat) household (open and close drawers, make bed lock and unlock doors, open and close windows, get into and out of chair at table) and miscellaneous (wind watch, hold newspaper turn pages, pick up objects from table and floor open and close safety pins, turn door knobs, use pull cord) Other institutions have developed similar forms for checking patients' accomplishments. One must be realistic and practical, for with some disabilities many of these activities are beyond the realm of accomplishment.

Prior to institution of any treatment the individual receives a complete initial medical examination and evaluation and periodic re-evaluations of his physical, mental and vocational abilities. Physical function depends primarily on the integration of range of joint motion, muscle strength and coordination. Range of joint motion can be evaluated with a goniometer (protractor) and a ruler. For example, normal range of motion of the elbow would be recorded as extension-flexion, 0 to 145 degrees (Fig.

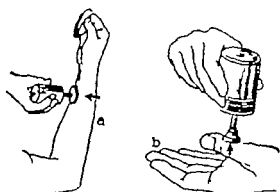


Figure 2 Measurement of muscle strength. a, The strength of the flexor muscles of the right elbow being tested with the myometer. The patient is resisting the force attempting to overcome the strength of the flexor muscles. b, The strength of the abductor pollicis brevis muscle of the left hand being tested with the myometer

1a) Measurements for impaired range for the elbow may be recorded, for example, as 20 to 110 degrees. Two readings are always taken so that not only are the actual number of degrees of movement noted, but also the relation to the normal total range. Other joints are similarly measured. In most instances range of finger movement could be objectively evaluated with a ruler (Fig. 1b) Normal functional range would be considered when (1) the tips of the fingers can touch the palm when they are completely flexed, (2) the fingers can be completely extended and (3) the tip of the thumb can touch the tips of the four fingers of the hand. Both active and passive range of joint motion are to be measured for complete evaluation of movement.

Muscle strength can be measured with a muscle dynamometer. Figure 2a shows the method of testing the strength of the flexor muscles of the right elbow with the myometer. This small hydraulic device accurately measures the resistance offered by a muscle, the reading on the gauge being equal to the force necessary to overcome the isometric contraction of the muscle under test. With the myometer a realistic scientific value of muscle strength in pounds or fractions thereof can be secured repeatedly. On the other hand, in manual muscle testing, many terms are used to describe muscle strength, such as moderate assistance, minimum pressure, slight resistance, great deal of resistance and full resistance, which are empiric, unscientific and inaccurate since they mean different things to various testers. The strength of the abductor pollicis brevis muscle is measured as shown in Figure 2b. The strength of the other muscles or muscle

groups, pronation and supination of the forearm and the hand grip can be similarly evaluated.

When range of joint motion and muscle strength are re-evaluated it is important that the patient and the part being tested be placed in the same position as in all previous tests.

Coordination can be tested by the patient's ability to perform movements such as writing, handling eating utensils, shaving, brushing teeth and picking up small objects. Work capacity can be determined by totaling the amount of activity the patient has performed over a specified time.

When properly instructed and with periodic re-evaluations many patients upon discharge from the hospital can continue certain activities such as therapeutic exercises and heat applications at home and thus shorten their hospitalization period. Frequently therapists can visit the patient at home in order to continue treatment. The home-care program must be under close direction and supervision of the physiatrist. Definite economic and social benefits will be gained, including greater independence with an earlier possibility of gainful employment.

ELECTRODIAGNOSTIC PROCEDURES

Electrodiagnostic techniques are of definite aid in evaluating the status of neuromuscular disorders, specifically those of lower motor neurons. The response of motor nerve and muscles to suitable electrical stimuli must be analyzed and properly interpreted. Formerly the response was noted when using the tetanizing (faradic) and direct current (galvanic) thereby indicating the state of innervation. In denervated muscle, there is a classical response called RD

(reaction of degeneration). Testing for this response is helpful when dealing with a degenerated nerve or a denervated muscle but the procedure is of little value in detecting regeneration.

At present it appears that the use of (1) tetanizing current (2) direct current (3) electromyography and (4) electrical skin resistance measurements can contribute valuable information in diagnosing degenerating, denervated and regenerating nerve-muscle disorders. In electrodiagnosis a small active electrode approximately $\frac{1}{2}$ to 1 inch in diameter is placed over the motor point of the muscle. The large dispersive electrode is placed on the body some distance from the active electrode.

Tetanizing Current The tetanizing current frequently used is a type of alternating current induced in the secondary of an induction coil by the rapid interruption of the current in the primary coil commonly called faradic current. Normally innervated muscles respond to tetanizing current. However in from ten to fourteen days after a nerve is severely injured the nerve and muscle do not respond to this current unless the strength is greatly increased beyond pain tolerance. This is the simplest, most accurate, and earliest electrical sign of a severe nerve injury.

Direct Current Impulse Stimuli. A direct current impulse stimulator is extremely useful in electrodiagnosis. It may be a simple battery or a more complex generator producing square waves which can be varied in polarity, in duration from 0.1 to 1000 milliseconds, in the interval between stimuli in milliseconds and in intensity of current in milliamperes. The type of contraction of the muscle being tested is carefully observed.

Table 1 Electrodiagnostic Data Guide

	NORMAL	DEGENERATING	DENERVATED	REGENERATING
Rheobase	Normal	High to lower than normal	Below normal	Higher than normal
Stimulus-duration curve	Continuous flat slope	Discontinuous steep slope	Continuous steep slope	Discontinuous steep slope
Chronaxie (milliseconds)	Less than 1	More than 15	More than 15	Less than 10
Direct current tetanus ratio	3.5 to 6.0	10.0 to 1.0	1.0 to 1.5	2.0 to 20.0
Repetitive stimulation	Descending curve	Ascending curve	Ascending curve	Ascending curve to level
Rheobase ratio	1.5 to 3.0	3.0 to 1.0	1.0 or less	1.0 to 8.0

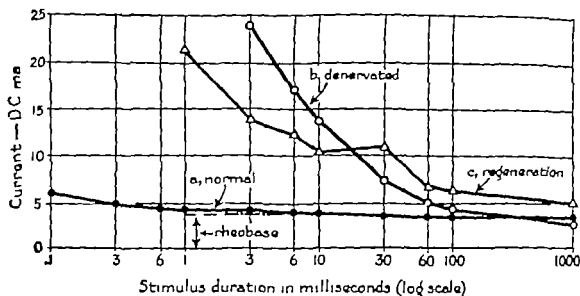


Figure 3 Strength-duration curves. a, Normal—continuous flat slope; b, denervated—continuous steep slope; c, regeneration—discontinuous steep slope.

Normally a rapid twitch is obtained, while in denervation a slow contraction is seen. Usually muscles both proximal and distal to the injury are tested.

The following data, if obtained during examination are helpful: rheobase, strength-duration curve, chronaxie, direct current tetanus and its ratio, repetitive stimulation and rheobase ratio (Table 1).

Rheobase. This is the minimum amount of direct current, lasting a minimum of 500 milliseconds, necessary to produce a minimal muscle contraction.

Strength-duration curve. This is obtained by plotting the current in milliamperes necessary to produce a minimal contraction with decreasing duration of stimulus down to 0.1 millisecond. Figure 3 shows strength-duration curves.

Chronaxie. The number of milliseconds required to produce a minimal contraction when the strength of the stimulus is twice the rheobase is the chronaxie. Normally this is less than 1 millisecond.

Direct current tetanus ratio. This is the quotient resulting from dividing the intensity of the direct current required to produce a tetanic contraction by the rheobase. When the intensity of the direct current for rheobase is increased three to five times, normal muscle remains in tetanic contraction (direct current tetanus).

Repetitive stimulation. The intensity of the direct current stimuli of 1 millisecond required, at intervals of 1.5 and 12 milliseconds between stimuli, to produce a tetanic contraction is recorded as a strength-interval curve for repetitive stimulation. The thresh-

old normally is less for 12 than for 1 millisecond (descending curve). Abnormally this is reversed (ascending curve).

Rheobase ratio. This is the value obtained by dividing the anodal rheobase by the cathodal rheobase. The normal is 1.5 to 3.0.

All of the above guide data must be interpreted in relation to the time elapsed since injury or surgery, the length of the distal segment, edema of the skin at the site of the stimulating electrode, traumatic muscle damage and situations of dual or "overlap" innervation. An increase in rheobase and in direct current tetanus ratio is frequently the earliest sign of nerve regeneration. However, the appearance of discontinuities in the strength-duration curve may appear first. Chronaxie is usually last to return to normal during nerve regeneration.

Electromyography. The electromyograph is an electronic device by means of which the electrical voltages generated by muscle activity can be seen, heard, photographed and recorded (Table 2). There are several methods that can be used to "pick up" the voltages from the muscle being studied. It can be done by means of skin surface electrodes, one surface and one monopolar intramuscular needle by the use of two intra-muscular needles or by means of a single or double coaxial (concentric) needle. Needle electrodes are the most sensitive and reliable. After being amplified these voltages are converted to visible wave forms by a cathode ray oscilloscope and into sound by means of a loud speaker. For permanent records, the wave form on the oscilloscope is photographed and also recorded on a mag-

Table 2 Electromyographic Data Guide

	AMPLITUDE	DURATION	FREQUENCY	SOUND PRODUCE
Normal resting muscle	0	0	0	Silent
Normal motor unit	500-2000 microvolts	10 millisecond	5-30 per second	"Sharp rumble"
Fibrillation potential (denervation)	5-100 microvolts	1 millisecond or less	2-30 per second not constant	"Clicking"

etic recorder for future playback. The electromyograph is of greatest value in demonstrating lesions of lower motor neurons.

Various types of motor unit potentials are seen on electromyographic examination, the type of potential depending on the neuromuscular conditions at the time of examination (Fig. 4). There are many normal variations in the so-called characteristic potentials observed during tests. Electromyographic examination of a normal muscle at rest reveals no demonstrable electrical potential, there being an undisturbed isoelectric base line seen on the oscilloscope, and is referred to as electromyographic silence (Fig. 4A). A normal contracting muscle produces rather characteristic motor unit potentials (Fig. 4B). It will be noted that small positive phases both precede and follow the large characteristic negative phase.

Approximately two weeks after a nerve or nerve root has been severed or severely injured, spontaneous denervation fibrillation voltages (Fig. 4C) and denervation positive voltages (Fig. 4D) appear even though the muscle is at rest. The denervation fibrillation

potentials are of extremely short duration having an initial positive phase immediately followed by an almost equal negative phase. The denervation positive potentials show an initial positive phase having a rapid rise and a long negative phase of relatively low amplitude. Denervation fibrillation potentials and denervation positive potentials are continuous as long as the muscle is not completely fibrosed or degenerated or until it becomes reinnervated. These potentials do not occur in upper motor neuron lesions.

When a nerve is degenerating or regenerating, complex or highly polyphasic motor unit potentials are frequently seen on electromyographic examinations during voluntary effort (Fig. 4E). These potentials have many positive and negative phases.

During electromyographic examination of myotonic muscles there is a rapid discharge of very high frequency potentials immediately after insertion of the needle electrode (Fig. 4F). The sound of these potentials over the loud speaker has been compared with the sound of a diving airplane.

In spinal lesions resulting in faulty innervation as for example in progressive muscle atrophy and chronic poliomyelitis fasciculation potentials are frequently seen on electromyographic examination. These are spontaneous, involuntary complex or highly polyphasic potentials.

Electromyography is an excellent aid in localizing the level of a lesion which is producing nerve root pressure, such as those that may be associated with herniated nucleus pulposus, vertebral fractures, arthritic spurs, vascular anomalies and tumors. The technique for localizing the level of the lesion consists in exploring the muscles which derive their innervation above, at and below the suspected level. The muscles innervated by the involved nerve root will show denervation fibrillation potentials on electromyographic examination. This examination is not conclusive from the negative aspect until at least two to three weeks have elapsed since the onset of symptoms. Like many diagnostic

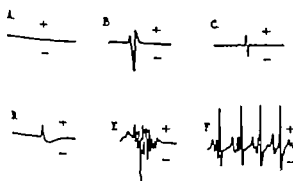


Figure 4 Electromyographic wave forms. A, Normal muscle at rest showing electromyographic silence. B, Normal motor unit potential produced on voluntary effort. C, Spontaneous denervation fibrillation potential from a denervated muscle (lower motor neuron disease). D, Spontaneous denervation positive potential from a denervated muscle (lower motor neuron disease). E, Complex or highly polyphasic motor unit potentials during voluntary effort from a recently re-innervated muscle. F, High frequency potentials from a muscle of a patient having myotonia.

procedures, electromyography must be correlated with the complete clinical picture.

When a dual beam cathode-ray tube is used in the electromyograph, action potentials of reciprocal innervation of the agonist and antagonist muscles can be studied simultaneously. This can be of definite aid in the exact study of kinesiology. In difficult problems of neuromuscular re-education, the patient can understand and co-operate better when he is able to see or hear the action potentials produced while attempting to contract his muscles. The degree of muscle relaxation can also be established.

With the aid of the electromyograph, conduction velocity of a nerve can be measured, augmenting the neurophysiologic studies helpful in the diagnosis and understanding of neuromuscular disorders.

Electrical Skin Resistance Measurements. To further aid in the diagnosis of certain neurologic conditions, electrical skin resistance measurements can be of practical aid. The resistance of the body to small amounts of direct current is almost entirely by the skin. Skin resistance, which depends upon the sympathetic innervation, decreases with sweat gland activity and increases when the sweat glands are inactive; thus denervation increases skin resistance.

Normally, in areas of the body where there is an abundant supply of sweat glands as in the oral region of the face, in the palms of the hands, soles of the feet and the axillae, the electrical resistance is low. In other areas and in scar tissue the electrical resistance is high. In testing electrical skin resistance the resistance of the skin to a small amount of direct current (maximum 50 microamperes) is measured. The deep tissues of the body are good conductors while the skin is a poor conductor of electricity. The surface of the skin supplied by the sympathetic fibers and the sensory fibers is almost identical.

The instrument for measuring skin resistance consists essentially of a microammeter (0 to 100 microamperes) dry cell batteries the necessary resistances and switches and two electrodes. One electrode is attached to the lobe of the ear while the other which is the exploring one, is moved over the skin from high to low resistance, thereby mapping the area of sympathetic loss. There is usually a rather sharp line of demarcation between denervated and normal areas if the lesion is distal to the point where the sympathetics join the nerve. In conditions in which the nerve injury is proximal to the point where

the sympathetic fibers join the nerve, as following posterior rhizotomy there will be no change in the electrical skin resistance even though there is loss of sensation. For example in a herniated nucleus pulposus between L5 and S1 there may be sensory changes corresponding to the root distribution with no change in electrical skin resistance. Further a lesion of the sciatic nerve in the thigh would result in sensory loss over the nerve distribution together with a marked increase in electrical skin resistance over the corresponding area.

SWEATING PATTERN TESTS

The skin area with loss of sweating (anhidrosis) corresponds very closely to the area of sensory loss which accompanies peripheral nerve injuries, since the skin area supplied by the sympathetic nerve fibers corresponds in the main to the area supplied by the homologous sensory fibers. Sweating pattern tests should not be substituted for careful sensory examinations.

Sweating pattern alterations can be demonstrated by colorimetric changes using any of the following techniques.

Starch Iodine Test. The area to be tested is first painted with aqueous iodine solution and after drying is uniformly dusted with

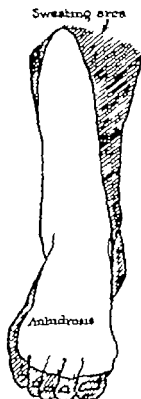


Figure 5 Sweating pattern after severe injury to the common peroneal nerve of the right leg.

powdered starch. Whenever sweating occurs a blue-black color will result; the dry areas remain white.

Cobalt (Chloride Test) A saturated cobalt chloride solution in 95 per cent alcohol is painted over the skin area to be studied. The area where sweating occurs will change from blue to red.

Quinizarin Dye Test The area to be tested is uniformly covered with the powdered reddish-gray dye. Sweating will change the color to a deep purple, thereby outlining the pattern.

In all of the three tests, the patient is subjected to dry heat or placed in a warm room in order to induce sweating. The sweating pattern areas can be photographed or traced for permanent records to aid in evaluating any recovery. Figure 5 shows the sweating pattern following a severe shell fragment injury to the common peroneal nerve of the right leg.

HEAT THERAPY (THERMOTHERAPY)

The value of heat, which is frequently used with other measures in the treatment of disabilities, has been well established. When indicated and judiciously used heat is extremely valuable for both local and systemic effects, such as improving blood and lymph circulation, in relieving pain, for its beneficial effects in inflammatory conditions and for its relaxing effect. Mild heat applied to short-circuited structures for approximately thirty minutes prior to and during the stretching of contractures will greatly facilitate this procedure.

Intense heat even for short periods is definitely contraindicated and dangerous. Heat in any form should be used with extreme caution, or not at all over areas with impaired sensation or over extremities with inadequate circulation as in peripheral vascular disease. Heat is further contraindicated over malignant areas, in hemorrhagic diseases and usually in very acute traumatic lesions.

The different varieties of apparatus for heating tissues have been classified into three main types: external heat applicators, radiant heat generators and diathermy apparatus.

External Heat Applicators. With these types of applicators, heat is applied to the body by means of hot water bottles, hot water packs, electric pads, chemical pads, paraffin baths, hydrotherapeutic apparatus (warm baths, whirlpool baths, Hubbard tanks, contrast baths), cabinets and other

devices wherein heated air is used. These provide a simple but superficial type of heat.

For the paraffin bath a mixture of seven parts of paraffin and one part of paraffin oil is heated to approximately 120° F and into this the hand or foot is immersed for thirty to forty-five minutes. When a shoulder neck or back is to be treated the paraffin can be painted on the part with a brush. The paraffin coating can be kept in place for several hours, thereby maintaining hyperemia.

Hydrotherapy is extremely valuable for heat application and in addition, because of the buoyant effect of water, the patient can actively exercise the extremity even with minimal muscular strength, thereby enhancing function. In the presence of ulcers and wounds, a bland liquid soap or mild detergent should be slowly dripped into or added directly to the continually changing water for both cleansing and bacteriostatic effects (Fig. 6). Following hydrotherapy, the area should be carefully dried with warm air by means of the ordinary hair dryer. This is followed by local ultraviolet radiation to the ulcer or wound.

Contrast baths, whereby the extremity is alternately immersed in warm (103° F) and cold (65° F) water for short periods, may aid circulation and decrease pain.

With the exception of artificial fever therapy and other situations in which the entire body is exposed to heat, treatments should

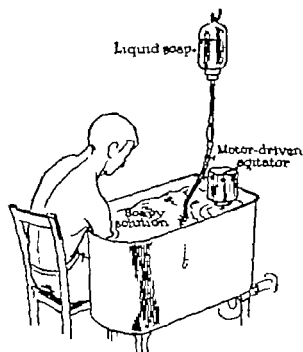


Figure 6 Method for producing soapy solution in the whirlpool tank when hydrotherapy is given in the presence of open wounds.

usually be given for prolonged periods several times each day using very mild heat.

Radiant Heat Generators. These types of generators permit the application of dry heat without direct contact between the source of heat and the body and produce tissue heat somewhat deeper than that produced by external heat applicators. Radiant heat generators consist of electric incandescent lamps (tungsten or carbon filaments) or resistance coils frequently mounted in a parabolic reflector for local application. When the spectral band of the radiation being generated is invisible, the device is known as an infrared generator. The heating unit can also be mounted in a frame or hood and such generators are known as light cradles or bakers.

Diathermy Apparatus. By this method, known as medical diathermy the heating effect results from the resistance of the tissues of the body to the passage of high-frequency currents. The frequencies of the currents used are sufficiently high so that nerves and muscles are not stimulated. The intensity of current is below that which would destroy or impair tissue function. Diathermy is applied locally by two methods *short wave diathermy with an electric field using air space or insulated electrodes or cuffs or a cable or drum operating at a frequency of 27.12 megacycles (wave length of 11.6 meters)* and *microwaves using a reflector type of applicator the frequency being 2400 to 2500 megacycles (wave length of approximately 12 cm)*. Diathermy produces the deepest heat penetration of tissue.

COLD THERAPY AND HYPOTHERMIA

Prompt application of cold in acute strains, sprains and fractures, for perhaps the first twelve to twenty four hours after injury is usually indicated as it not only diminishes pain but also hemorrhage and exudation, consequently there is less swelling. The lowering of body temperatures to about 60° F has been reported to relieve pain in generalized metastatic carcinoma. Cold has also been used locally over an extremity to relieve pain and to diminish circulation and metabolism following an embolism when the patient's condition precludes immediate surgery. It further aids in controlling toxemia and pain in extensive gangrene of an extremity while awaiting the opportune time and site for amputation.

Generalized hypothermia (lowering of body temperature) which results in a slowing of all physiologic processes is used in con-

junction with some cardiovascular surgical procedures, blood vessel grafting and neuro-surgical operations.

THERAPEUTIC EXERCISE

Therapeutic exercises are an extremely important phase of physical medicine and rehabilitation and, if properly prescribed and administered, are rarely contraindicated. Exercises should be specifically prescribed and instituted only by those having a thorough knowledge of functional anatomy the existing pathologic conditions and the physiologic effects of the exercises. Movement is a physiologic procedure while prolonged or injudicious rest is unphysiologic and debilitating. Exercise aids in improving and maintaining proper functioning of the cardiovascular respiratory muscular and nervous systems. Prolonged bed rest often results in a negative nitrogen balance and other metabolic changes associated with significant loss of calcium, phosphorus and potassium. Further muscle strength and work capacity as well as the ability to perform speed and coordination tests, are diminished, while patient irritability is increased.

In the care of the sick, a scientific balance between activity and rest must be established. No fixed rules can be instituted, the criteria being the general over all conditions and the specific exercise needs of the patient. Preliminary mild warming of the body or the parts to be exercised, may enhance activity.

Exercises, especially those associated with posture training, correcting faulty body mechanics and ambulation are best performed in front of a full length mirror.

There are four types of exercises active exercise active assistive exercise, active resistive exercise and passive exercise.

Active Exercise. Active exercise is that produced by the patient's voluntary contraction of muscles without any external aid, is often referred to as "free exercise" and should be instituted whenever possible as it increases blood flow and muscle strength. All types of exercise should be performed slowly and rhythmically without any jerky movements through as full a range as possible. This is especially true when stretching contractures. Exercises should never be performed to the point of exhaustion.

In instances of marked muscular weakness, it may be necessary to position the part to be exercised so as to decrease the effect of gravity so the patient can institute his own active movement. This can also be accom-

plished by supporting the part on a padded board or on a board which moves freely on ball bearing casters or by means of suspension slings. The effect of gravity can also be utilized to assist movement. If active movement is still impossible to accomplish assistance to the movement must be given; this is designated as active assistive exercise.

Isometric muscle contraction, sometimes designated as muscle setting or static exercise, is accomplished without any change in muscle length and is employed when the joint cannot be moved because of a cast, splint or brace. Vigorous isometric contractions will help to prevent or delay atrophy and maintain circulation. Movement in an involved extremity can be frequently enhanced by the simultaneous active resistive exercises of the opposite extremity.

Exercises for correcting and improving faulty posture and body mechanics, as well as for enhancing respiratory movements, are frequently indicated. Parallel bars have proved to be indispensable in training the disabled person, such as the hemiplegic, paraplegic, amputee, and arthritic, to stand, balance and ambulate. In addition, they are extremely safe and effective.

Scientific relaxation exercises, both local and general, are frequently an important phase of any activity program to aid in improving both function and general well-being.

Active Assistive Exercise. Active assistive exercise is the patient's voluntary movement aided by external assistance, as from the

physician or therapist or through the use of pulleys and weights. This type of exercise is often employed when the patient's muscular strength is insufficient to satisfactorily move the part and also to increase range of joint motion when limitation may be due to muscle shortening, contractures, adhesions and other joint change in such conditions as rheumatic disease, neurologic disorders and orthopedic conditions.

In the DeLorme technique of progressive load assistive exercises, the load assists the movement of the part of the body with some modifications. The minimum amount of load assistance the muscles need to perform ten repetitions (ten repetitions minimum load) is determined. With the first ten repetitions use an assistive load equal to twice the ten repetitions minimum, with the second ten repetitions use one and one half times the ten repetitions minimum load, and with the third ten repetitions use ten repetitions minimum load. Figure 7a shows load assistive exercises to develop the elbow flexor muscles.

Active Resistive Exercise. Active resistive exercise is used to aid further in the development of muscle strength by giving resistance to the movement. To produce maximum hypertrophy and strength of the muscle, progressive resistive exercise is initiated. Muscles contracting repeatedly with relatively few repetitions against heavy resistance will increase in size and strength, in contrast to a muscle contracting against light resistance for numerous repetitions which will increase its endurance with very little change in strength. Usually it is desirable to first improve strength and later endurance.

DeLorme's technique of progressive load resistive exercise (the load resists the movement of the part of the body) is in general, as follows: Determine the maximum load that can be lifted for ten repetitions (ten repetitions maximum). Three sets of exercises, each set consisting of ten repetitions, are performed once daily four to five times each week. All resistances are based on ten repetitions maximum load. The first ten repetitions are done with one-half of the ten repetitions maximum load, the second ten repetitions with three-fourths of this load and the last ten with ten repetitions maximum load. Figure 7b shows load resistive exercises to develop the elbow flexor muscles. With the "Oxford technique," the DeLorme procedure is modified so that the load is decreased with each ten repetitions maximum.

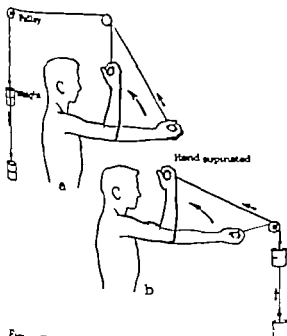


Figure 7 Elbow flexor exercises. a. Load assistive; b. Load resistive.

There appears to be evidence that a lesser number of repetitions against maximum resistance is sufficient to secure maximum strength. This is certainly true after maximum strength has been reached, for in order to maintain maximum it is only necessary to secure one or two repetitions with maximum resistance three to four times each week. However it is important that during exercise the part be moved slowly and rhythmically through the maximum possible range and that each contraction be held for five seconds before relaxing.

Active resistive exercise is often employed to stretch a shortened agonist muscle when opposed by a weakened antagonist. For example, the flexor muscles of the elbow may be shortened in the presence of weak elbow extensors. Active resistive exercises of the extensor group will not only aid in stretching the shortened flexors but will strengthen the weakened extensors.

Passive Exercise. Passive exercise sometimes referred to as relaxed movement, is done entirely by the physician or therapist or through the application of external force. The part being exercised must be properly supported and relaxed. The main therapeutic value in passive exercise is for the maintenance of range of joint motion by preventing adhesions, contractures and joint fixation. For example, in peripheral nerve injury with paralysis, normal joint motion is maintained through passive exercise while nerve regeneration is taking place. This is of tremendous importance for without this type of movement, fixed joints with loss of function will result even though the nerve eventually may regenerate. In contrast to active exercise, passive exercise produces little change in blood flow or muscle strength.

Portable Pulley Apparatus. To aid the patient in improving the function of the upper extremity and shoulder a portable overhead pulley apparatus is helpful (Fig. 8). The horizontal member is notched so that pulley positions can be adjusted to secure the most favorable direction of pull. The baseboard, which has metal glides attached to the undersurface for ease in moving, is large so that it will hold a wheel chair, ordinary chair or stool, giving stability to the apparatus. This exercise arrangement permits the patient to institute all forms of exercises in functional impairment of upper extremities as seen in neurologic conditions, various types of rheumatic disease and orthopedic conditions. If the patient cannot grasp the hand grip at the end of the rope,

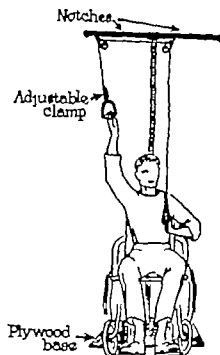


Figure 8 A portable overhead pulley apparatus with adjustable pulley positions being used by a patient with right hemiparesis to improve function of the right upper extremity.

he wears a glove which is then strapped to hold the fingers in position on the grip.

Neuromuscular Re-education. This is another useful technique to enhance function. The patient repeatedly attempts to contract muscles, thereby aiding in the establishment of voluntary movement patterns for purposeful movements. These are at first simple in pattern and gradually progress to more complex ones. For example, following a muscle transplant the individual through this type of training may learn to use the muscle as an extensor instead of a flexor as it was formerly used. Frequently by producing a contraction of a muscle by electrical stimulation at the same moment that the individual attempts to contract the muscle voluntarily, neuromuscular re-education is facilitated. The use of tonic reflexes to facilitate voluntary movement of an involved extremity in an upper motor neuron lesion should be tried.

MASSAGE

Massage with its various techniques is essentially manipulation of the body tissues, usually with the hands, and at times by means of a mechanical vibrator. Even though this technique has been used since ancient times, its scientific value for increasing circulation as compared to heat, exercise, electrical stimulation and physical means

is rather limited. Its psychologic effect through the "living in of hands" may in some instances have a definite therapeutic value. There is frequently a relaxing or sedative effect from mild slow superficial massage or mild vibration as accomplished with a mechanical vibrator.

Circulation in an extremity is enhanced to a much greater degree when active exercise of a part can be instituted than when massage is used. In patients with flaccid paralysis of the extremities it is necessary to employ a vigorous forceful deep kneading massage in order to produce a significant increase in blood flow. This is a traumatic procedure and may result in muscle hemorrhage and eventually fibrosis. Under these circumstances mild heat, passive exercise and electrical stimulation of the paralyzed muscles would not only enhance the circulation and retard the development of contractures but also maintain passive joint range of motion.

Massage should not be used in skin diseases, in the presence of benign or malignant growths, in acute phlebitis or thrombosis over inflamed joints or in any inflammatory condition of the skin or underlying structures.

There are several techniques usually employed in massage namely superficial and deep stroking or effleurage, kneading or compression massage also known as petrissage, friction massage and percussion or tapotement massage.

Superficial and Deep Stroking. Superficial stroking is frequently used for its sedative effect on the patient. It consists of slow, long, smooth, rhythmic movements with light pressure, usually in a centripetal manner. The area to be massaged is lubricated with talcum powder, mineral oil, cocoa butter or lanolin. Deep stroking is similar except that heavy pressure is used in the movements of the hands. Superficial or moderately deep stroking carefully applied may be indicated in patients having painful conditions such as rheumatic diseases, orthopedic conditions and certain neurologic disorders.

Kneading, or Compression Massage. Kneading, or compression massage is accomplished by rolling or squeezing the muscles between the hand and the underlying structures or between both hands, the movements being in a rolling or circular movement, following the pattern of the large muscle groups and primarily in a centripetal direction. Frequently kneading and deep stroking are used simultaneously.

Friction Massage. Friction massage is

done with or between the tips of the fingers in a small circular or lifting movement without the use of any lubricant on the skin. Contrary to the stroking and kneading type of massage there is no movement between the fingers and the skin when friction massage is used. The skin is caused to move over the underlying tissues thereby aiding in loosening scars and adhesions.

Percussion Massage. Percussion massage which is rarely used consists of tapping, cupping, hacking or beating movements done with the fingers, palms or edges of the hands.

ASSISTIVE AND SELF HELP DEVICES

In order for some disabled individuals to perform self-care activities assistive and self help devices, splints, braces and crutches are needed. There are numerous excellent devices that serve many useful purposes. These help in protecting weakened muscles in preventing and correcting contractures and deformities, in holding the part of the body in a better functioning position and permitting voluntary movement of the nonparalyzed muscles while aiding in the movement of the paralyzed muscles. For example in a radial nerve injury the assistive dynamic splint (Fig. 9) holds the wrist and the metacarpophalangeal joints in a functional position by means of elastic or springs and yet permits the patient to flex the fingers and wrist voluntarily in order to grasp. Upon relaxation of the flexor muscles the device will bring the fingers and wrist back to the neutral or extended position ready for the next grasping movement.

In conditions in which for all practical purposes there are no voluntary movements in the hands following a cervical cord or brain lesion or in the chronic ankylosing type of arthritis, a simple assistive splint can be easily fabricated of plastic or metal that is placed over the palm of the hand and held

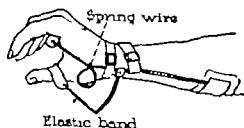


Figure 9 Dynamic splint for radial nerve paralysis. The spring wire holds the wrist and metacarpophalangeal joints in extension while the elastic band maintains the thumb in extension and abduction. This type of splint permits the patient to perform numerous hand activities.

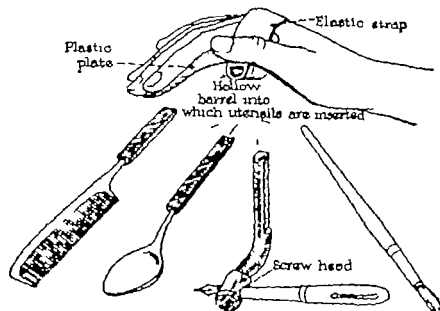


Figure 10 Assistive device for functionless hand. A plastic plate is held in the palm by an elastic strap. Numerous detachable and interchangeable utensils can be inserted into the hollow barrel enabling the patient to perform many self-care activities.

in place with an elastic or leather strap (Fig 10). This basic device can be fitted with a detachable fork, spoon, pencil, brush, safety razor and numerous other daily used items. If the individual cannot grasp completely in order to hold objects, then a simple assistive hand device can be fabricated, consisting of a large hollow plastic handle with detachable interchangeable utensils such as a comb, brush, fork, spoon, toothbrush, razor and small tools. The size of the handle will be governed by the patient's function and can be decreased as the patient's grasp improves. A combination knife-fork eating utensil is available for those who must eat with one hand, such as hemiplegics, certain amputees and others. This device permits the person to cut and then bring the food to his mouth using only one hand.

Recently hand moving devices have been developed which are operated by wrist motion, shoulder-controlled cables or motors. Simple assistive devices are easily designed to enable the seriously disabled person to type, use a telephone, walk and perform many coordinated activities without which he would be quite helpless and dependent upon others for assistance.

Training in the correct use of properly fitting crutches (full-length and forearm type), canes, braces and wheel chairs further aids in the performance of many self-care and daily living activities. Braces help in supporting body weight, in the prevention and correction of contractures and deformal-

ties and in controlling abnormal movements. Since the majority of persons who wear braces also require crutches in order to ambulate, it is extremely necessary that proper gait training be instituted.

Gait training, either within or outside the parallel bars, is best accomplished in front of a full length mirror. In this way the patient can observe his locomotion and posture and thereby attempt to correct any faulty body mechanics.

Crutch Ambulation. For successful crutch ambulation the following factors must be carefully considered:

1. The development of function of the muscles necessary for ambulating with crutches.

2. Selection of the proper type and measurements of the crutches, such as the wooden full length crutches (Figure 11a) or the metal forearm (Canadian) crutches (Figure 11b).

3. The selection and training in the best suited crutch gait. Much depends on the disability and the general over-all medical condition of the patient and one must be realistic in selecting those who should be taught crutch ambulation. The energy expenditure may be beyond the patient's physical and mental abilities and reserves, for with some patients the weight of the body must be supported by the shoulder girdle and upper extremities. The flexors of the shoulders move the crutches forward, the extensors of the elbows and wrists permit

raising of the body from the floor the shoulder girdle depressors and downward rotators support the body when it is raised from the floor and the flexors of the fingers grasp the hand grip on the crutch.

The shoulder portion of the crutch should be covered with a resilient axilla pad while the end has a large 1½ inch diameter suction tip to prevent slipping.

A simple method for measuring for long crutches is to have the patient supine and secure the length from the anterior fold of the axilla to the foot and add 2 inches. The hand grip is so placed that the elbows are flexed at 30 degrees.

Prior to the use of crutches the patient should master the indicated gait in parallel bars. As part of crutch ambulation training, the patient must first be taught to balance and shift his weight, preferably while standing against a wall. The body weight is taken mainly on the hands, the elbows being slightly flexed and the shoulders down. The crutches must clear the axillae. Ambulation should be taught on level ground, inclines, curbs and stairs when feasible. In those patients in whom non weight bearing of one extremity is indicated the shoe on the opposite extremity should be built up approximately 1 to 1½ inches.

Types of crutch gaits and their sequences

1. Four Point Alternate Crutch Gait right crutch, left foot left crutch and right foot
2. Two-Point Alternate Crutch Gait right

crutch and left foot simultaneously then left crutch and right foot simultaneously. The gait is just a speeding up of the four point alternate gait.

3. Three Point Crutch Gait both crutches and the weaker lower extremity simultaneously then the stronger or normal lower extremity. This gait is for those having one lower extremity that cannot take full weight bearing while the other lower extremity can support full body weight.

4. Tripod Crutch Gaits

a. Tripod Alternate Gait right crutch, left crutch then drag body forward

b. Tripod Simultaneous Gait both crutches simultaneously then drag body forward

5. Swinging Crutch Gaits

a. Swing to Gait both crutches simultaneously then left and swing body to crutches

b. Swing Through Gait both crutches simultaneously then lift and swing the body beyond the crutches

The metal forearm crutches are for those patients who have developed sufficient strength, balance and coordination for safe ambulation. However it is well even for these individuals to first master ambulation with full length crutches.

It should be stressed that a very careful evaluation must be made of the patient's total disability and his general medical condition in order to (1) decide whether the patient should be taught crutch ambulation, (2) select the proper type of crutches and fit them properly, and (3) provide sufficient training in the properly selected gait to meet the specific needs of the patient. There will be certain disabled patients for whom crutch ambulation would be unrealistic and contraindicated.

Wheel Chairs. For patients with severe disabilities wheel chairs can be secured with special features which will enable the individual to perform many additional self-care activities. In general wheel chairs should be of the folding type with the large driving wheels placed posteriorly and with arm and adjustable footrests and a thick foam rubber cushion seat. Hand brakes are essential for stability especially for getting in and out of the chair. When indicated, a zipper back is indispensable for entering and leaving the wheel chair from the rear as in getting in and out of bed and on and off the toilet. For those wheel chair patients who have the use of only one arm, a special "one-arm" drive is available. There are other additional special

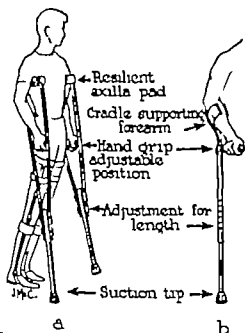


Figure 11 Crutch ambulation. a, Full length wooden adjustable crutches having resilient axilla pads, suction tips and adjustable position for hand grip. b, Metal forearm (Canadian) crutch.

devices and attachments such as a semireclining back, commode seat, detachable arms and adjustable leg rests, which should be considered to fill the specific needs of the wheel chair patient. A battery-operated electric wheel chair is available for those severely disabled persons who are unable to use the hand powered chair.

All disabled persons should be carefully and selectively fitted with assistive devices for specific needs so they can perform activities essential for maximum rehabilitation.

It is imperative that neuromuscular reeducation and therapeutic exercises be continued while the patient uses an assistive device. If voluntary function is eventually accomplished, these various devices can be modified or entirely eliminated.

ELECTRICAL STIMULATION

Electrical stimulation to produce contraction of a denervated muscle has therapeutic value. A frequent and difficult problem encountered in denervated muscle is that of atrophy and fibrosis. Electrical stimulation of the denervated muscle is used to help retard atrophy so that, in the event reinnervation occurs, there will be better functional recovery. Although this subject is still controversial, most of the recent evidence indicates that atrophy and loss of muscle weight can be retarded by properly instituted electrical stimulation. This form of therapy must be started as soon after denervation as possible. The stimulation should be capable of producing vigorous muscular contractions starting with the muscle in a stretched position. During the movement of the part, resistance is applied by the operator's hand or by means of elastic, springs or weights. Stimulation may be given for a period of fifteen minutes twice each day. However, it appears that best results are obtained by shorter periods given three to four times each day. Vigorous contraction also has a favorable influence upon the nutrition of the muscle by increasing circulation.

Interrupted direct current or an alternating current of proper intensity with a carrier frequency of 10 to 25 cycles per second, amplitude modulated so that the muscle contracts approximately fifteen to twenty times per minute, can be used. The ideal type of stimulating current for any given situation is not thoroughly understood. Since the threshold of reaction of the muscle varies with the type of current used for stimulation, it is well to select the current frequency and

wave form that produce the most satisfactory contraction. Electrical stimulation of denervated muscles is at best a poor substitute for the physiologic nerve impulses in a normally innervated muscle. Electrical stimulation is sometimes used in the flaccid stage of hemiplegia to aid in preventing and correcting contractures. In upper motor neuron lesions, using the electrical current to maintain the spastic muscle in a firm tetanic contraction for twenty to thirty minutes twice each day may be of help in decreasing the muscle spasticity and the frequency and intensity of the spasms. This no doubt produces fatigue of the muscle which is a factor in decreasing the spasticity. It has also been recommended as an aid in preventing and "freeing" adhesions and in removing traumatic exudates following sprains and contusions. Electrical stimulation is helpful as an aid in teaching the patient neuromuscular re-education.

There are two principal methods of electrical application that can be used for electrically stimulating muscles to produce contraction. The first method is to use an active and a dispersive electrode. The active electrode is relatively small, being approximately 1 to 4 square inches in area, and is placed over the point of maximum electrical sensitivity of the muscle to be stimulated. This point is designated as the motor point. The dispersive electrode is large, being many times the size of the active electrode and is placed on the body at a distance from the muscle being stimulated.

The second method utilizes two small electrodes of equal size, which are placed over the ends of the belly of the muscle to be stimulated. With this technique, there is less spread of the electrical field to the adjacent muscles and, at times, it may be more satisfactory for stimulating smaller muscles.

ULTRAVIOLET RADIATION

The various sources of ultraviolet energy are the sun, which is a natural source, and artificial sources which are the carbon arc lamp, the low voltage (hot) mercury vapor quartz lamp including the Kromayer lamp, the high-voltage (cold) mercury vapor quartz lamp and the sunlamp.

Ultraviolet is a form of invisible radiant energy having a wavelength of 10 to 3900 Å.U. (Ångström units). The therapeutic dosage of ultraviolet radiation is based on the reaction of the individual to the radiation from a specific lamp and is determined by having small areas of 1" x 1" unit.

skin, usually over the forearm or back exposed for varying times with the ultraviolet lamp placed at a fixed distance usually 30 inches from the skin surface. The time of exposure producing the slightest erythema which becomes visible in from four to twelve hours is called the M.E.D. (minimal erythema dose). This M.E.D. for a specific distance is used as a guide in prescribing ultraviolet radiation. This test however should be repeated every few months as the output of ultraviolet generators decreases with use.

Local and general ultraviolet radiation has been used in the treatment of many conditions. However its greatest value appears to be in certain skin disorders. Because of its mild bactericidal effect and beneficial stimulation of granulation tissue ultraviolet is also of value in the healing of decubitus and indolent ulcers and wounds.

If hydrotherapy is used in the treatment of this type of lesion the area, after hydrotherapy, should be gently dried by using an electric hair dryer in order to counteract the macerating effect of the warm water and is then exposed to local ultraviolet radiation. In order to secure and record the exact size and shape of these lesions a simple plastic tracing device is used.

There is usually a so-called general tonic effect from extremely mild full body exposures. Caution should always be observed to avoid excessive sunburn the possibility of flare-up of certain skin diseases and various untoward reactions in light sensitive or sensitive individuals and in those who are using drugs or having ointments applied to the skin that is being exposed to the radiation. Ultraviolet radiation of blood known as the Knott technique, has been used for many years in numerous conditions including bac-

teremia wound infections and bronchial asthma without as yet acceptable scientific clinical proof of its worth.

Ultraviolet lamps can be used as diagnostic aids. Many organic substances including certain pathogenic fungi fluoresce under ultraviolet radiation. Ringworm infections of the skin can be detected by the fluorescence seen when a Wood's filter or its equivalent is used to screen out most of the visible radiation.

ULTRASOUND THERAPY

Ultrasound therapy in recent years has been used in the treatment of many disorders and consists of vibrations, usually at a frequency of 1 000 000 cycles per second. These waves are produced by a quartz plate which is caused to vibrate synchronously with a high frequency electric field of 1 000 000 cycles per second. These ultrasound vibrations are emitted only from the face of the treatment head which contains a quartz plate. The energy density at the head can be controlled up to approximately 4 watts per sq cm in some machines. The face of the treatment head averages approximately 5 sq cm.

Ultrasound energy transmission from the treatment head into the body tissues can be accomplished by three kinds of coupling, namely direct contact, underwater contact and with water cushions. Treatment by means of direct contact of the head to the skin is the simplest (Fig. 12a). Inasmuch as air forms an almost impenetrable obstruction to the passage of ultrasound radiation, mineral oil spread over the skin is used as the coupling medium between the treatment head and the skin. During treatment, which may be given for five to eight minutes, depending on the condition being treated and the wattage used, the treatment head is con-

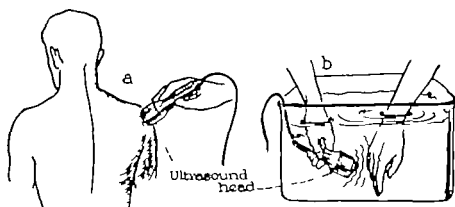


Figure 12. Ultrasound therapy a, Ultrasound therapy being given to the shoulder by the direct contact method. Mineral oil is used as the coupling medium. b, Ultrasound therapy being given to the hand by the underwater contact method. The ultrasound head is kept at a short distance from the hand.

tinually and slowly moved over the involved area.

When for example, small areas or joints in the hands and feet are to be treated the underwater method is preferred (Fig 12b). With this technique the part of the body such as a hand is immersed in a vessel filled with degassed water and the ultrasound treatment head is placed at a distance of 1 to 2 inches from the body surface.

In conditions in which it is difficult or impractical to use the direct contact or underwater technique, such as in the region of the head axilla or perineum, the water cushion method is used. This cushion consists of a thin rubber bag completely filled with degassed water which is placed between the treatment head and the skin surface.

Ultrasound therapy is also given over both the local area of involvement and the spinal nerve roots which supply the involved area. For example, in treatment of rheumatoid arthritis of the hand, ultrasound therapy by the underwater contact method may be given for approximately five minutes at $\frac{1}{2}$ to 1 watt per sq. cm. and approximately the same dosage may be given over the seventh and eighth cervical and first thoracic spinal nerve roots just lateral to the vertebral spinous processes. The total dosage in any condition is varied according to the specific disorder being treated.

When ultrasound energy is directed into the tissues, there are in all probability a number of physiologic physical and biologic changes that occur. Many workers have reported beneficial results from the use of ultrasound therapy in various types of rheumatic diseases (rheumatoid arthritis degenerative joint disease bursitis fibrositis) inflammatory processes (lymphangitis carbuncles) relief of pain in neuromas and scars and numerous other conditions. Further research both basic and clinical is still in progress in order to evaluate the indications, results and contraindications to ultrasound therapy.

TRACTION

Cervical Traction. Patients with cervical lesions such as fractures, fracture-dislocations, herniated nucleus pulposus and cervical arthritis, who have pain in the neck region with or without radicular radiation of pain to the shoulders and arms are frequently benefited by properly instituted cervical traction.

Traction is secured by using a head halter while heat is applied to the neck and poste-

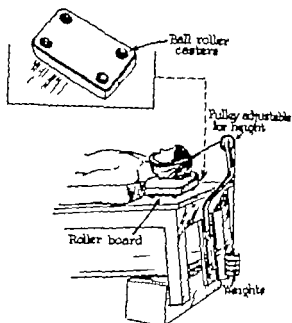


Figure 13 Cervical traction. The patient's head is supported on a padded roller board. The height of the pulley is adjusted for the proper angle of traction. Heat is applied to the neck and shoulders during traction.

rior shoulder muscles by means of an electric pad or warm compresses. The head is supported on a well padded board having four ball-roller casters attached to the under surface (Fig 13). This board, when placed on a hard surface, gives almost frictionless movement so that the force of traction on the neck is practically equal to the weight applied. The best results are usually obtained by gradually increasing the weights until a maximum is reached that can be comfortably tolerated by the patient while in a lying position. This amount of pull is maintained for long periods during which the patient, at periodic intervals, slowly rotates his head as far as possible to the right and then to the left. This procedure tends to increase the range of rotation of the head, which is frequently limited. At the end of this period, the weights are gradually decreased and traction terminated. It is important to adjust the height of the pulley in order to secure the proper angle of pull.

In those patients in whom true radiculitis is present due to narrowing of the vertebral foramen the best results from cervical traction occur when the neck is in partial flexion. In this position there is greater widening of the foramen than when the head is in a straight line with the long axis of the body or when it is extended. The head end of the table is elevated approximately 7 inches to prevent the patient from lying in the direc-

tion of the traction. Prolonged traction with a moderate force while the patient is in a horizontal position is preferred to a strong pull for a relatively few minutes. The exact direction of traction and the amount of force applied should be individualized for the specific conditions under consideration.

Lumbar Pelvic Traction. In the conservative management of low back pain with or without evidence of herniated nucleus pulposus or sciatic pain, lumbar pelvic traction may be used. It is important, however, to use proper apparatus and technique in order to achieve best results.

A simple but effective arrangement for lumbar-pelvic traction is shown in Figure 14. Traction is applied to the lumbar spine and muscles by means of a properly fitted pelvic corset or belt, to which is attached the necessary straps and spreader bar and from which an adjustable weight is suspended over a pulley. In order to actually secure a known effective amount of pull on the lumbar region when a certain weight is suspended on the cable passing over the pulley, it is important to reduce to a minimum the resistance of the patient's hips and lower extremities to this pull. This is accomplished by having only the hips and lower extremities resting on a well-padded roller board having six ball-roller casters fastened to the underside; this rolls almost without friction on a hard surface, such as metal, formica or similar ma-

terial. The upper portion of the body above the lumbar area rests on a padded surface which is held on the table by a strap. A mattress split completely across can be used under the patient, one section being placed over the roller board to support the hips and both lower extremities while the other section is placed under the upper half of the body. With this method, the force of traction on the lumbar area is practically equal to the weight suspended over the pulley. The roller board is self-aligning during traction.

It must be remembered that if treatment is applied with the patient lying on a bed or treatment table without the use of a roller board under the hips and lower extremities, a tremendous force is necessary depending on the size of the individual to overcome the weight of the hips and lower extremities (approximately one half of the total body weight) before any pull is actually exerted on the lumbar spine and muscles. Even then the exact amount of pull on these structures is not readily known.

In order to keep the patient's body from sliding in the direction of the traction, the foot end of the table is elevated approximately 7 inches. The height of the pulley should be adjusted so that the direction of the traction is at the proper angle, usually with the patient's pelvis somewhat flexed. As a rule the best results are obtained by gradually increasing the weights during the first

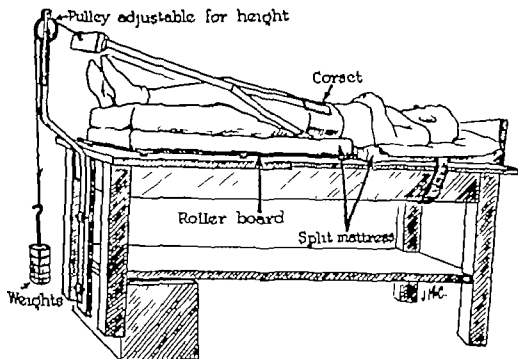


Figure 14. Lumbar pelvic traction. The patient's hips and lower extremities are supported on a padded roller board while the upper portion of the body rests on the upper section of a split mattress or pad. The pulley height is adjusted for proper angle of traction.

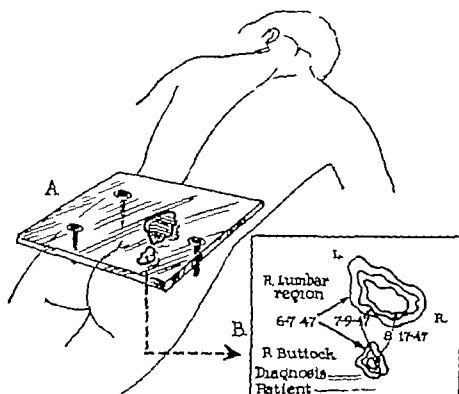


Figure 15 Tracing device for evaluating surface lesions. A Device is shown in position for tracing a decubitus ulcer B, Periodic tracings show changes in contour of the lesion.

thirty minutes until a maximum pull is reached that is comfortable to the patient. This amount of pull is maintained for one hour or longer and then gradually decreased for the final fifteen minutes when the traction is terminated.

During the entire period of traction, in order to secure added comfort and additional therapeutic effects heat is applied to the lumbar area by means of an electric pad or warm compresses. A pillow under the head, and others properly placed to hold the knees and hips partially flexed, results in a more restful position during traction. During the traction treatment, the patient can move his upper and lower extremities and even roll somewhat to the right and to the left in order to avoid the discomfort of lying in a fixed motionless position.

In addition to traction as part of conservative treatment, proper therapeutic exercises should be gradually instituted to correct faulty posture and abnormal body mechanics and to improve muscle tone and strength as well as mobility of the neck and back.

EVALUATION OF SURFACE LESIONS

In order to check the progress of any surface lesion, such as decubitus ulcers, varicose ulcers, wounds of all types and other skin lesions, a simple tracing device can be

used to secure and record the exact size and shape of the lesion. Tracings of the lesion can be made periodically and checked with the previous tracing to note any change in size or contour.

The tracing device consists of a transparent rectangular plastic plate having three adjustable legs (Fig 15). This device is placed over the surface lesion, but direct contact should be avoided. By looking through the plastic plate onto the lesion, and with a skin marking pencil, the operator draws the outline of the lesion (Fig 15A). When the tracing is completed the device is removed from the patient. A standard size sheet of transparent paper is placed over the tracing device and the outline then traced on this paper with pen or pencil. This sheet, together with the patient's name, diagnosis, dates of tracings and other information becomes a permanent part of the patient's record. Periodic tracings of the same lesions are made on the original sheet in different colors for comparison (Fig. 15B).

FRACTURES, DISLOCATIONS, SPRAINS AND STRAINS

Objectives. In conjunction with the indicated medical regimen, the objectives of physical medicine and rehabilitation are to alleviate pain, enhance the healing process,

prevent or correct deformities, secure maximum function possible of the involved portion of the body and maintain the greatest degree of physical fitness, all of which lead to the return of the disabled person to maximum usefulness.

Physical Medicine and Rehabilitation Procedures. *Gold.* During the first twelve to twenty-four hours after injury, the application of cold is indicated to diminish pain and swelling.

Massage. If the injured part is accessible, superficial stroking, progressing to deeper stroking as healing progresses, may be of benefit.

Heat. After the first twenty-four to forty-eight hours, mild heat will frequently relieve pain and aid in the absorption of exudates. Heat should not be applied through a cast, as it is impossible to judge the amount of heat being absorbed by the tissues and the procedure may predispose to burns. Further, any sweating under a cast would tend to macerate the skin.

Ion transfer. Ion transfer by direct current of a 1 per cent procaine hydrochloride solution into painful tissues is in some instances helpful for relieving pain associated with sprains and strains, thereby permitting active exercise of the involved part. The active electrode is saturated with the procaine solution and is connected to the positive terminal of the battery or direct current generator. The dispersive electrode is placed on the body at a distance from the active electrode and is connected to the negative terminal.

Therapeutic exercise. When the injured part is immobilized, isometric muscle contractions should be started. As soon as motion is indicated, gentle active and active resistive exercise should be instituted with gradual transition to progressive resistive exercise to secure maximum range and strength of movement. Active exercise of one extremity will improve circulation and delay atrophy in the contralateral extremity, even though the latter may be immobilized in a cast or splint. In hip arthroplasty, special therapeutic exercises are extremely helpful in restoring function. Conditioning exercises to all of the body should be started early to avoid the ill effects of inactivity and deconditioning.

Occupational therapy, manual arts therapy and educational therapy. These activities help to further improve function, assist the patient to perform self-care activities, and the patient psychologically in his hospitaliza-

tion and serve as an exploratory activity to determine the future type of employability.

NEUROLOGIC DISORDERS

Physical medicine and rehabilitation is indicated and of benefit in most neurologic disorders as an aid in diagnosis, prognosis and treatment. Diagnostic procedures include:

Measurement of range of joint motion and muscle strength.

Sensory and reflex tests.

Electrodiagnostic tests including the use of direct and alternating currents, electrical skin resistance measurements and electromyography, including nerve conduction tests.

Sweating pattern tests.

Performance of self-care and daily living activities.

UPPER MOTOR NEURON DISORDERS

Upper motor neuron lesions may result in a variety of disabilities such as partial or complete hemiplegia, paraplegia and quadriplegia—with or without speech disorders such as aphasia. In many instances motor and sensory disturbances are combined and present a most difficult medical situation, taxing the ingenuity of all concerned.

Objectives. The objectives of treatment are to prevent and treat any deformity promptly, to aid circulation, to develop as much function in the involved extremities as possible, to use any possible assistive devices—including braces, crutches, canes, wheel chairs—leading toward the performance of self-care activities and ambulation. Speech therapy should be judiciously instituted for those with aphasia. The patient should be motivated toward the acceptance of his disability and the treatment program with gainful employment as the goal if feasible. Many neurologic patients, such as those with paraplegia, can be taught to drive safely an automobile equipped with proper operating hand controls.

Physical Medicine and Rehabilitation Procedures. *Proper positioning.* The patient is properly positioned in bed to aid in preventing contractures. The use of pillows, sand bags and simple splints is an extremely important preventive measure. For example, in a patient with hemiplegia, the extremities must be placed in proper position and alignment so as to prevent contractures in the upper extremity resulting in deformities of flexion and adduction of the shoulder and flexion of the elbow, wrist and fingers. In the lower extremity, contractures usually result in deformities of flexion and external rota-

tion of the hip flexion of the knee and plantar flexion and inversion of the foot.

Therapeutic exercises Passive exercises and, in those patients having some voluntary movement, active assistive and active exercise should be instituted to prevent or correct contractures and to secure the maximum possible degree of function in the involved as well as the uninvolved extremities.

Spasticity and spasms are a frequent and difficult problem and, therefore, all exercises, and especially those for stretching, must be done extremely slowly so as to avoid stretch reflexes as much as possible. Pulleys and sling suspension exercises are of benefit. Relaxation procedures, especially for muscles with spasticity, should be started early. General conditioning exercises are also included. Fatigue should be avoided.

Neuromuscular re-education and relaxation In order to secure maximum results these procedures must be taught to the patient and continued by him long after he is discharged from the hospital.

Heat Mild heat, including hydrotherapy is indicated to relieve pain and facilitate stretching of shortened structures. This may also be helpful in some patients to diminish spasticity and spasms. Massage may be of additional value if done slowly and superficially and if it does not elicit spasms.

Self-Care Activities. The performance of these activities with the further aid of occupational therapy, manual arts therapy, and educational therapy is a vital part of the total program. A portable double bilateral assistive sling device (Fig. 16) is extremely valuable when the patient does not have sufficient strength in the upper extremities to bring the hands toward the head in order to eat, shave and brush the teeth, even though the utensils employed may be held in the hands. The adjustable slings are counterbalanced by sufficient weights to assist in moving the extremities. The elbow rests in the posterior sling, which assists shoulder movements; the wrist and forearm rest in the anterior sling, which assists elbow flexion. The device can be constructed with coiled springs instead of cables and counterweights and also be made to be fastened to a chair wheel chair or to the head of the bed. Ambulation should be started whenever realistic, even though for short distances, with or without the aid of braces, crutches or canes. The paraplegic patient may need bilateral long leg braces and crutches, while the hemiplegic patient usually requires a short leg brace to prevent foot-drop and a cane in order to at-

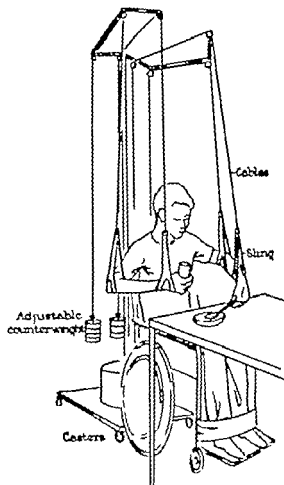


Figure 16. Portable double-bilateral assistive slings. A partial quadriplegic patient feeding himself with the aid of the assistive slings. Similar devices can be attached to the wheel chair or bed and also made with suspension coiled springs instead of the cables and counterweights.

tempt ambulation after sufficient training in the parallel bars. If ambulation is beyond the realm of possibility, then the patient should at least be placed in an upright weight-bearing position for short periods once or twice each day. This can be accomplished by means of bilateral long leg braces or on a tilt board or table. Patients who are unable to stand because of paralysis can be placed in the parallel bars and held securely in a vertical position by means of well-padded straps. One strap is placed behind the feet, one in front of the knees and the other across the buttocks (Fig. 17). If needed, for example, with a quadriplegic patient, an additional strap can be placed in front of the chest for further stability during standing. These techniques are of definite help in preventing decubitus ulcers, genitourinary complications and nutritional deficiencies and for their favorable influence in diminishing the frequency and severity of spasms. It is ex-

tremely important in treating the difficult problems associated with upper motor neuron lesions, that both preventive and definitive physical medicine and rehabilitation procedures be instituted early intensively and for a sufficient time.

Many patients with severe disabilities may require the use of a wheel chair for varying periods during and after their rehabilitation. Other patients will be unable to ambulate and will have to lead a so-called wheel chair life. A great deal of consideration should be given to the exact type of wheel chair a patient is to use for many variations and attachments are available. A complete evaluation should be made of the patient's abilities and a wheel chair secured to meet his needs.

Postural hypotension may be associated with a variant of neurologic lesions and presents a definite problem in rehabilitation. When the patient is placed in a sitting or standing position the blood pressure drops, the pulse becomes weak and very rapid and the individual becomes pale and may even lose consciousness. A tilt table that can be gradually elevated from a horizontal to a vertical position is useful in the management of this condition (Fig. 18). The patient is placed on the table in a horizontal position and held securely by several well padded straps. The table is then elevated approximately 5 or 10 degrees. The blood pressure, pulse and general condition are observed at frequent intervals. The degree of elevation and the time in this position are gradually

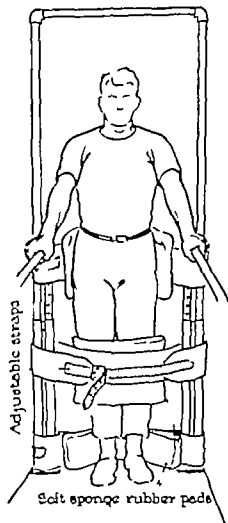


Figure 17. A paraplegic patient standing in the parallel bars prior to the use of long leg braces. Note the three well-padded straps for stabilization. When necessary an additional padded strap can be placed across the front of the chest.

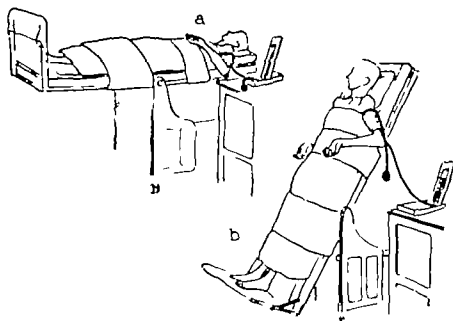


Figure 18. Tilt-table showing quadriplegic patient with postural hypotension. The patient is gradually elevated from a horizontal to a vertical position.

increased until the patient can remain in a vertical position without the development of these untoward effects.

LOWER MOTOR NEURON DISORDERS

Objectives. The aims of treatment are to prevent or correct promptly any deformities to stimulate circulation of the involved part to institute any indicated dynamic splinting so that if reinnervation takes place the extremity will be able to function in response to voluntary nerve stimuli, to improve and maintain the general physical condition, and to aid in the psychologic adjustment of the patient. In those individuals having poliomyelitis with respiratory involvement requiring the use of a mechanical respirator the physical medicine procedures must be accomplished during the period the respirator is in use as well as after its employment has been discontinued.

Physical Medicine and Rehabilitation Procedures. *Heat* To stimulate and maintain circulation of the involved parts, mild heat should be given.

Therapeutic exercise Until active motion is possible, passive exercises to prevent contractures and preserve function of the joints are promptly instituted. Exercise to prevent general deconditioning is imperative.

Electrical stimulation To delay atrophy and aid circulation electrical stimulation of the paralyzed muscles is indicated. The value of electrical stimulation in poliomyelitis is questionable, for during the process of stimulating the paralyzed muscles the current may spread and produce contraction of the unparalyzed antagonist muscles, resulting in a greater degree of the already existing muscular imbalance.

Dynamic splints When indicated, appropriate splints should be worn by the patient to prevent overstretching of the involved muscles. The springs or elastic on the splint will compensate for the lost motion while the patient voluntarily contracts the uninvolved muscles. This procedure frequently permits functional activity on the part of the patient.

Massage Massage must be cautiously applied so as not to traumatize flaccid muscles. Heat, passive exercise and electrical stimulation are more effective in these conditions.

Neuromuscular re-education. This procedure should be started as soon as there is evidence of muscle reinnervation.

Traction. Nerve root lesions in the cervical and lumbar regions due to herniated nucleus pulposus and arthritic changes frequently

produce local as well as radicular pain and limitation in motion of the spine and corresponding extremities. Rest, heat and properly instituted traction are indicated as conservative measures.

In cervical lesions traction can be secured by means of a head halter while heat is applied to the neck and posterior shoulder muscles. The head is supported on a well-padded board having four ball-bearing casters attached to the undersurface.

In lumbar lesions traction can be applied by means of a pelvic corset or belt while heat is given to the back muscles. The pulley and weights are attached to the foot end of the table which is slightly elevated to prevent the patient from sliding in the direction of the pull.

Therapeutic exercises to correct faulty posture and abnormal body mechanics, to increase muscle tone, and to restore mobility of the spine should be gradually started as soon as the acute pain subsides. Proper bed positioning—back or side-lying with the hips and knees flexed—is indicated. Generally flexion movements of the spine should be limited. The patient should be taught the proper way to lift objects, to get into and out of his automobile and into and out of a bath tub, and other activities with a minimum of back flexion. In those patients requiring lamectomy the therapeutic exercises should be continued postoperatively.

AMPUTATIONS

Objectives. In addition to the program being planned to return the amputee to a useful occupation, it is necessary to include the other important objectives such as the psychologic and social adjustment to the loss of one or more extremities. The securing of a stump having maximum function insofar as range of motion, muscle strength and endurance is concerned, the securing and proper fitting of a suitable artificial extremity to meet the specific needs of the amputee, and sufficient training in the use of the artificial extremity to enable the individual to perform a sufficient number and type of activities that would justify its use. The value of the finest fitting artificial extremity is markedly decreased in direct ratio to the lack of function of the stump and the motivation of the amputee.

Physical Medicine and Rehabilitation Procedures. *Proper positioning.* The patient and the stump must be properly positioned to prevent contractures of the stump activating and stabilizing muscles.

Bandaging of the stump To aid in shrinking and shaping the stump proper bandaging should be started at least as soon as the wound has healed.

Therapeutic exercise Probably the most valuable form of therapy for the stump is therapeutic exercise which should be started early in the form of gradual active and active assistive exercise and then proceed on to progressive resistive exercise to secure a stump having maximum function. In elective amputations, therapeutic exercises should be started prior to amputation and promptly resumed postoperatively. Figure 19 shows some of the exercises used to develop function of an above-elbow stump. These exercises are performed bilaterally and as soon as indicated, they should be done with progressive resistance.

Artificial extremities Many types and sizes of both voluntary opening and voluntary closing terminal devices (hands and hooks) each having relative merits depending on the type of work to be accomplished are available for the upper extremity amputee. The hooks are functionally better than the hands which are cosmetically superior. In the voluntary opening type the pull of the control cable opens the terminal device while with the voluntary closing type the pull closes it. An extremely practical voluntary closing hand and interchangeable hook suitable for many amputees is the APRL (Army Prosthetic Research Laboratory) type, which may have the standard above-elbow harness or one of other variety. Most above-elbow amputees desire the dual control harness. The cable when pulled, serves to flex the elbow to the desired position. The

elbow is locked then when the cable is pulled again it activates the hook or hand. When the pull on the cable is completed, the terminal device automatically locks and maintains the grasp force on the object. When the grasp is to be released the next pull of the control cable unlocks the device which then automatically returns to the fully opened position. In below-elbow amputations a biceps cineplasty to operate the terminal device should be considered in the light of the amputee's occupation and his cosmetic requirements. It is important that the biceps muscle be well developed by resistive exercises started preoperatively and resumed postoperatively. The amputee should then be taught to disassociate elbow flexion from biceps contraction which now will be used only for operating the terminal device.

For selected individuals with above-knee amputations the suction-socket type of artificial leg, which eliminates the pelvic belts and shoulder harness used with the conventional type is frequently the most suitable. In putting on the suction-socket leg, a thin piece of stockinet is placed over the stump, the stump is inserted into the socket and then the stockinet is pulled off and out through the opening in the socket for the air valve. After the stump is comfortable the weight is put on the leg and the valve is inserted to seal the socket. The leg is held on the stump by a slight negative pressure, or "suction" created in the closed socket by the weight of the leg as it is lifted from the ground assisted by the adherence of the stump to the walls of the socket. The muscles of the stump control all movements of the artificial leg.

Training in use of artificial extremities. Adequate training is indispensable if the amputee is to achieve a measure of success with his new limbs.

In lower extremity amputations, a pylon (Fig. 20) is usually indicated for training as soon as the wound has healed and further aids in the shaping and shrinking of the stump. The pylon is easily, quickly and inexpensively fabricated and aids the amputee in developing a proper gait, in addition to improving the stump function. This also serves as a therapeutic test in older individuals as to their future ability to use an artificial extremity. The use of parallel bars (Fig. 20) greatly facilitates training in balance, ambulation and proper posture. The parallel bars have adjustable guide rails which aid in developing the proper gait by helping to correct common faults such as circumduction

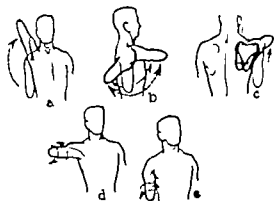


Figure 19 Exercises for above-elbow stump: a, Abduction and adduction of stump; b, flexion and extension of stump; c, abduction and adduction of the scapula; d and e, internal and external rotation of stump. These exercises are done bilaterally and with progressive resistance as soon as indicated.

increased until the patient can remain in a vertical position without the development of these untoward effects.

LOWER MOTOR NEURON DISORDERS

Objectives. The aims of treatment are to prevent or correct promptly any deformities to stimulate circulation of the involved part to institute any indicated dynamic splinting so that if reinnervation takes place the extremity will be able to function in response to voluntary nerve stimuli, to improve and maintain the general physical condition and to aid in the psychologic adjustment of the patient. In those individuals having poliomyelitis with respiratory involvement requiring the use of a mechanical respirator the physical medicine procedures must be accomplished during the period the respirator is in use as well as after its employment has been discontinued.

Physical Medicine and Rehabilitation Procedures. *Heat* To stimulate and maintain circulation of the involved parts, mild heat should be given.

Therapeutic exercise Until active motion is possible, passive exercises to prevent contractures and preserve function of the joints are promptly instituted. Exercise to prevent general deconditioning is imperative.

Electrical stimulation. To delay atrophy and aid circulation, electrical stimulation of the paralyzed muscles is indicated. The value of electrical stimulation in poliomyelitis is questionable, for during the process of stimulating the paralyzed muscles the current may spread and produce contraction of the unparalyzed antagonist muscles resulting in a greater degree of the already existing muscular imbalance.

Dynamic splints When indicated appropriate splints should be worn by the patient to prevent overstretching of the involved muscles. The springs or elastic on the splint will compensate for the lost motion, while the patient voluntarily contracts the uninvolved muscles. This procedure frequently permits functional activity on the part of the patient.

Massage Massage must be cautiously applied so as not to traumatize flaccid muscles. Heat passive exercise and electrical stimulation are more effective in these conditions.

Neuromuscular re-education This procedure should be started as soon as there is evidence of muscle reinnervation.

Traction Nerve root lesions in the cervical and lumbar regions due to herniated nucleus pulposus and arthritic changes frequently

produce local as well as radicular pain and limitation in motion of the spine and corresponding extremities. Rest, heat and properly instituted traction are indicated as conservative measures.

In cervical lesions, traction can be secured by means of a head halter while heat is applied to the neck and posterior shoulder muscles. The head is supported on a well-padded board having four ball bearing casters attached to the undersurface.

In lumbar lesions traction can be applied by means of a pelvic corset or belt while heat is given to the back muscles. The pulley and weights are attached to the foot end of the table which is slightly elevated to prevent the patient from sliding in the direction of the pull.

Therapeutic exercises to correct faulty posture and abnormal body mechanics, to increase muscle tone, and to restore mobility of the spine should be gradually started as soon as the acute pain subsides. Proper bed positioning—back or side-lying with the hips and knees flexed—is indicated. Generally flexion movements of the spine should be limited. The patient should be taught the proper way to lift objects, to get into and out of his automobile and into and out of a bath tub, and other activities with a minimum of back flexion. In those patients requiring laminectomy the therapeutic exercises should be continued postoperatively.

AMPUTATIONS

Objectives. In addition to the program being planned to return the amputee to a useful occupation, it is necessary to include the other important objectives, such as the psychologic and social adjustment to the loss of one or more extremities; the securing of a stump having maximum function insofar as range of motion, muscle strength and endurance is concerned, the securing and proper fitting of a suitable artificial extremity to meet the specific needs of the amputee, and sufficient training in the use of the artificial extremity to enable the individual to perform a sufficient number and type of activities that would justify its use. The value of the finest fitting artificial extremity is markedly decreased in direct ratio to the lack of function of the stump and the motivation of the amputee.

Physical Medicine and Rehabilitation Procedures. *Proper positioning* The patient and the stump must be properly positioned to prevent contractures of the stump activating and stabilizing muscles.

Valuable data can be secured by skin surface temperature tests performed at controlled environmental temperature and humidity and under basal metabolic conditions.

Following application of heat to the upper extremity the Landis Cihlon test records the change in skin surface temperature of the lower extremities through the reflex vasodilatation mechanism.

Plethysmography involves the measurement of volume changes in a given body segment.

Alterations in skin surface temperature and sweating patterns are noted following sympathetic nerve blocks.

The step cadence is timed with a metronome in order to have a controlled rate for measured distances to aid in evaluating the circulatory requirements of the lower extremities.

Therapeutic techniques. Proper skin care and the avoidance of trauma and exposure to extremes in temperature are of vital importance.

In patients with impaired venous circulation, hydrotherapy or direct mild radiant heat of low intensity to the extremities can be used, depending upon the severity of involvement. In arterial insufficiency this procedure is dangerous and it is safer to apply heat to the abdomen, chest or back in an attempt to improve the peripheral circulation through reflex mechanisms.

By means of direct current ion transfer of vasodilating drugs, such as histamine or Mecholyl, into the tissues can be of value in improving circulation and healing ulcers in selected patients. When histamine or Mecholyl is used the drug is placed on the tissues under the active electrode which is connected to the positive terminal of the battery or direct current generator while the dispersive electrode, which is placed on the body at a distance from the active electrode is connected to the negative terminal.

The following mechanical procedures can be tried, if no contraindication exists. The results obtained must be carefully evaluated under controlled conditions.

Massage may be helpful in reducing edema. It is definitely contraindicated in the presence of any infection.

The Buerger Allen exercises consist of a series of changes in the position of the patient and the extremities which alters the hydrostatic pressure in the blood vessels of the extremities and may enhance circulation.

The rocking type of bed rhythmically oscillates through a variable arc of approxi-

mately 20 degrees head-down and 30 degrees feet-down thereby slowly producing elevation and lowering of the entire body with associated changes in the arterial and venous pressures in the lower extremities.

The environmental pressure surrounding the extremity is increased and decreased rhythmically in relation to atmospheric pressure while the part is in a pavex (passive vascular exercises) boot.

By means of a rhythmic inflation and deflation of a cuff around the upper thigh, the venous circulation is occluded for approximately one to three minutes during each cycle and released for similar periods, depending upon the circulation to produce intermittent venous compression.

The vasopneumatic machine consists of a number of narrow inflatable cuffs applied to the entire extremity wherein a peristaltic pressure wave of controlled constriction can be produced both in a centrifugal and centripetal direction depending upon the circulatory condition being treated.

In those patients with Intermittent claudication a lift under the heel is frequently helpful in increasing the walking distance before the development of leg pain. With this procedure the calf muscles during the walking process contract minimally and therefore require less blood.

The application of cold is beneficial in preventing or diminishing pain and inflammation and in lessening the absorption of toxins in a severely infected or gangrenous extremity.

THORACIC SURGICAL CONDITIONS

There are specific indications for physical medicine and rehabilitation in thoracic surgical conditions, both tuberculous and non-tuberculous to further aid in the more rapid return of the individual to his community.

Objectives. The aims are the institution of measures that will aid in returning the patient to useful endeavors and prevent a "chest or respiratory cripple" relief of pain prevention and correction of deformities of the trunk and restriction of motion and function of the upper extremity. Mobilization of the thorax and re-expansion of a lung, when indicated and compatible with the medical condition and the surgery performed, should be accomplished in order to secure maximum respiratory function. Maintenance of good posture and general physical condition is essential.

Physical Medicine and Rehabilitation Procedures. Orientation. The patient should be

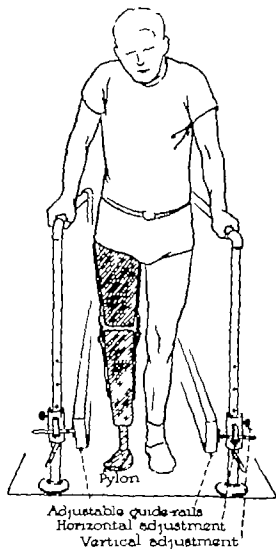


Figure 30 Above-knee amputee learning to ambulate with a pylon. The parallel bars have two guide-rails that are adjustable in both horizontal and vertical positions in order to limit circumduction and abduction while walking.

and extreme abduction of the legs. The guide rails are adjustable in both horizontal and vertical positions. All gait training, either within or outside of the parallel bars, should be done in front of a full-length mirror.

In bilateral above-knee amputations, the pylons should be constructed about 6 inches shorter than the original length of the legs when the patient is first learning ambulation because it lends to greater balance stability and ease in gait training. These are called "stubbies." Later the pylons can be lengthened to give full height to the amputee.

With the additional program of occupational therapy and manual arts therapy the arm amputee is taught to perform the maximum number of self-care activities and increase his work capacity. Training should al-

ways start with the hook and later with the hand. Selected amputees can be taught to drive safely an automobile having the proper operating controls. The amputee is further taught the proper care of the stump and the artificial extremity.

General conditioning exercise. The early institution of graduated general exercises to the entire body is essential and an important part of the total program. Proper performance with an artificial extremity is extremely difficult with a weakened body.

Heat. To aid in relieving pain and correcting contractures, mild heat is indicated. In the presence of ulcers and wounds, hydrotherapy is beneficial for cleansing and healing.

Ultrasound therapy. As an aid in relieving pain associated with neuromas and scars, ultrasound therapy has been utilized.

Research. Intensive coordinated research has been in progress by the Veterans Administration, the Armed Forces, various engineering institutions, artificial limb manufacturers, and others to develop improved artificial extremities utilizing plastics, metals, electronic devices and other materials and appliances. This research also incorporates the study of body mechanics and the fitting and alignment of the prosthesis.

PERIPHERAL VASCULAR DISORDERS

Physical medicine and rehabilitation is indicated as an aid in diagnosis, prognosis and treatment in selected patients having peripheral vascular disorders.

Objectives. The aims are to improve circulation in the extremities by decreasing arterial tone and spasm, to establish collateral circulation, to prevent trauma and infection and to aid the individual to perform the maximum number of self-care activities including ambulation.

Physical Medicine and Rehabilitation Procedures. The instituted program will depend greatly upon the type and extent of the pathologic processes. In many patients, the earlier that conservative treatment is utilized, the greater the possibility of benefit, even perhaps to the saving of an extremity.

Diagnostic techniques. Procedures for evaluating peripheral circulation include:

Palpation of peripheral arterial pulsation of all extremities should be performed.

Oscillometric tests are used to measure the pulsatile increase of the volume of an extremity with each cardiac systole which is a reflection of the amount of blood flowing through the main arteries.

tion in ambulatory patients than in those who are confined to bed.

Occupational therapy, manual arts therapy, educational therapy, and speech therapy. These additional physical medicine and rehabilitation activities properly prescribed can be of tremendous benefit to the patient not only as an aid in increasing work capacity but also in preventing or overcoming the many psychologic and emotional problems that exist in the minds of many of the sick, thereby making for better hospital adjustment.

This type of activity participation can also be of an advisory or exploratory nature toward future employment. These far-reaching beneficial effects especially for one who cannot return to his former type of occupation because of his disability definitely aid in the getting well process.

SUMMARY

It is extremely significant that there be an awareness and understanding, on the part of all concerned with the care of the sick, of the importance of physical medicine and rehabilitation in the total rehabilitation of the disabled. It is not offered to supersede or supplant the best medical and surgical procedures, for its greatest benefits are derived when it is completely integrated and coordinated with all of the other professional contributions. The procedures indicated for the various conditions are to be used only as guides and suggestions. There are no routine measures, for with some patients certain techniques may be definitely contraindicated. Coexisting medical conditions may frequently alter the physical medicine and rehabilitation procedures under consideration. The patient's condition governs the type and amount of rehabilitation. It is imperative that intensive research be continued for additional and improved techniques for both diagnosis and treatment.

The role of physical medicine and rehabilitation in geriatric medicine is of extreme importance. The care of the aging and aged has added significance since aging is part of living and the older age group is increasing with time and the advances in medicine. The physiologic changes that are produced during normal aging are further complicated by the effects of disease and injury. Geriatric medicine is a challenging field in which physical medicine and rehabilitation contributes toward the usefulness of elderly individuals.

Physical medicine and rehabilitation is all ways medically prescribed with precise objectives toward a definite realistic goal to meet the self-care and daily living requirements of the individual person so that he may obtain the maximum possible benefit toward his physical, mental, social and economic usefulness. Frequently after discharge from the hospital the person may benefit by temporary employment in a sheltered workshop before attempting full employment in a competitive environment. By minimizing the degree of handicap total rehabilitation can bring to the disabled a satisfactory measure of happiness and contentment leading toward a fuller life.

READING REFERENCES

- A Manual of Cerebral Palsy Equipment. Chicago: The National Society for Crippled Children and Adults, 1950.
- A New Approach to the Rehabilitation of the Blind. V.A. Pamphlet 10-32, Washington, D.C., Veterans Administration, February 1950.
- Arnold, A. J., Newman, L. B., and Fitzell, J. A. Electromyography. *Arch. Phys. Med.* 32:320, 1951.
- Bierman, W. and Licht, S. *Physical Medicine in General Practice*. 3rd ed. New York, Paul B. Hoeber, 1952.
- Corrective Therapy in Veterans Administration Hospitals, Centers, Outclinics, and Regional Offices. Technical Bulletin 10A-35, Washington, D.C., Veterans Administration, March, 1954.
- Davis, L. *The Principles of Neurological Surgery*. 4th ed. Philadelphia, Lea & Febiger, 1953.
- Deaver, C. C. What Every Physician Should know about the Teaching of Crutch Walking. *J.A.M.A.* 142:470, 1950.
- DeLorme, T. L. and Watkins, A. L. *Progressive Resistive Exercise*. New York, Appleton-Century-Crofts, Inc., 1951.
- Educational Therapy for Patients in Veterans Administration Hospitals. Technical Bulletin 10A-118, C1, Washington, D.C. Veterans Administration, February 1948.
- Goldeth, J. C., and Fitzell, J. A. A Constant Current Impulse Stimulator. *Arch. Phys. Med.* 28:154, 1947.
- Handbook of Physical Medicine and Rehabilitation. Philadelphia, Blakiston Company, 1950.
- International Conference on Ultrasonics in Medicine. *Am. J. Phys. Med.* 3:107-113, 1958.
- Karpovich, P. V., Starr, M. P., and Weiss, R. R. Physical Fitness Tests for Convalescents. *J.A.M.A.* 126:873, 1944.
- Kessler, H. H. *The Principles and Practices of Rehabilitation*. Philadelphia, Lea & Febiger, 1950.
- Keys, A. Introduction to the Symposium on Convalescence and Rehabilitation. *Fed. Proc.* 3:189, 1944.
- Knudson, A. B. C. Dynamic Aspects of Physical Medicine in the Veterans Administration. *Arch. Phys. Med.* 29:29, 1948.
- Krusen, F. H. *Physical Medicine and Rehabilitation*

oriented preoperatively as to the planned program. This will also include preoperative preventive treatment. Postoperatively the treatment procedures should be gradually reinstituted as soon as medically feasible, which is usually on the first postoperative day.

Correct bed posture Proper pillow arrangement and positioning of the patient in bed are important in helping to prevent postural deformities.

Therapeutic exercise General body relaxation exercises are indicated and should be initiated early, particularly during the bed rest period. Active and active assistive exercises of the upper extremity and shoulder to prevent restriction of movement, especially in abduction and external rotation of the shoulder should be promptly started. As soon as indicated, graduated resistive exercises should be instituted. General conditioning exercises should also be instituted early. Wands, pulleys and weights are helpful aids, especially for symmetrical exercises which, whenever possible should be performed before a mirror.

Breathing exercises Hemithorax measurements of the respiratory movements of both halves of the thorax, taken simultaneously, and spirometric readings should be secured periodically to evaluate progress.

When lung expansion is desired, diaphragmatic (abdominal) breathing, as well as segmental chest movements, is started. When not contraindicated, the use of "blow bottles" is helpful to re-expand a collapsed lung. In pulmonary tuberculosis and following collapse therapy such as in thoracoplasty breathing exercises of the abdominal type only are advisable.

Heat To aid in the relief of pain and as an adjunct in the stretching of contractures, mild heat is beneficial.

Work capacity A selected, graded and controlled program of activity including occupational therapy, manual arts therapy and educational therapy will aid in motivating the patient in overcoming his psychologic and emotional problems and in increasing his work capacity and also will serve as an excellent vocational exploratory technique.

OTHER SURGICAL CONDITIONS

There are numerous other surgical conditions, such as abdominal operations which can be benefited by both preoperative and postoperative properly prescribed physical medicine and rehabilitation measures.

Objectives. Through a therapeutic exercise

program including early ambulation, the individual develops a sense of well-being and is greatly aided toward a more rapid return to normal activity and economic usefulness. Many complications are prevented or minimized and the patient's hospitalization period is reduced.

Physical Medicine and Rehabilitation Procedures. The type and extent of the conditioning and early ambulation program must be guided by the type and extent of the surgical procedures and the general condition of the patient. The closest coordination between the surgeon and the physiatrist is imperative. The patient must be oriented to the necessity and benefits of the activities, for this will help to allay anxiety, apprehension and any fears thereby obtaining his maximum cooperation toward the total rehabilitation regimen.

Therapeutic exercise Graduated general body conditioning exercises, under close supervision, should be instituted preoperatively if possible and promptly resumed postoperatively to avoid the debilitating effects of bed rest. Activity frequently aids in controlling the ill effects that are temporarily produced by the procedures associated with the actual surgical intervention.

Ambulation Shortly after surgery ambulation is desirable and necessary. The patient should be taught the proper method of getting into and out of bed and walking with the least effort. This is easier to accomplish shortly after surgery than after the patient has been confined to bed for a number of days without activity. Walking distances should be gradually increased and attention given to correct posture. Proper breathing exercises are also included.

Prolonged immobilization in bed interferes with the normal physiologic processes of the body in the normal individual, but more so during illness, and also predisposes to psychologic and emotional problems. The latter may be a contributory factor to "hospitalitis."

Contraindications to a judiciously prescribed and individualized exercise and early ambulation program are perhaps relatively few. At certain times ambulation may not be advisable, however graduated conditioning therapeutic exercises may be indicated. Shock, internal hemorrhage and severe systemic infections are usually contraindications to early ambulation. It has been observed over many years that postoperative fever is frequently lower and of lesser dura-

THE VASCULAR SYSTEM

The Veins

By HAROLD LAUFMAN M.D.

HAROLD LAUFMAN was born in Wisconsin and educated at the University of Chicago and Rush Medical College. His graduate education in surgery was pursued further at Northwestern University. There he has continued to make important research contributions to surgery. His talents as an artist have won recognition among that group of surgeons who have an unusual creative artistic gift along with their surgical abilities.

When man assumed an upright posture his venous system—at best a relatively inefficient conduit system—became subject to added burdens. The veins serve as little more than channels to return blood to the heart. Propulsion of blood through veins is primarily dependent upon what force remains after arterial blood has passed through the capillary bed. Venous flow is assisted by such auxiliary measures as the sucking action of respiration, skeletal muscle contraction, the valves, and gravity except in the lower portion of the body where gravity hinders venous flow. These aids to venous circulation have not prevented the venous system of man from falling heir to the consequences of stagnant flow. When extraneous influences such as injury, infection, and obstruction to flow become superimposed, the veins and the spent,

poorly oxygenated blood they carry become ready ground for pathologic alteration.

ANATOMY AND PHYSIOLOGY

In the extremities and mesentery the veins are more or less paired with the arteries. This relationship, however, is not constant throughout the body for in some areas, such as the liver, cranium and chest, the veins and arteries run independent courses. The muscular coat of veins is considerably thinner than that of arteries, and possesses neither the contractile power nor strength of artery muscle. Consequently spasm in veins, though it does occur, is not extreme and distention may become marked with moderate increases in venous pressure. Persistently elevated venous pressure can overstretch and attenuate the muscular coat of vein walls to such an

- for the Clinician. Philadelphia, W. B. Saunders Company 1951.
- Lehmann, J. F.: The Biophysical Basis of Biologic Ultrasonic Reactions with Special Reference to Ultrasonic Therapy. *Arch. Phys. Med.* 34:139 1953.
- Licht, S.: Electrodiagnosis and Electromyography. New Haven, Elizabeth Licht, 1956.
- Licht, S.: Therapeutic Heat. New Haven, Elizabeth Licht, 1958.
- Manual Arts Therapy in Veterans Administration Hospitals and Centers, and Regional Offices. Technical Bulletin 10A-348. Washington, D. C., Veterans Administration, October 1953.
- Manual of Upper Extremity Prosthetics. California, Department of Engineering, University of California, 1952.
- Medical Rehabilitation Board. Manual M-2, Part VIII, Washington, D. C. Veterans Administration, July 1955.
- Newman, L. B.: Tracing Device for Surface Lesions. *Arch. Phys. Med.* 29:42, 1948.
- Newman, L. B.: A New Device for Measuring Muscle Strength—The Myometer. *Arch. Phys. Med.* 30:234 1949.
- Newman, L. B.: Lumbar Pelvic Traction Apparatus. Dept. of Medicine and Surgery. Information Bulletin IB 10-92, Washington, D. C. Veterans Administration, January 1957.
- Newman, L. B., Colwell, C. A. and Jameson, E. L.: Decontamination of Articles Made by Tuberculous Patients in Physical Medicine and Rehabilitation, Study Using Carbamide Gas. *Am. Rev. Tuberc.* 71:272, 1955.
- Newman, L. B., and Jameson, E. L.: Assistive Devices for the Disabled. *Occup. Therapy* 30:8, 1951.
- Occupational Therapy Technical Bulletin 10A-14* (C3 Amended by TB 10A-187 10A-190) Washington, D. C., Veterans Administration, July 1948.
- Physical Therapy in Veterans Administration Hospitals, Centers, Domiciliaries, Regional and V. A. Offices. Technical Bulletin 10A-348, Washington, D. C., Veterans Administration, September 1953.
- Pollock, L. J., and others. Electrodiagnosis of Lesions of Peripheral Nerves in Man. *Arch. Neurol. & Psychiat.* 60:1 1948.
- Richter, C. P.: Instructions for Using the Cutaneous Resistance Recorder or "Dermometer" on Peripheral Nerve Injuries, Sympathectomies and Paravertebral Blocks. *J. Neurosurg.* 3:181 1946.
- Self-Care Activities, Functional Evaluation. Form 10-2617. Washington, D. C., Veterans Administration, June, 1953.
- Self Help Devices for Rehabilitation, New York University Bellevue Medical Center Duboque, Iowa, William C. Brown Company 1958.
- Stieglitz, E. J.: Chronic Illness and Senescence. *J.A.M.A.* 150:481 1952.
- Waldin, L. G.: The Physiologic Aspects of Therapeutic Exercise. *J.A.M.A.* 142:100, 1950.
- Wepman, J. M.: Recovery from Aphasia. New York, Ronald Press, 1951.

are higher than normal and walking causes no drop or only a moderate drop. Actual reversal of flow has been demonstrated in some individuals with varicose veins.

The two main diseases of veins are varicosities and thrombophlebitis. It is of more than passing interest that these diseases often occur together as interdependent cause-and-effect entities, although they may appear independently.

VARICOSE VEINS

Varicose veins appear as tortuous dilated distortions of otherwise relatively straight veins. Varices may appear in several areas of the body. The commonest types aside from those of the lower extremities are esophageal varices, usually resulting from portal hypertension; varices of the spermatic veins; dilatation of the inferior hemorrhoidal veins;

varices of the broad ligament; urinary bladder and prostatic venous plexus; and varices of the central nervous system.

Incidence. Various estimates of the incidence of varicose veins have been made, but in general it probably can be said that about 10 per cent of all adults over thirty-five years of age have some degree of varicose change in their saphenous venous system.

Etiology. The fundamental defect responsible for varicosities in the legs is probably incompetence of the valves in the connecting veins between the superficial and deep venous systems (Fig. 1). When incompetent, the valves no longer prevent backflow and thus allow blood with a greater than normal pressure-head to fill the superficial system. Important factors in the etiology of varicose veins are heredity, increased postural strain, trauma, compression or constriction of veins and phlebitis of the deep venous system.

Congenital weakness of veins appears to be a familial tendency. One can often find the same defect in one or both parents. The offspring of parents with varicosities tend to develop varicosities at a young age. In addition to familial or perhaps congenital weakness of vein walls, other etiologic factors may be endocrine or even chemical changes.

Prolonged standing is said to produce enough undue strain to cause varicosities. However, examination of people who might be predisposed by their occupations (e.g., floor walkers and policemen) does not show a higher incidence of varicose veins in this group than in any other segment of the population. On the other hand, it is possible that such occupations may aggravate previously existing tendencies to varicose vein formation.

Other factors tending to increase venous pressure in the legs include constricting garters, enlarged lymph nodes, pelvic tumors and similar agents. The relatively high incidence of symptomatic varicose veins during pregnancy has aroused curiosity regarding a possible relationship. Occasionally the most severe symptoms occur in the early stages of pregnancy, long before the growing uterus reaches its greatest size. This observation has led to the speculation that an endocrine factor may produce weakness of the vein walls. However, the congestion within the pelvis during the early stages of pregnancy may more than offset the relatively small size of the uterus as a pressure phenomenon.

The superficial venous system of the leg is less able to cope with the stress of increased

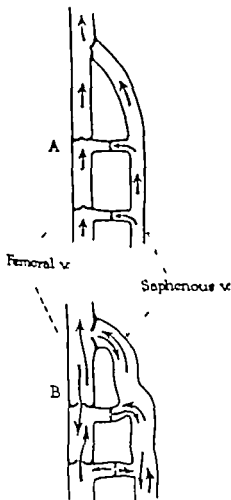


Figure 1 Schematic representation of venous blood flow in superficial and deep veins of the legs. A, Normal relationship. Competent valves permit blood to flow only proximally and only from superficial to deep veins. B, Incompetent valves allow reversal of flow from deep to superficial veins, causing the latter to dilate and become tortuous. Valves in deep vein may remain competent.

extent that the muscle loses its homeostatic ability to resume normal tonus. The result may be irreversible ballooning and tortuosity. Alterations in the vein wall itself may weaken its structure.

The veins of the extremities contain valves, whereas those of the abdomen, thorax and cranium do not. Venous valves are arranged in such a way that blood flows past them toward the heart. They are cup shaped, with their concave face pointing proximally. Their function apparently is to prevent backflow under sudden increases in venous hydrostatic pressure and to aid in the directional flow of blood when venous pressure falls to low levels. Valves are located rather irregularly along the main stem veins but invariably occur at the branching sites between deep and superficial vein systems in the extremities.

The deep and superficial sets of veins in the leg bear special consideration since their differences and their relationship with each other may be the background for disabling disease. The superficial set of veins—the saphenous system—lies between the deep fascia and skin surrounded only by weak superficial fascia and a variable amount of subcutaneous fat. The deep set—the femoral vein system—is housed in muscular compartments, or fascial spaces. The two systems connect at two main points, in the groin (saphenofemoral junction) and in the popliteal space (lesser saphenopopliteal junction). Valves exist at these junctions. The two systems are also connected by a number of connecting (so-called perforating or communicating) veins, each containing valves, as well as by many smaller anastomotic veins. Thus, blood flowing from the toes to the heart has many alternate routes of flow. Communications between the deep and superficial venous systems of the leg are most numerous in the foot, less in the calf and least in the thigh.

The great saphenous vein runs the entire length of the leg from foot to groin. Its course is along the medial aspect of the leg at the ankle it is anterior to the medial malleolus; it ascends posteromedially behind the medial condyle of the femur then directly to the fossa ovalis of the superficial fascia inferior to the inguinal ligament. It dips medially and deep to join the femoral vein. Branches at or near the saphenofemoral junction are of surgical importance, since all must be recognized and divided in order that excision of the great saphenous vein for varicoseities will not be followed by recurrence of the condition. As a rule, five branches with

or without variations are seen. These are the superficial inferior epigastric vein, which ascends from the junction to run in a cephalad direction; the superficial circumflex iliac vein, which takes an obliquely lateral course; the superficial external pudendal vein, which runs medially; the medial and lateral femoral cutaneous veins, which take an obliquely downward course. Variations on this basic anatomic pattern may be extreme, and as many as twelve or more branches may be found.

The lesser saphenous vein collects superficial venous drainage from the lateral malleolar area and posterolateral region of the calf and ascends to the popliteal space, where it dips below a strong layer of the superficial fascia to join the popliteal vein.

Neither the great nor the lesser saphenous vein is accompanied by arteries.

The deep veins of the leg, on the other hand, are paired with arteries. The anterior and posterior tibial veins approach the midline as they ascend in the leg and join approximately in the posterior midline to form the popliteal vein behind the knee. The popliteal vein perforates the adductor magnus muscle to reach the adductor canal, at which point it is known as the femoral vein or superficial femoral vein. The femoral vein and artery are closely associated in the remainder of their cephalad course, the vein lying lateral and posterior to the artery in the lower thigh, and medial to it at the inguinal area.

The profunda femoris or deep femoral vein, collects blood from the deep thigh muscles and emerges to join the superficial femoral vein about an inch and a half below the inguinal ligament. The profunda femoris vein has generous anastomotic connections with the branches of the inferior gluteal vein above and popliteal vein below.

Above the juncture of the deep and superficial femoral veins, the common stem is known as the common femoral vein. As it dips behind the inguinal ligament it becomes the external iliac vein. In the pelvis it is joined by the internal iliac (hypogastric) vein to form the common iliac vein. The latter joins its opposite fellow to form the inferior vena cava.

Normally venous pressure in the lower extremity is highest upon standing and lowest when the legs are elevated. Under normal circumstances, after an initial slight rise, it drops precipitously during walking. The motion undoubtedly has a milking effect which aids in emptying the veins. In the presence of incompetent valves, the standing pressures

the saphenous and communicating veins (Fig. 2). With the patient sitting or lying the leg is elevated above the level of the pelvis so that the superficial veins empty by gravity assisted by upward massage by the examiner's hand. Compression is then made over the fossa ovalis in the groin either by the examiner's fingers or by a tourniquet in order to compress the saphenofemoral junction. The patient is then asked to stand. If the saphenofemoral valve is the sole source of the incompetency the veins will remain empty for a minute or two before filling down from below. Removal of the compressing fingers or tourniquet before complete filling permits rapid filling from above. Rapid filling from below in the presence of the pressure indicates incompetency of one or more perforating veins below the level of the constriction. The site of the incompetency can be found by repeating the maneuver with the tourniquet placed at various lower levels. The short saphenous vein may be a contributing or the sole source of the varicosities. Incompetency of the valve at the junction of the short saphenous and popliteal veins can be tested by a similar method at that level.

Perthes test is performed in order to test the patency of the deep venous system. The test is carried out by constricting the saphenous trunk in the thigh by use of a tourniquet. The patient is asked to exercise his leg by some such maneuver as kicking it vigorously back and forth or by walking about the room. With exercise blood normally is routed into the deep veins, causing the varicosities to empty. If the deep veins are not patent or if there is increased hydrostatic pressure in the deep veins the varicosities will not diminish in size. Perthes test is not of much practical value in most instances because the deep veins are almost never blocked so completely that some channel will not be available for the flow of venous blood.

Several other tests have been devised to locate the position of incompetent perforating veins. However most of these tests are of little more than academic interest to the surgeon. In the present-day surgical approach to the problem, the entire main stem is usually eradicated by stripping. Thus all perforating branches are interrupted where they join the great saphenous vein and their exact location is not crucial.

Complications. So-called *varicose ulcer* occurs characteristically in the lower third of the leg or in the area of the malleoli. Almost

without exception a varicose ulcer appears directly over a vein. For this reason its pathogenesis has been ascribed to periphlebitis which occurs as a sequela to localized phlebitis. The inflammatory reaction about the vein is said to choke off the nutrient blood supply to that portion of the skin. When such an area of skin is irritated by trauma it does not heal normally. Instead the tissue may break down and ulcer formation results. Therefore varicose ulcer is not due to varicose veins alone for if it were ulcers would occur in other areas of the legs where varicosities may be more extensive. Nor is stasis alone responsible since ulcers rarely occur on the foot. The relatively poor blood supply to the skin in the area above the malleoli and the characteristic location of an ulcer directly over veins in this area are factors responsible for the phlebitis concept. As a rule varicose ulcers occur more readily in limbs which have been previously afflicted with deep vein thrombophlebitis, complicated by chronic circulatory changes in the skin.

Although *edema* is not always a characteristic sequela to otherwise uncomplicated varicose veins it occurs in a large number of the patients. The examiner must be careful to exclude the possibility of systemic causes of ankle edema if the condition is bilateral. Edema which occurs with otherwise uncomplicated varicose veins tends to be accentuated toward the end of the day and to subside with elevation of the limb. If the edema is associated with pain and with shiny skin and tenderness of tissue one should suspect deep vein occlusion or thrombophlebitis.

Rupture of varicosities may take place with loss of considerable blood. This may occur without warning, particularly in patients who have superficially placed varicosities. In such individuals the veins have a deep blue color and are covered only by a thinned layer of dermis. Such simple activities as crossing the legs may result in bleeding, first noticed by a surprisingly large flow of blood down the leg, without pain. Such bleeding is usually easily controlled by elevation of the limb and the application of a pressure bandage.

Thrombophlebitis is a relatively frequent complication of varicose veins. Local areas of thrombophlebitis of the superficial veins are probably more the result of stasis and trauma than of bacterial invasion. The usual clinical picture is that of a painful red tender area of the skin overlying a firm segment of vein. Such involvement may vary from a low grade

hydrostatic pressure than is the deep system since it lies relatively unsupported in the subcutaneous tissue. Venous flow through the deep system is aided by the support of relatively strong tissues such as muscle and fascia, and by the contractions of the muscles. Undue increases in hydrostatic pressure tend to follow paths of least resistance. Increased femoral vein pressure is thus transmitted through communications to the superficial veins of the leg. The valves at these junctions give way and become incompetent and the disproportionately high pressures in the poorly supported saphenous system may lead to tortuosity.

So-called secondary varicosities may follow by months or years the development of deep vein thrombophlebitis. A greater load of venous blood than is normal is shunted into the superficial venous system following blockage of the deeper veins and the ensuing incompetence of their valves after recanalization.

Pathology. Microscopic examination of the dilated, tortuous, thinned-out veins reveals areas of musculature hypertrophy interspersed with areas of remarkable thinning of the vein wall. Fibrosis is widespread. Low-grade inflammatory changes are frequent. Phlebosclerosis, with actual calcium deposition as a sequela to fibrosis, is a rather common finding, even in the relatively young patient.

Occasionally the venous stasis is carried into the tiny intradermal radicals. When this occurs, so-called spider burst or spider web varicosities are seen in the skin. Microscopic examination reveals these areas to be tortuous enlargements of the subpapillary plexus of intradermal veins.

Arteriovenous fistulae particularly of the minute multiple type, are often associated with varicosities and have been ascribed an etiologic role in certain cases.

Symptoms. Great disparity often exists between the clinical appearance of varicose veins and the symptoms they produce. Thus patients with minimal varicosities might have severe symptoms while some with markedly tortuous veins have minimal or no symptoms. As a rule the early symptoms are heaviness, drawing sensations and cramping, particularly in the region of veins, and especially while standing. Pain is a variable symptom and most often occurs in conjunction with localized areas of thrombosis. However it may occur in the absence of thrombosis and may be accentuated by pressure or by the presence of a cluster of varices. The most severe symptoms are due to the complica-

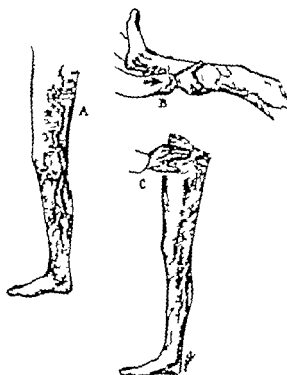


Figure 2. Trendelenburg test. A, Incompetent great saphenous vein with marked varicosities. B, Leg elevated above level of pelvis to empty veins. Emptying aided by gentle upward massage. C, Occlusion of saphenofemoral junction by examiner's fingers, veins remain relatively empty when patient stands; refill rapidly from above when hand is removed.

tions of varicose veins such as dermatitis, ulceration and superficial thrombophlebitis. Curiously symptoms may be more severe in the earlier developmental stages of varicose veins than later.

Diagnosis. Because patients with varicose veins often have other infirmities of middle age, symptoms are often erroneously ascribed to the veins. On examination static deformities, arthritis, sciatic neuritis, myositis and arterial system symptoms may be the actual cause of the symptoms present in a patient who has varicose veins. Patients often express great disappointment when symptoms do not disappear upon removal of the varicose veins. Thus careful history taking and physical examination are important. A complete physical examination should accompany every local examination for varicose veins to search for constitutional diseases, infectious diseases, peripheral arterial disease and abdominal tumors.

Observation of the legs should begin with the patient standing. Once the extent and location of the varicose veins have been determined, it is traditional to perform the Trendelenburg test and Perthes test.

The Trendelenburg test is made to determine the location of valves in

infection or death from pulmonary embolism. This phenomenon, known as thromboembolism, unfortunately is still as prompt a cause of death today as it was before the advent of modern advances in treatment and prevention. Our inability to recognize the existence of bland thrombosis in the lower limbs early enough and our ever increasing success in surgery upon poor risk patients probably keep the incidence high. Bland thrombosis or "silent" thrombosis originally described by Homans in 1931 more recently has been termed phlebotrombosis by Ockner and DeBakey. The most usual site for this type of thrombosis is the deep veins of the legs.

Etiology and Pathogenesis. In the earliest stages of bland thrombosis, a fresh clot appears in a vein which otherwise may exhibit no obvious changes. Many theories have been devised to explain this occurrence, some based upon sound experimental or clinical

evidence others deduced from known applicable facts.

Most instances of venous thrombosis in the lower extremities occur without a demonstrable precipitating factor. Possible causative factors may be divided into three main groups: local vessel changes such as intimal damage, venous stasis, and changes in the coagulating properties of the blood.

It is plausible to suspect that intimal damage due to local injury or disease in the vein wall is a likely cause of bland thrombosis (Fig. 3). While extensive studies of the veins of patients dying of thromboembolism do not appear to bear out this concept, experimental thromboses can be produced by deliberate intimal damage.

Venous stasis has long been suspected as an etiologic influence in phlebotrombosis. Supportive evidence for this relationship has been provided by close examination of fresh small thrombi seen to originate in areas

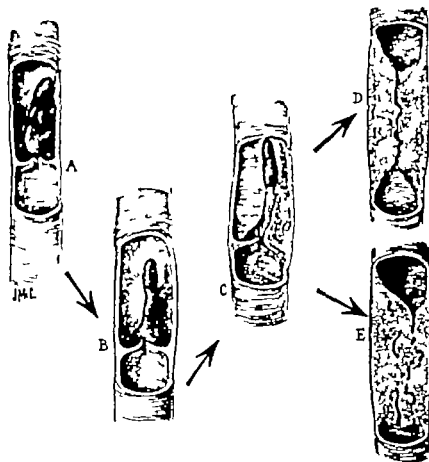


Figure 3. Natural history of venous thrombosis in lower extremity. A, Fresh clot forming on proximal surface of valves. B, Retraction of clot (more pronounced in anemia) and attachment to vein wall, beginning involvement of vein wall in reactive process. C, Further organization of clot and involvement of vein wall (new valve structure not easily discernible from organized clot on section); propagation of fresh "tail" clot on thrombus (this portion may be swept away as an embolus). D, Recanalization of organized clot with one main channel. E, Recanalization of organized clot with several smaller channels. Note in sketches D and E that vein wall and organized thrombus appear almost indistinguishable from each other (adopted from Hamey).

inflammatory reaction to a severe suppurative type of lesion with systemic septic phenomena. As a rule, inflammatory types of thrombophlebitis in superficial veins do not produce embolism.

Varicose dermatitis varies from the leathery scaly discoloration so often seen in patients with long-standing varicose veins to an acutely inflammatory red, itching, burning type of lesion. The dermatitis is usually located over the lower third of the limb. The lesion has been termed varicose eczema and may be due in some cases to allergic phenomena. Secondary pyogenic or fungal invasion is not uncommon.

Treatment. The treatment of varicose veins can be divided into conservative and operative measures. The conservative treatment is reserved for patients who for some reason cannot withstand surgery and for those whose involvement is minimal. As a temporary palliative measure when surgery cannot be undertaken wearing elastic stockings from toes to groin offers symptomatic palliation. They compress the superficial veins and shunt venous flow through the deep veins. When the veins are minimal and consist only of an occasional small, tortuous twig in the absence of a positive reaction to the Trendelenburg test, such a small segment can usually be obliterated by the injection treatment.

Injection treatment consists of the injection of a sclerosing solution directly into the vein. This mode of treatment is usually temporary. There are some definite contraindications to injection treatment. It should not be carried out in the presence of an acute upper respiratory infection, uncontrolled diabetes, local ulcers or inflammation or advanced arterial disease of the legs and it should be avoided if the patient has a history of cardiovascular accident. A few deaths from embolism following the injection treatment of varicose veins have been reported. An obvious criticism of this form of treatment is that it only obliterates the immediately visible segment and there is no assurance that the obliterated vein will not become recanalized. Hence, in the presence of a positive reaction to the Trendelenburg test, the sclerosing treatment of varicose veins is of no value, since the main stem is incompetent. Also when the varicosities are secondary to deep vein disease this type of treatment is worthless.

Today the greatest usefulness of the injection treatment lies in the postsurgical period, at which time small remaining vari-

ces may be obliterated if they persist after main-stem stripping.

A wide variety of solutions have been used, including hypertonic glucose solutions and alkaline soap solutions such as sodium morrhuate, sodium ricinoleate and sodium oleate. They act by producing a local thrombus.

Injectations are made with the patient standing and with no tourniquet on the leg. Care must be exercised not to allow any of the irritating solution to reach the perivascular tissues. If this occurs, a painful irritation may be set up, which may lead to sloughing of the skin. Injectations are usually made no more often than a week apart.

Surgical treatment for varicose veins consists in the ligation and separation of the great saphenous vein at the saphenofemoral junction, ligation and severance of all the branches arising from this area of the saphenous vein, and removal of the entire length of the great saphenous vein from the groin to the ankle. If indicated, the lesser saphenous vein at its juncture with the popliteal vein behind the knee is similarly ligated and stripped. Separate incisions may have to be made to remove clusters of veins which are only indirectly connected with the main stream. Only by such a complete maneuver can one expect to remove most of the involved veins as well as the main, incompetent channels. If new small varicosities should appear in the months following surgery these may be injected or excised. Despite this radical type of treatment, a few patients particularly those with secondary varicosities pursuant to deep vein thrombophlebitis will have recurrences.

A most important feature of surgical treatment is patient long-term follow up, so that new varicosities may be handled appropriately.

PHLEBOTHROMBOSIS AND THROMBOPHLEBITIS

As the name implies thrombophlebitis is a composite disease consisting of thrombosis of blood within a vein and inflammatory involvement of the vein wall. A wide variety of types exists depending upon degree of severity extent of involvement, anatomic location and amount of inflammatory reaction.

The problem of thrombophlebitis is an important one, not only because of its local effects but because of the propensity for portions of the venous clot to break off and travel back to the heart and into the pulmonary artery. The result may be pulmonary

usually does not give rise to emboli. Thrombophlebitis occurring in an uninjured limb of an otherwise healthy person may be the first clinical indication of thromboangiitis obliterans—this happens in some 10 per cent of patients with thromboangiitis obliterans—and as an occasional first sign of carcinoma of the pancreas or lung or of certain blood dyscrasias. Hence the occurrence of otherwise unexplained thrombophlebitis is reason enough for careful search for undetected neoplastic or systemic disease.

Surgeons have long held the impression that major time-consuming abdominal operations, especially those involving great blood loss or those producing hypotension, are more liable to be followed by venous complications than are the more superficial or shorter operative procedures. Acutely ill medical patients appear to be more susceptible than do many chronic invalids. Bed rest is certainly not the only precipitating cause, as evidenced by the extremely low incidence of thrombotic disease among chronically bed-ridden patients.

Partial obstruction to return flow from the lower limbs may result from tumors of the pelvis, unusual positioning during surgery, abdominal distention and pregnancy. Stagnation and thrombosis may result.

Sites of Thrombosis in Lower Extremities (Fig. 5) It has been supposed that thrombi originate predominantly in the smaller deep veins of the calf. However, meticulous post-mortem dissections reveal the distribution of clots to be extremely widespread and their location appears to bear little relationship to ante-mortem local symptoms. In some 40 per cent of the patients thrombi occur bilaterally in the lower extremities. The deep femoral venous system of the thigh contains thrombi oftener than does the superficial femoral venous system in the thigh and the calf, including the so-called deep veins of the calf. The internal iliac veins contain thrombi in only 6 per cent of the subjects. Thus it would appear that phlebothrombosis actually is more common in the deep femoral vein branches of the thigh than in the calf veins, although clinical symptoms are more readily detectable when it is in the calf.

It has been suggested that thrombi in the legs begin during surgery although they may not become clinically evident for several days, or perhaps not at all.

Incidence. Despite all the therapeutic and diagnostic advances in recent years the incidence of phlebothrombosis, thrombophlebitis and thromboembolism has not changed ap-

precisely. Failure to recognize a bland thrombus until embolism occurs has no doubt kept the incidence of embolism virtually constant. A review of reports from all over the world involving over three million operations showed an incidence of fatal pulmonary embolism ranging from 0.1 to 0.57 per cent, averaging 0.14 per cent with remarkably little change over the past fifty years. When statistics were based upon autopsy findings the incidence of death from pulmonary embolism varied from 0.2 to 23 per cent, with an average of 2.8 per cent. In these long range statistics there appeared an interesting but unexplained wavelike periodicity even in the same institution. On a shorter range basis thromboembolism appears to be more prevalent in the spring and autumn months in temperate climates. Since 95 per cent of all fatal pulmonary emboli are said to originate in the lower extremities this anatomic site has naturally attracted the most attention for diagnosis and prophylactic treatment.

Among hospitalized patients sixty of every

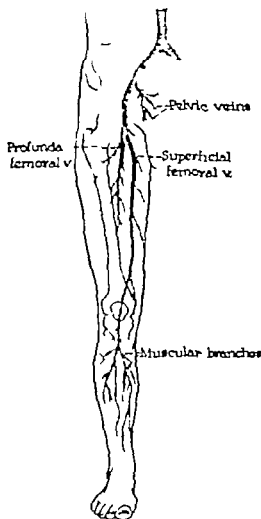


Figure 5 Usual sites of origin of phlebothrombosis in the lower extremity and pelvis.



Figure 4 A, Thrombosis of twenty-four hours duration (phlebothrombosis). Vein wall has normal appearance. B, Thrombosis of three days duration. Note beginning involvement of vein wall (thrombophlebitis). C, Thrombosis of four days duration. Clot shows early organization and covering by endothelial cell layer. Vein wall thickened. D, Thrombosis of seven days duration. Organized clot almost indistinguishable from the greatly thickened vein wall. Beginning recanalization.

where stagnation is maximal—in the cul-de-sacs formed by the proximal surfaces of venous valves. As the clot grows and becomes organized, this localization becomes less and less obvious because of propagation and involvement of the entire contiguous vein wall.

Changes in the coagulative properties of the blood have been found postoperatively in some patients, but studies on blood fibrinogen, antithrombin, fibrinolysin and platelet counts have been unfruitful. Under certain circumstances, such as following splenectomy increased platelet counts do parallel an increased tendency to thrombosis.

Many clinical conditions seem to predispose to venous thrombosis. These include surgical operations, injuries, burns, infectious diseases, cancer especially pulmonary and pancreatic, dehydration, shock, heart failure, myocardial infarction, polycythemia vera, anemia, advancing age, bed rest, obesity, pregnancy and the puerperium. A perusal of this list reveals that slowed circulation or stagnation is common to most of the conditions, that hypoxia prevails in others and that perhaps some enzymatic change in the cir-

culating protein moieties of the blood may be held responsible in certain instances, as in carcinoma of the pancreas and lung.

Bland thrombosis usually originates in the deep veins of the legs although occasionally other sites may be involved. The more inflammatory types of thrombophlebitis, which may vary from low-grade nonbacterial inflammation of the vein to full blown suppurative lesion, are best known to occur in superficial veins of the legs, in pelvic veins, in the appendiceal vein (pylphlebitis) and in veins accompanying the arteries in thromboangitis obliterans (Buerger's disease).

Microscopic examination of the site of clinically bland phlebothrombosis reveals that after forty-eight to seventy-two hours this lesion is indistinguishable from actual thrombophlebitis (Fig. 4). Thus strictly speaking, the term phlebothrombosis applies only to lesions of perhaps less than four days duration.

Suppurative thrombophlebitis may occasionally follow postpartum or postabortal sepsis. So-called migratory thrombophlebitis usually involves only the superficial veins and

thigh and foot. This examination should be done with only moderate pressure between the bellies of the gastrocnemius muscle in the popliteal space and along the medial and posterior aspects of the thigh. The knee is moderately flexed during palpation. The examiner must learn to distinguish between the response to abnormal degrees of pain on the one hand, and the discomfort produced by too strenuous palpation on the other. This sign is not very reliable since other causes for calf tenderness may confuse the picture.

5. *Homan's sign*, or dorsiflexion test. This maneuver consists of dorsiflexing the foot of the patient while the knee is extended. If dorsiflexion of the foot elicits pain in the calf or popliteal area the reaction to the test is positive. However, as in the palpation maneuver the test is not pathognomonic for thrombosis.

6. *Sphygmomanometer cuff pain test* (Lowenberg). The pneumatic cuff of the sphygmomanometer is placed about the calf or thigh and slowly inflated. Normally patients do not register discomfort at pressures below 180 mm. Hg on the calf or thigh. In the presence of thrombosis the patient complains bitterly of pain at a pressure significantly below the normal. At this point the cuff is immediately deflated and the test is repeated on the other calf. The patient should not be alerted to the possibility of pain but should be watched for alteration of facial expression or a withdrawal reaction, such as flexing the thigh. This test appears to be the most sensitive and perhaps the most valuable of the diagnostic procedures available today to detect early bland thrombosis in the legs.

7. *Elicitation of pain on calf muscle rock*. This is an acceptable clinical test perhaps somewhat more sensitive than the palpation test, but not as reliable as the cuff test. The knee is slightly bent with the heel resting on the bed. The calf muscle group is rocked back and forth in its relaxed state. Severe pain or discomfort is suggestive of thrombosis.

8. *The cough pain test* (Lawrence). The reaction to this test may occasionally be suggestive of thrombosis. Local pain on coughing has been claimed to be of some value especially in acute thrombophlebitis.

9. *Search for other local signs* such as superficial venous congestion, dilated pretibial veins, increased local heat, redness and swelling, cyanotic mottling and other similar indications. These may be present when the sign is pronounced, but as

a rule they are absent in most patients with early bland phlebothrombosis.

Prognosis of Thrombophlebitis of Lower Extremities. Thrombophlebitis may progress along one of four lines eventually no residual detectable sign of the original disease may be found chronic venous insufficiency of the involved leg may give symptoms for life death from pulmonary embolism may occur pulmonary infarction may follow nonfatal pulmonary embolism.

No residual detectable lesion. Probably in the majority of the milder cases of thrombophlebitis of the lower extremities the disease subsides completely in due time, in some cases because of good treatment, in others without adequate treatment. In the latter group are perhaps many originally unrecognized cases. In many patients mild residual edema of one extremity with or without pain will eventually subside completely. It must be remembered however that one attack predisposes to another particularly after another surgical operation or injury or recurrent illness such as heart failure. Consequently a painstakingly careful history regarding leg symptoms after previous surgical procedures or illnesses is mandatory. Only by such screening can prophylactic measures be properly instituted.

Chronic venous insufficiency (postphlebotic syndrome). In a large percentage of patients the first indication of postpartum or postoperative thrombosis is the development of a pale swollen painful leg as ambulation is resumed postoperatively. This type of limb



Figure 7 Phlegmasia alba dolens (milk leg)

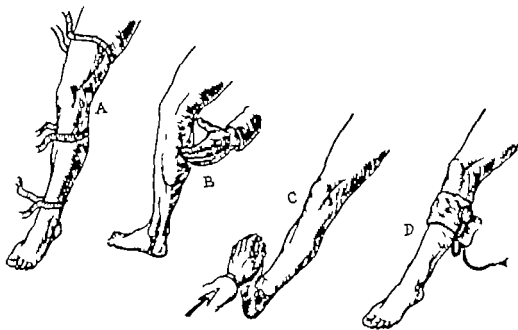


Figure 6 Some useful maneuvers to detect phlebothrombosis. A, Circumferential measurements using fixed sites on limbs. B, Palpation for tenderness between bellies of gastrocnemius muscle and popliteal area. C, Homans' sign or dorsiflexion test. D Sphygmomanometer cuff pain test (Lowenberg) on calf or thigh.

100 patients with pulmonary embolism are on medical services the remaining forty coming from surgical services. In 20 to 25 per cent of the patients the first embolus is the fatal one. From 53 to 94 per cent of patients who die of pulmonary embolism, or who exhibit pulmonary embolism at post mortem examination, were not known to have had any thromboembolic disease. In one series, the first signs of thrombosis were recognized in 74 per cent of patients only after massive pulmonary embolism.

It is obvious from these figures that significant numbers of patients have thrombotic disease without its being recognized. Therefore the true incidence of phlebothrombosis is not known. More important is the obvious inability on the part of the clinician to diagnose the condition early enough.

Clinical Picture of Phlebothrombosis and Thrombophlebitis. Superficial thrombophlebitis is ordinarily easy to diagnose because of the readily observed local signs such as redness, local heat and subjective pain, supplemented by the usual systemic signs of inflammation, such as fever, tachycardia, rapid sedimentation rate and leukocytosis. The systemic signs vary in degree according to the amount of inflammation.

The diagnosis of deep vein thrombosis of the lower extremities is much more elusive and, as mentioned, the lesion often goes unrecognized. The diagnosis can sometimes be derived inferentially by an otherwise unexplained occurrence of the systemic signs

listed above for thrombophlebitis. When such signs appear they are usually soon supported by the detection of local signs of venous thrombosis or by those of pulmonary embolism.

The incidence of phlebothrombosis in a given institution is usually proportional to the diligence with which the condition is searched for. If daily examination of the legs is carefully carried out on both the surgical and medical services, the number of detected cases will be higher than if such examinations are not done.

The routine examination for silent deep vein thrombosis includes the following (Fig. 6):

1. Precise questioning about soreness or discomfort in the legs. Unless specifically questioned along these lines, at least half of all postoperative patients fail to mention spontaneously aches in the calf muscles or other comparable minor symptoms.

2. Inspection of legs for slight changes in color. Incipient cyanosis of the skin may not be obvious unless the legs are dependent, as in sitting or standing. Comparison of one leg with the other makes for more accurate judgment than examining each leg separately. This test is not reliable in the earliest cases of bland thrombosis.

3. Measurement of the circumference of the legs at given points in the thigh, calf and ankle and, most important, comparison with preoperative measurements.

4. Palpation for tenderness in the calf

thigh and foot. This examination should be done with only moderate pressure between the bellies of the gastrocnemius muscle in the popliteal space and along the medial and posterior aspects of the thigh. The knee is moderately flexed during palpation. The examiner must learn to distinguish between the response to abnormal degrees of pain on the one hand and the discomfort produced by too strenuous palpation on the other. This sign is not very reliable, since other causes for calf tenderness may confuse the picture.

5. *Homan's sign*, or dorsiflexion test. This maneuver consists of dorsiflexing the foot of the patient while the knee is extended. If dorsiflexion of the foot elicits pain in the calf or popliteal area, the reaction to the test is positive. However, as in the palpation maneuver, the test is not pathognomonic for thrombosis.

6. *Sphygmomanometer cuff pain test* (Lowenberg). The pneumatic cuff of the sphygmomanometer is placed about the calf or thigh and slowly inflated. Normally, patients do not register discomfort at pressures below 150 mm. Hg on the calf or thigh. In the presence of thrombosis, the patient complains bitterly of pain at a pressure significantly below the normal. At this point the cuff is immediately deflated and the test is repeated on the other calf. The patient should not be alerted to the possibility of pain but should be watched for alteration of facial expression or a withdrawal reaction, such as flexing the thigh. This test appears to be the most sensitive and perhaps the most valuable of the diagnostic procedures available today to detect early bland thrombosis in the legs.

7. *Elicitation of pain on calf muscle rock*. This is an acceptable clinical test, perhaps somewhat more sensitive than the palpation test, but not as reliable as the cuff test. The knee is slightly bent with the heel resting on the bed. The calf muscle group is rocked back and forth in its relaxed state. Severe pain or discomfort is suggestive of thrombosis.

8. *The cough pain test* (Lawrence). The reaction to this test may occasionally be suggestive of thrombosis. Local pain on coughing has been claimed to be of some value especially in acute thrombophlebitis.

9. Search for other local signs, such as superficial venous congestion, dilated pretibial veins, increased local heat, redness and swelling, cyanotic mottling and other similar indications. These may be present when the inflammatory element is pronounced but as

a rule they are absent in most patients with early bland phlebothrombosis.

Prognosis of Thrombophlebitis of Lower Extremities. Thrombophlebitis may progress along one of four lines eventually no residual detectable sign of the original disease may be found, chronic venous insufficiency of the involved leg may give symptoms for life, death from pulmonary embolism may occur, pulmonary infarction may follow nonfatal pulmonary embolism.

No residual detectable lesion. Probably in the majority of the milder cases of thrombophlebitis of the lower extremities the disease subsides completely in due time in some cases because of good treatment in others without adequate treatment. In the latter group are perhaps many originally unrecognized cases. In many patients mild residual edema of one extremity with or without pain will eventually subside completely. It must be remembered, however, that one attack predisposes to another particularly after another surgical operation or injury or recurrent illness such as heart failure. Consequently a painstakingly careful history regarding leg symptoms after previous surgical procedures or illnesses is mandatory. Only by such screening can prophylactic measures be properly instituted.

Chronic venous insufficiency (postphlebotic syndrome). In a large percentage of patients the first indication of postpartum or postoperative thrombosis is the development of a pale, swollen, painful leg as ambulation is resumed postoperatively. This type of limb



Figure 7 Phlegmasia alba dolens (milk leg)



Figure 8 Some sequelae of deep vein thrombosis. A, Chronic swelling with discoloration of skin of lower third of leg. B, Parchment-like appearance of skin. C, Ulceration. D, Secondary varicosities and temporarily healed ulcer.

has been referred to as *phlegmasia alba dolens* or more commonly milk leg (Fig. 7). The lowered surface temperature of such a limb is apparently due to reflex arteriospasm which commonly accompanies venous occlusion. This condition may gradually spontaneously subside or it may persist for life.

If the condition progresses, the venous blockage results in development of other more serious sequelae (Fig. 8). Secondary varicosities are usually the result of shunting of the major portion of the venous return to the superficial leg veins. As a rule, this is more often the result of incompetency of the valvular system of the deep veins than of persistent blockage. As the deep vein thrombus becomes organized, the vein wall and its valves become an integral, inseparable part of the conglomerate thrombus. In due time, recanalization occurs, often with multiple channels, but the blood in the new channels is no longer aided along its course by valves. As a consequence a high degree of stasis develops in the lower third of the leg. The skin and underlying tissues become poorly oxygenated, stasis thrombi form in small subcutaneous veins and the skin about the malleoli loses its normal color and consistency. It may become shiny and thinned.

Induration may begin in a small area overlying a perforated vein and involve skin and underlying fat. Fibrosis of the underlying fat either accompanies or follows induration of the skin. The skin gives the appearance of parchment paper under which the fatty layer is atrophic and hard.

The least trauma may result in chronic ulceration. Episodes of acute cellulitis or infected dermatitis may supervene. When associated lymphatic thrombi occur the skin becomes thickened and leathery and in extreme instances elephantiasis may develop.

The patient unfortunate enough to suffer from chronic venous insufficiency with the manifestations mentioned usually must be resigned to lifelong care of the limb.

In decreasing order of frequency the post phlebotic leg exhibits edema, pigmentation, secondary varicosities, pain, dermatitis, induration and ulceration. Most of these sequelae can be obviated if proper early treatment is instituted and good care is taken of the leg afterward.

Pulmonary thromboembolism. While death from pulmonary embolism occurs in an extremely small percentage of patients with thrombotic disease of the lower extremities, it is likely to follow bland phlebothrombosis. Since as stated, phlebothrombosis often goes unrecognized, pulmonary embolism usually occurs without warning. As mentioned previously, an overwhelming proportion of patients who die of pulmonary embolism are not known to have had clinically demonstrable thromboembolic disease, nor did they show signs or symptoms requiring therapy prior to the fatal embolism. Not all large pulmonary emboli are fatal. Some of them, particularly smaller pulmonary emboli, may produce pulmonary infarction. However in 20 to 25 per cent of cases, the first embolus is the fatal one.

Animal experimentation has demonstrated that nonfatal pulmonary emboli do not invariably produce infarcts. Pulmonary infarction following pulmonary embolism is apparently dependent on the presence of other factors, such as diminished aeration, interference with blood supply and infection. If none of these factors is present, an embolus may organize in the pulmonary vessels and produce no demonstrable effects.

Certain concepts have been developed from the search for factors which may precipitate the embolization of a thrombus. For example in the presence of anemia, clot retraction following coagulation is known to be greater than normal. It is therefore

possible that exaggerated clot retraction if it occurs following thrombosis might tend to draw the clot away from contact with the vessel walls and permit it to be dislodged by the passing stream of blood. Excessive clot retraction occurs in either chronic anemia or in the anemia following acute blood loss from surgery.

Another concept of embolization is based on the nature of thrombus propagation. The oldest portion of the clot adheres to the intima, while the clot propagates by adding a soft tail of freshly clotted blood elements. The tail is only loosely attached over a small area to the adherent portion, but its largest surface area, exposed as it is to the stream of venous blood, makes ready ground for deposition of circulating platelets, cells and other clotting elements. The tail of a thrombus may later become attached to the vessel wall. But any sudden increase in venous pressure, as may accompany coughing, sudden motion, straining at a stool or any other physical expression of the Valsalva maneuver, may easily cause the tail to break from its mooring and become an embolus. Similarly if the tail of the thrombus extends past a branching vessel which brings blood from a neighboring part the current from the branch may sweep off a portion of the soft clot.

The classical clinical picture of pulmonary embolism is the sudden onset of constricting chest pain, painful breathing, anxiety, shock and cyanosis. In a few patients, hemoptysis or cough with frothy pink sputum may occur. Rib cage tenderness, pleural friction rub and an x-ray shadow suggestive of infarction in the presence of these symptoms are prima facie evidence of pulmonary infarction. Tachycardia, fever and leukocytosis are later sequential phenomena. Early clinical differentiation from acute coronary occlusion may be difficult.

Pulmonary embolism may cause death within a few moments; however several hours may elapse with the patient in a state of shock, with or without loss of consciousness, before death supervenes. It is in such patients that certain therapeutic measures may be attempted.

Electrocardiographic findings immediately after an attack are not easily differentiated from those due to posterior coronary vessel occlusion. Later the changes of acute cor pulmonale are usually demonstrable.

In general, it can be said that a massive pulmonary embolism is usually lethal. Small or emboli usually result in infarction with

pleuritis often with effusion. Many smaller emboli result in no demonstrable pulmonary pathologic process, provided the lung is not previously diseased.

A "paradoxical arterial embolism" occurs when a venous embolism passes through a patent foramen ovale from the right to the left side of the heart and into the arterial system. Such an embolus may lodge in a femoral vessel, cerebral vessel or other peripheral artery.

Treatment Prophylaxis of venous thrombosis. No known test or method can accurately foretell whether a patient will or will not have venous thrombosis. The recently introduced antithrombin and heparin tolerance tests have not proved reliable. Nonetheless, there are some measures which can be undertaken to reduce the risk. Also the realization that thromboembolism may occur under certain circumstances should serve to alert the surgeon to identify and treat phlebothrombosis if possible.

Since early recognition of thrombosis is the only hope of preventing embolism, daily examination of the legs, as previously outlined in all postoperative patients and in severely ill medical patients, is mandatory.

Prophylactic measures during surgery include prevention of shock and maintenance of effective circulating blood volume when blood loss is great. Postoperatively venous stasis in the legs may be discouraged by such measures as prevention of excessive abdominal distention, avoidance of tight abdominal binders and by not permitting prolonged periods of immobility or dependency of the legs.

Elastic stockings or bandages compress the superficial veins and thus tend to cause more rapid flow of venous blood through the deep veins. As a result, this measure is excellent prophylaxis against deep vein thrombosis. If elastic bandages are used they must be applied carefully and reapplied daily. Elastic compression is particularly useful in patients with varicosities. The elastic stockings or bandages can be applied prior to surgery and worn by the patient during and after surgery. Of course, in the presence of inflammation or ischemia, elastic compression cannot be tolerated by the patient because of the local pain.

Early ambulation after surgery is undoubtedly a highly important preventive measure against thromboembolism. If ambulation cannot be carried out for any reason, a definite routine of gentle leg massage, leg exercises, and periodic deep breathing may be insti-

tuted. All such measures are aimed at preventing venous stasis in the legs.

The prophylactic postoperative use of anticoagulant drugs such as heparin or bishydroxycoumarin (Dicumarol) has its adherents and its opponents. Those who favor this therapy apply it to all patients with a past history of thrombosis or embolism. In order not to risk hemorrhage, the anticoagulant effect is not instituted until forty-eight to seventy-two hours after surgery. The opponents claim that application of the method may risk hemorrhage and is probably not practical, since the onset of thrombosis usually occurs during surgery or within twenty-four hours afterward; hence they claim the late institution of anticoagulant therapy does not actually prevent the formation of thrombi and that after cessation of therapy embolization may still occur.

Active treatment Inasmuch as the two outstanding features of thrombosis are pulmonary embolism and disability due to postphlebitic syndrome, therapy is aimed at prevention of each of these complications once the thrombosis is discovered.

If the diagnosis is *deep vein phlebothrombosis or thrombophlebitis*, it is customary to wrap the legs in elastic bandages from toes to groin and immediately institute anticoagulant therapy. At this time the legs are neither elevated nor lowered but are kept flat in bed. Jack-knifing the patient to a full sitting position is to be avoided since this may encourage venous pooling or stagnation at the hip level.

Anticoagulant therapy is begun with heparin for immediate response and at the same time a coumarin derivative is administered orally. The latter drug usually takes forty-eight hours for full effect upon the prothrombin. When prothrombin levels reach therapeutic ranges the heparin is withdrawn. Careful check on both coagulation time and prothrombin time is made each day. Anticoagulant therapy is carried on somewhat longer than the week of bed rest. Therapy may cover a period of ten to fourteen days. In some instances anticoagulants are continued up to three weeks.

If embolic phenomena occur despite the treatment outlined above, bilateral femoral vein ligation below the saphenofemoral junction is carried out with the patient under local anesthesia. If a thrombus is found at the site of proposed ligation, it may be necessary to do a thrombectomy or occasionally a vena cava ligation. For a number of years in certain institutions, bilateral femoral vein liga-

tion was carried out as the initial procedure upon discovery of thrombosis in the legs. Later comparison of results however indicated no significant difference between the incidence of embolism in these patients and that in patients given anticoagulant therapy. Hence the procedure is now reserved for patients who show signs of embolic phenomena despite anticoagulant therapy. The operation is usually done without restoring normal coagulation.

When a patient with deep vein thrombosis is allowed to become ambulatory, he is fitted with elastic stockings from toes to knees to minimize the edema.

If the diagnosis is *acute inflammatory thrombophlebitis of the deep or superficial veins of the leg*, initial treatment is similar to that outlined above except that elastic bandages cannot be used immediately because of the discomfort they cause. Curiously, in the acute stages, such patients usually require much larger doses of anticoagulant drug than the usual ones in order to elevate their coagulation time to therapeutic ranges. Another useful measure in the presence of severe pain, usually due to distention of veins in spasm, is to block the sympathetic impulses to the legs. If this is done by paravertebral sympathetic ganglionic block, it is best to begin the block before instituting anticoagulant therapy. Other methods of blocking the sympathetics include continuous caudal block and the use of parenteral ganglionic blocking agents, such as hexamethonium or related compounds. Antibiotics have been used empirically together with anticoagulants, but their role remains to be evaluated since most of these lesions are not caused by bacterial invasion. If only superficial veins are involved, the affected segments may be excised once the early acute inflammatory stage has subsided. Pulmonary embolism is not a common sequel to this lesion.

Ambulation is usually not attempted before a week or ten days or as soon as it is possible to wear elastic stockings.

Massive venous occlusion. In rare instances massive thrombosis occludes practically all the veins of the leg, including the main stems. When this occurs the entire leg up to mid thigh becomes deeply cyanotic and extremely painful and swollen within a few hours. A patient with such a lesion may go into shock from blood loss into the extremity. Treatment consists of elevation, with knee bent, immediate anticoagulant therapy and intravenous administration of fluids and

blood to treat the shock. If it is present. Such treatment may be rewarded by gradual return of almost normal color to the skin within hours. In an occasional patient the limb may become gangrenous and require amputation despite good treatment. Sympathetic block is usually of no avail in these patients.

Chronic venous insufficiency (postphlebotic syndrome) The chronic sequelae of thrombophlebitis present one of the most difficult therapeutic problems in surgery.

For patients whose skin is not ulcerated but whose main symptoms are pain on standing and edema with or without skin discoloration, certain precautions must be taken. These individuals are instructed to avoid even minor trauma to the area of the ankles and lower legs, not to stand for a long time when sitting for long periods, to keep the legs elevated when walking, to wear elastic stockings, not to rub the skin of the legs too vigorously when drying after bathing, to apply linolin ointment to the skin of the ankles before retiring at night and if obese to go on a strict weight reduction regimen.

If varicosities are present the great saphenous vein, and short saphenous if indicated, should be stripped and individual clusters of veins excised. The rationale for this procedure in the presence of chronic deep vein insufficiency is based on the supposition that venous return from the legs will be hastened through deep, uninvolved veins as well as recanalized veins. If the stagnant superficial system is removed.

If ulceration occurs treatment depends upon the extent of tissue destruction. Superficial, small ulcers (Fig. 9) usually heal with a few days of bed rest with elevation and sterile saline dressings. However, as ambulation is resumed, the ulcers may recur unless the superficial venous system is stripped or proper elastic compression is applied.

Deep infected ulcers often present a serious problem in therapy. Local application of antibiotic preparations is usually of no avail unless the basic principles of elevation, compression and cleanly care are applied. Purgent, pseudomembranous granulation tissue can usually be converted into clean red flat surfaces without the use of antibiotics, enzymatic debriding agents or other chemical means. In some patients a split thickness skin graft can be applied to such healthy granulations. In others, surgical excision of the ulcerous area and neighboring poor tissue, of ten including the underlying fascia, may be necessary after attempts to clean the ulcer base have been carried out. It is known that

granulation tissue which appears healthy grossly is most receptive to grafting when its pH is over 7.4.

Occasionally the skin continues to break down repeatedly after healing with or without a graft despite the measures outlined. Various modes of treatment have been prescribed for such patients including sympathectomy, ligation of the popliteal vein or femoral vein and extensive excision of all superficial veins of the leg through long flap incisions. These measures have yielded good results in some patients.

The largest percentage of milder ulcers can be healed while the patient is ambulatory by means of weekly or semiweekly application of Unna's paste boot after painting the ulcer with gentian violet or a similar dye. The rationale for this treatment is one of compression, protection and maintenance of moisture and softness in the area. The boot consists of wrapping the leg, from the base of the toes to the knee, with gauze impregnated with a zinc oxide-gelatin-glycerin mixture. The method permits healing over a number of weeks or months. It has been replaced by and large by the more direct, shorter term treatments mentioned above.

Infected dermatitis pursuant to chronic venous insufficiency is usually best treated by identification and eradication of sec-



Figure 9 Postphlebotic syndrome. Appearance of skin of leg immediately after healing of ulcer. Wrinkles in skin denote reduction in edema. Note tissue-paper thinness (parchment appearance) of skin.

ondary factors, such as epidermophytosis, sensitization to various allergens, secretions, medication or bacterial invasion. As stated, most patients, nonetheless, will respond to elevation, clean care and, later compression.

It is important to impress upon the patient that despite the success of specific immediate therapy lifelong care of the limb is mandatory.

Thromboembolism Death from massive pulmonary embolism often supervenes within seconds of the embolization. Some patients may survive several hours or even days. In roughly 50 per cent of patients so afflicted there is time to apply one or more of the emergency measures.

Since the first of these measures is immediate administration of oxygen by mask, it is always best to have oxygen readily available on surgical wards. The subcutaneous injection of atropine sulfate, $\frac{1}{60}$ grain is advised to overcome reflex vagal effects if they are

present. The intravenous or subcutaneous administration of papavarine, $\frac{1}{4}$ grain, has been recommended to overcome bronchial spasm and to counteract ventricular fibrillation. Immediate heparinization is also advised.

Because one cannot predict which patients will survive no valid statistics are available as to the efficacy of this form of treatment.

In a few centers in the world, pulmonary embolectomy has been successfully carried out. Obviously the results are better in the slowly fatal type in which there is time to perform thoracotomy, expose the pulmonary artery and remove all its contained blood clot. With the proper selection of patients, such drastic surgical measures may save an occasional life.

THROMBOSIS OF THE UPPER EXTREMITIES

Thrombophlebitis of the superficial veins, especially the antecubital vein, commonly



Figure 10 Venogram showing thrombosis of axillary vein. Note collateral channels t-area of occlusion.

follows intravenous infusions. It may be due to hypertonicity or irritability of the solution, contamination or intimal irritation from the needle.

As a rule the area is painful and tender. The overlying skin is red and warm. The vein usually can be palpated as a firm cord under the skin. Moist hot packs usually cause the condition to subside in a few days.

Thrombosis of the axillary and subclavian vein may arise from a variety of causes. Direct trauma and prolonged compression are known etiologic factors. So-called effort thrombosis usually follows some unusual physical exertion involving compression of the vein by the subclavian muscle tendon or by the scalenus anticus muscle. In addition, fractures, hematomas, tumor growths and postoperative mastectomy compression have been responsible for axillary vein thrombosis. The axillary vein may also become involved in phlebitis migrans.

Symptoms are usually ushered in with pain at the site of thrombosis. In twenty-four to forty-eight hours the arm appears somewhat swollen and the skin assumes a burgundy color. Cyanosis of the deep blue variety may occur. Superficial veins become dilated. The arm feels heavy and full to the patient.

Strangely, the incidence of pulmonary embolism following upper extremity venous thrombosis is extremely low. Collateral channels about the axilla and shoulder are rich and soon assume the added burden of venous circulation, becoming visibly dilated.

A diagnostic aid is the injection of radiopaque medium into an arm vein in order to visualize the site of the occlusion on an x-ray film (Fig. 10). This should not be done for at least forty-eight hours in order not to dislodge the clot. Diagnosis without venography is preferred.

Treatment depends upon the location of the thrombosis and the severity of the symptoms. If symptoms are minimal, simple elevation of the arm and anticoagulant therapy for seven to ten days may suffice. Sympa-

thetic and stellate ganglionic blocks with 1 per cent procaine solution may be carried out to minimize spasm. If the vein occlusion is at the level of the scalenus anticus muscle, scalenotomy may be done as a late procedure provided recanalization has been unsatisfactory. Thrombectomy is only rarely necessary. If symptoms persist after many months, sympathectomy may be performed to alleviate both arterial and venous spasm.

READING REFERENCES

- Butcher, M. B. Jr., and Hoover, A. L.: Abnormalities of Human Superficial Cutaneous Lymphatics Associated with Stasis Ulcers, Lymphedema, Scars and Cutaneous Autografts. *Ann. Surg.* 142:632, 1955.
- DeBakey, M. E.: A Critical Evaluation of the Problem of Thromboembolism. *Surg. Gynec. & Obst.* (Internat. Abst. Surg.) 88:1, 1954.
- de Takats, G.: Postphlebitic Syndrome. *J.A.M.A.* 161:1601, 1957.
- Freed, P. H., Perilstein, P. K., and Wagner, F. B., Jr.: The Hormonal Cause of the So-called Varicose Veins of Pregnancy. *Arch. Surg.* 72:253, 1958.
- Homans, J.: Thrombosis of the Deep Veins of the Lower Leg Causing Pulmonary Embolism. *New England J. Med.* 211:993, 1934.
- Huxley, H. H.: Venous Thrombosis of the Lower Extremities. *GP* 10:58, 1954.
- Laufman, H.: Ancillary Care in Postphlebitic Syndrome. *S. Clin. North America* 39:183, 1959.
- Lawrence, E. D.: The Cough-Pain Sign in Acute Superficial Thrombophlebitis. *J. M. Soc. New Jersey* 47:164, 1950.
- Lowenberg, R. I.: Early Diagnosis of Phlebothrombosis with Aid of a New Clinical Test. *J.A.M.A.* 135:1566, 1934.
- Ochsner, A., and DeBakey, M. E.: Therapeutic Considerations of Thrombophlebitis and Phlebothrombosis. *New England J. Med.* 235:207, 1941.
- Paterson, J. C., and McLachlin, J.: Precipitating Factors in Venous Thrombosis. *Surg. Gynec. & Obst.* 98:96, 1954.
- Roach, H. D., and Laufman, H.: Relationship between Pulmonary Embolism and Pulmonary Infarction: An Experimental Study. *Ann. Surg.* 142:82, 1955.
- Zimmerman, L. M., Miller, D., and Marshall, A. N.: Pulmonary Embolism: Its Incidence, Significance, and Relation to Antecedent Vein Disease. *Surg. Gynec. & Obst.* 88:373, 1949.

The Arteries

By HARRIS B SHUMACKER, JR., M D

HARRIS B SHUMACKER, JR. is a Mississippian who was educated at the University of Chattanooga, Vanderbilt University and Johns Hopkins University Medical School. From Yale University he returned to Hopkins then back to Yale and now he is the Professor of Surgery at the Indiana University Medical School. His interest in vascular surgery is evidenced by his basic contributions to that field.

The arteries and arterioles serve as conduits for the transport of blood from the heart to the capillaries where certain constituents of the blood are released into the surrounding tissue spaces and certain metabolites pass across the capillary membrane into the blood. They are also of importance in the regulation of blood pressure.

Natural arteries are not rigid tubes but are capable of wide variation in caliber. Spasm or constriction of these vessels may occur as the result of reflex sympathetic response or from certain direct stimuli such as trauma and cold. Though all sizes of arteries are capable of such alterations, the smaller arteries and arterioles undergo more pronounced changes as a consequence of sympathetic stimulation or paralysis than do larger arteries. Furthermore, the arteries supplying the skin, subcutaneous tissue, small muscles and viscera appear to undergo wider changes with sympathetic activity or paralysis than do those supplying large muscle masses. These variations in the tone of arteries and arterioles are of importance not only in determining blood flow into a given part of the body but also in regulating central blood pressure. The sympathetic control of blood vessel tone provides us with one of the most useful tools for dealing with ischemic disorders, namely sympathetic paralysis by infiltration anesthesia or operative denervation.

Most of the problems relating to the arteries concern difficulties with flow of blood into extremities. The various disorders can ordinarily be sorted out with considerable accuracy by carefully obtaining a history and carrying out a complete physical examination. A family history of diabetes, hypertension or arteriosclerosis is important. One must

inquire into the patient's history for symptoms suggestive of diabetes, hypertension, cardiac disorders and local cold injury. The use of tobacco and the character of the patient's work are of interest. One should try to determine whether the patient has had high, low or average vascular tone. The description of the present illness should be obtained in an orderly sequential fashion. Circumstances under which symptoms are present should be noted, for example, whether at exercise or at rest. Any variations observed with changes in environmental circumstances are of importance. The common vasospastic disorders may produce symptoms more or less continuously as is true of acrocyanosis, or as in Raynaud's disease, episodically. The common symptoms are coldness of the hands or feet, color changes such as pallor or cyanosis, hyperhidrosis and on occasion numbness and tingling. Digital ulceration and areas of superficial gangrene may occur but extensive gangrene is rare. The chronic obliterative diseases produce symptoms both at rest and with exercise. One of the commonest complaints is intermittent claudication, which is the onset of muscle fatigue or aching after walking a given distance with prompt relief of symptoms after a momentary period of rest and return of symptoms again after the same amount of exercise. In any case, intermittent claudication occurs sooner after walking rapidly uphill or against the wind for a shorter distance. The walking distance is greater when patients walk slowly or downhill. Patients may also complain of numbness of the feet after exercise. At rest the patient may notice coldness of the feet or hands, pallor or pain. The pain is particularly apt to be present when the patient is recumbent.

and the beneficial effects of gravity are not at play. Edema is likely to develop in ischemic limbs kept dependent and inactive for long periods.

The examination should include a careful general survey. It is important to evaluate the cardiac status, determine the blood pressure and carry out funduscopic study of the retinal vessels. The extremities should be inspected thoroughly. Any atrophy of muscles or atrophic changes in nails, hair or skin should be noted. It is important to observe whether the skin is dry, normally moist or excessively wet. The presence of normal color, pallor or rubor is important. Pallor on elevation above heart level and rubor in the dependent position are indicative of occlusive arterial disease. The degree of filling of the superficial veins of the hands and feet is important, as is the rapidity or slowness with which these vessels fill when the extremity is made dependent after a period of elevation (venous filling time). Fissures, calluses, ulcers and areas of superficial or deep gangrene must be noted. Superficial phlebitis and other venous abnormalities may be of importance. One should determine whether the extremities are normally warm, excessively hot or cool and whether any asymmetry with regard to temperature exists. The environmental circumstances under which the examination is conducted are of great importance with respect to color, temperature, sweating and the quality of the pulses. All the peripheral pulses should be felt. It is not sufficient to palpate the radial and dorsal pedal pulses. The brachial, ulnar, abdominal, aortic, iliac, femoral, popliteal and posterior tibial pulses must be examined as well. In a small percentage of normal individuals, dorsal pedal pulses are not felt, but the posterior tibial pulses are always normally present. The ulnar pulses are more commonly obliterated by disease than are the radial. It is important to note whether the arteries palpated are soft and compressible or firm, hard and beaded. One should detect aneurysmal dilatation if present and, when aneurysm, arteriovenous fistula, dilatation or partial constriction of an artery is suspected, one should feel for a thrill and listen for a bruit.

The degree of vascular tonus, the presence of vascular spasm and the capacity for vasodilatation can be estimated in a variety of ways. Changes noted in a warm and cold environment are of importance. A varying degree of release of vascular constriction and induction of vasodilatation may be noted when the patient is placed in a warm envi-

ronment, given a general or spinal anesthetic, subjected to immersion of one or more extremities in warm water, given diathermy over the abdomen and pelvis treated by the administration of an autonomic blocking agent or subjected to a somatic peripheral nerve block with procaine or to procaine infiltration of the regional sympathetic chain. These measures vary in their effectiveness in blocking vasoconstriction and inducing vasodilatation. The most reliable method is the carrying out of an effective sympathetic block with procaine. Another good method is the blockade with procaine of a somatic nerve such as the ulnar or posterior tibial. Vasodilatation is associated with better warmth, venous filling and color. When sympathetic blockade is effected, sweating ceases and the cutaneous resistance increases. Tests carried out in order to determine the capacity for vasodilatation should be performed in a cool environment. In evaluating the possible effect of sympathectomy upon pain by procaine sympathetic block, the patient should be subjected as soon as an effective injection is carried out to those circumstances most conducive of pain in the case under study—lying down, walking or exposure to cold.

Reactive hyperemia, or the response to a period of deprivation of arterial circulation is a simple test which gives one some insight into the adequacy of capillary circulation. It is usually performed by excluding the blood flow into the forearm or leg for a period of from three to five minutes with a sphygmomanometric cuff. After release of the pressure from the cuff, one notes the brilliance, extent, and rapidity of appearance of the resultant flush. In patients with good capillary blood flow, the flush is full and extends rapidly to the tips of the digits. A slow or incomplete flush indicates poor capillary circulation. The test can be carried out with the extremity horizontal or elevated a certain number of degrees above the heart level. The higher the elevation at which an adequate flush is observed, the better the circulation. The same test can be modified to study collateral blood flow about a given segment of a major arterial stem (the Matas test). This is of value in patients with aneurysm or arteriovenous fistula or in any in whom a contemplated operation may entail ligation of a major artery. The test is carried out in the usual manner except that the artery in question is carefully and completely occluded by digital pressure during the latter part of the period of exclusion of arterial inflow and for several min-

utes afterward. If under these circumstances, a good full flush develops in less than two minutes, one has reason to assume that the collateral circulation is adequate. Since, in patients with *obliterative occlusion of a radial or ulnar artery* the collateral circulation is ordinarily limited, a simple type of hyperemia test usually serves to confirm or disprove suspected absence of blood flow through a radial or ulnar artery (Allen's test). After both the radial and ulnar pulses are shut off by digital pressure, the patient squeezes the blood from his hand by making a tight fist. The digital pressure is released from the artery in question. If it is not patent, a flush fails to develop or is inadequate. If it is patent, a good flush occurs during continued compression of the other artery.

Rarely are special instruments or tests required in order to establish the diagnosis or outline a proper course of management. The oscillometer gives one some objective measure of the quality of pulsations in the main stem arteries. It serves largely to confirm conclusions reached from careful palpation of pulses. The magnitude of pulsations in the digits can be recorded by the digital plethysmograph or by impedance plethysmography. Thermocouples can be used to determine exactly the temperature of the skin, but one can train himself to judge temperature fairly accurately by palpation. Venous occlusion plethysmography permits reasonably good estimation of blood flow into a part. The rapidity with which radioactive sodium disappears from the site of its subcutaneous or intramuscular injection is related to the blood flow through the area. Arteriograms give a pictorial outline of the arterial tree (Fig. 11). They are of particular value in demonstrating the lower limit of a segmental occlusion and in establishing the adequacy of the distal arterial bed. These and other precise methods are of especial importance in investigative studies and in clinical evaluation of certain unusual or difficult cases. The trained observer can do a good job in the majority of cases without such special equipment and tests.

The management of the ischemic extremity is very important. When the circulation to an extremity is markedly impaired the comfort of the patient and, indeed, the survival or loss of the extremity may be dependent upon careful attention to details. If the circulation is badly impaired, it may be totally inadequate when the limb is in a position horizontal to or above the level of the heart. Under such circumstances it should be made de-



Figure 11 Femoral arteriogram showing segmental obstruction of superficial femoral artery

pendent to the heart level. Everything should be done to protect the ischemic hand or foot against injury. The pressure of bed clothes across the toes of a foot with markedly impaired circulation may be sufficient to initiate gangrene. The simplest and most effective way to obviate this difficulty is by use of a footboard so that the bed clothes do not press upon the feet. In general, it is wise to keep the toes separated by the use of cotton or thin layers of gauze. This is essential if there is maceration or if interdigital ulceration is present. Under certain circumstances, for example, acute arterial embolism, it may be advisable to use the anticoagulant drugs, heparin and the coumarin agents. One should avoid use of local heat. Hot water bottles, for example, may initiate gangrene and bring about the necessity for amputation.

Theoretically use of local cooling might aid in saving limbs threatened with gangrene, since local cooling of a part does decrease the metabolic requirements. Unfortunately local cooling also brings about still further reduction in arterial circulation since cold is one of the most effective stimuli for inducing vasoconstriction. Cold acts both directly and in a reflex manner to produce

vasospasm, and cold of significant degree or duration induces marked vasoconstriction even in extremities from which the sympathetic nerve supply has been removed.

Since it is impossible when cooling an extremity to be certain that the local metabolic requirements are being reduced proportionately more than the arterial inflow of blood into the limb and since indeed the reverse may be true, local cooling is not recommended as a measure for saving the ischemic extremity. Local refrigeration is an extremely useful tool for employment prior to amputation in patients in whom infection is present. If a limb is packed in ice or placed in a special refrigerating unit it soon becomes quite comfortable; a tourniquet can be placed tightly about it and for all purposes a physiologic guillotine amputation is accomplished. The general effects of the local infection disappear rapidly. Control of diabetes becomes remarkably easy and when the amputation is performed several days later, it can be carried out with much greater likelihood of primary healing. As long as there is any possibility of saving the ischemic limb, every effort should be made to improve the circulation to the part.

Obviously the most effective method of restoring good blood flow into the limb is to relieve any mechanical block of a main-stem artery which may be present. In the case of arterial embolus it is possible to remove the obstructing embolus. In segmental thrombosis, either as the result of trauma or arterial disease, one can often re-establish blood flow through the obstructed artery by thromboendarterectomy or a grafting procedure (see Fig. 17). When one is not dealing with segmental occlusion of a main-stem artery the most effective method for increasing the flow of blood into the hand or foot is to carry out a regional sympathetic denervation. Sympathetic denervation brings about permanent localized maximal, or near maximal, vasodilatation and thus the best possible blood flow through the patent arteries. The effectiveness of sympathectomy in relieving ischemia depends upon the number of patent arteries in the extremity and their capacity to undergo vasodilatation. Sympathectomy also produces anhidrosis in the area denervated (Fig. 12). Altogether this effect is a beneficial one in circulatory disorders. Active sweating contributes to loss of heat from the extremity by evaporation and thus to the need for greater circulation. Its elimination, therefore, somewhat reduces the circulatory requirements. Furthermore, excessive sweat

ing promotes maceration of tissues in some instances and may eventuate in ulceration or infection which may lead to the necessity for amputation. The dry skin of the sympathetomized extremity is ordinarily easy to manage. It can generally be kept in good condition by the application of lanolin to the dry areas each day. A third consequence of sympathetic denervation, the relief of reflex pain, is useful primarily not in diseases of the arteries but in those painful reflex disorders which follow trauma or inflammation.

Evidence obtained from carefully performed studies would indicate that the autonomic blocking agents, such as Priscoline and tetraethylammonium bromide have no part in the management of the severely ischemic extremity although such drugs are of considerable help in vasospastic disorders, particularly the mild ones and are invaluable in the management of hypertensive disease. When an extremity is without adequate blood flow as the result of an obstructive lesion such general blocking agents tend to produce vasodilatation in the other extremities in which there is no need for improve-

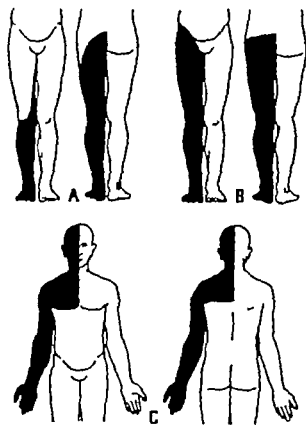


Figure 12 Drawings indicating the expected area of anhidrosis. A, Following removal of the second and third lumbar sympathetic ganglia. B, The first, second and third lumbar ganglia. C, Following preganglionic upper dorsal sympathectomy (Shumacker H. B. Surgery vol. 18)

ment of circulation and they are apt to produce trivial, if any improvement of circulation in the affected limb. Indeed not infrequently blood flow into the ischemic limb itself becomes worse as vasodilatation takes place in the normal or near normal extremities. The concomitant fall in blood pressure which sometimes takes place also tends to reduce the effectiveness of blood flow into the ischemic limb. Buerger's exercises may be of some help. They entail elevation of the hand or foot well above heart level until pallor is evident, followed by lowering the extremity below heart level until a flush develops. This maneuver is repeated a number of times and, when the limb is dependent, the digits, foot and ankle or the hand and wrist are exercised.

Wounds of Large Arteries. Wounds of large vessels constitute a real emergency. Objectives of treatment are the saving of the patient's life, of his injured limb and maintenance or restoration of as adequate circulation as is possible.

Bleeding must be brought under control without delay. The method will vary from case to case. Every effort should be made to stop the hemorrhage by some method which does not entail the use of a tourniquet. Digital compression is an ideal method but unfortunately can be used for only a relatively short time. Application of a firm compression dressing is sometimes effectual. On occasions it may be necessary to pack the wound itself with sterile gauze or preferably with some hemostatic absorbable material such as Oxy-coel gauze or Gelfoam. When packing is used, it should not be removed except under circumstances when the bleeding vessel can be dealt with in a definitive way and blood loss be replaced adequately in case hemorrhage recurs. An effective method for handling injured vessels of relatively little importance in the nutrition of the extremity is direct clamping with a hemostat. Clamping a main-stem artery however has the disadvantage that the local injury to the blood vessel may be increased. A tourniquet should be employed only when other methods cannot be utilized. For a tourniquet to be effective in stopping hemorrhage the extremity distal to the tourniquet is rendered entirely ischemic. Once applied, a tourniquet should never be released except by a physician prepared to replace immediately any blood that may be lost and to deal with the local vascular injury so as to prevent exsanguinating hemorrhage. When a tourniquet is left in place for a number of hours, the extremity may be

irreparably damaged by the prolonged ischemia and re-establishment of satisfactory blood flow may be impossible because of resultant thrombosis in the arterial tree. Release of the tourniquet after a long period may be attended by massive loss of fluids into the limb with the onset of tourniquet shock. If a tourniquet is used in a hopeless situation in which amputation obviously will be required, it should be placed as far distally as possible.

Simultaneously with efforts to control hemorrhage, treatment for blood loss should be carried out with the aim to prevent shock or to treat it if already established. It is preferable to use whole blood for replacement and, if blood is not available, plasma or a plasma expander such as dextran. If none is obtainable, glucose or saline solution may be of help for a time in maintaining adequate circulation. If shock is present, any narcotic required for the relief of pain should be given intravenously rather than subcutaneously.

The initial efforts to control the bleeding and replace blood loss should be carried out immediately. At the same time, plans should be made to expedite definitive treatment of the blood vessel injury. The treatment of the vascular injury depends upon the specific artery involved and the nature of the damage to it, the presence or absence of continued or recurrent bleeding, the presence or absence of ischemia and the character of any associated injury to the extremity. Large arteries tend to bleed more persistently than small arteries. Clean injuries without significant contusion of surrounding tissue favor continued bleeding. Hemorrhage tends to be greater if there is a ready route for escape of blood to the exterior. Arteries having incomplete lacerations are prone to continue to bleed, while completely divided arteries not infrequently cease to bleed with retraction and thrombosis of the divided ends. Blood loss is sometimes minimal when there is a concomitant injury of an adjacent vein into which the lacerated artery may bleed with the formation of an arteriovenous communication. When bleeding ceases spontaneously or as the result of first-aid treatment and the limb is not ischemic, definitive treatment can be deferred for a reasonable time. When the limb is ischemic or when bleeding continues or recurs, treatment must be carried out without any waste of time. If the injured artery is ligated rather than repaired, it is safer to divide the artery between ligatures than to ligate it in continuity. Every

effort, however, should be made to preserve or restore the continuity of the damaged artery and this is ordinarily possible. Depending upon the nature of the injury, lateral suture of the lacerated artery, end-to-end anastomosis, or replacement of the excised damaged portion by a vascular graft may be the treatment of choice (Figs. 13 to 15). At

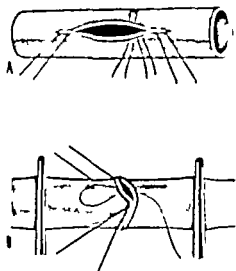


Figure 13. A, Technique for performing lateral anastomosis. B, End-to-end anastomosis by use of interrupted overlying mattress sutures (Shumacker II, B. J. A.M.A., vol. 151)

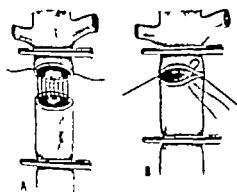


Figure 14. A useful method for end-to-end anastomosis when branches fix one segment of the artery so it cannot be rotated freely. This is essentially the technique utilized by Blalock for subclavian-pulmonary artery anastomosis (Shumacker II, B. J. A.M.A., vol. 151)

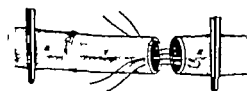


Figure 15. Technique for interpolation of vascular graft (Shumacker II, B. J. A.M.A., vol. 151)

the same time adequate debridement must of course be accomplished in all patients in whom there is contusion of tissues.

The Vasospastic Disorders. Normal individuals vary considerably in vascular tonus. Some have an unusually low vasomotor tonus. Their extremities tend to remain warm and to cool slowly in a cold environment. Others have a high vasomotor tone; their hands and feet tend to be cool and to lose temperature rapidly upon exposure to a cold atmosphere. In between these extremes is another group of individuals with moderate vascular tonus. The distinction between normal individuals with high vasomotor tonus and individuals suffering from a true vasospastic disorder is sometimes difficult. Evidence of vasoconstriction in the former group are looked upon as a personal peculiarity and perhaps somewhat of a nuisance. In the latter they constitute the basis for real complaints. There are a wide variety of vasospastic disorders.

Raynaud's disease is characterized by episodes of spasm of the digital arteries. It affects women predominantly although it does occur in males as well. It usually affects young individuals in their late teens or during the second or third decades of life. It may have its onset however in older individuals. It tends to affect the digits of the upper extremities predominantly although the lower extremities may be affected as well and, more rarely, the nose and ears. The disorder tends to be symmetrical. Under ordinary environmental conditions the circulation in the affected extremities is usually quite normal, although some individuals tend to have some coolness and hyperhidrosis of their extremities under most circumstances. The attacks occur as the response to cold and, in some upon emotional excitation. Characteristically the affected digits turn either white or blue during an attack and become quite numb. Upon return to a warm environment, the attack ceases, the digits become deep red in color and then normal color returns. As the part rewarms and color returns to normal the patient complains of uncomfortable paresthesias. The thumb is less commonly affected than are the fingers. Usually only the distal two phalanges are involved. The temperature which initiates attacks varies from patient to patient. Some develop attacks only upon exposure to intense cold, while others develop attacks in a moderately cool, or even in a slightly cool environment. The disorder may remain rather stationary for a period of years, but it tends to get progressively worse

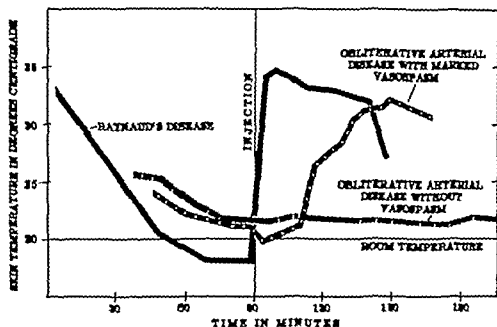


Figure 16 Chart showing common digital temperature responses to procaine anesthetization of the sympathetic chain in a patient with Raynaud's disease, and in patients with obliterative arterial disease with and without significant vasospasm (redrawn from Shumacker H. B. Surgery vol. 13)

Rarely is spontaneous improvement noted. As time passes, the patient is apt to develop painful digital ulcers, superficial necrosis of tissue takes place and, if healing occurs some of the substance of the tip of the digit is lost. Rarely does more extensive gangrene develop. In some individuals, scleroderma occurs in association with Raynaud's disease. The scleroderma may be progressive and disabling.

On examination in a warm environment the circulation usually is found to be perfectly normal. The major peripheral pulses are present. The hands and feet tend to be warm and normally moist, though in some individuals they may be somewhat cooler than normal and have excessive sweating. Characteristically the tips of the fingers become tapered and may be further deformed by the loss of tissue as a consequence of digital ulceration. Scleroderma may or may not be present. Attacks often cannot be reproduced by immersion of the affected hands or feet in ice water. They can invariably be reproduced by general body chilling. An effective method for bringing this about is the taking of a cold shower.

Entirely similar attacks are sometimes noted early in childhood and are known as hereditary cold fingers. In contrast to true Raynaud's disease, patients with hereditary cold fingers tend to get better as they grow into adult life.

A disorder otherwise quite like idiopathic

Raynaud's disease sometimes follows the use of vibrating tools and seemingly is caused by the use of such equipment.

Raynaud's phenomena, identical in every respect to the attacks which characterize Raynaud's disease, also are noted as a manifestation of obliterative arterial disease and in association with arterial compression syndromes. For this reason, in any individual having attacks of this sort it is important to rule out the presence of obliterative arterial disease such as arteriosclerosis, thrombocytopenic purpura or some compression syndrome such as may be due to a cervical rib or a tight anterior scalene muscle. The most difficult diagnostic problem is the differentiation between Raynaud's disease and non-specific obliterative arteritis, which also has a predilection for young women and for involvement of the upper extremities and is commonly associated with Raynaud like attacks. Figure 16 shows digital temperatures in these processes.

Another relatively common vasospastic disorder is known as acrocyanosis. This, too, tends to occur primarily in women, especially young women. Unlike Raynaud's disease, acrocyanosis constitutes a relatively persistent complaint. The hands and sometimes the feet are affected. They tend to remain cold, excessively wet and cyanotic. The coldness and cyanosis are aggravated by exposure to a cold environment but are usually evident even in a warm atmosphere. Only under cir-

circumstances conducive to extreme vasodilatation do the affected extremities become warm and normally colored.

Livedo reticularis occurs in both males and females and in all age groups. It is characterized by a peculiar reticular bluish mottling of the skin. The legs and feet are commonly affected; occasionally the upper extremities are affected as well. The mottling tends to be persistent. It is exaggerated by exposure to cold and by dependency. It tends to clear somewhat in a very warm atmosphere and more particularly after exercise. Under no circumstances does it disappear completely. It may be associated with ulceration of the skin. It is usually accompanied by vasospasm.

A great many individuals suffer from an ill-defined vasospastic condition. Both males and females in all age groups are affected, though it is more common in the young than in the old. Common complaints are coldness and hyperhidrosis of the extremities. Sometimes numbness is present particularly in cold weather. Occasionally some cyanosis is noted. Hyperhidrosis may be extreme. In some instances the hyperhidrosis may be associated with a foul odor. This condition is known as *bromhidrosis*.

Patients with vasospastic disorders should be persuaded to abstain from tobacco and should dress warmly in cool weather. The milder vasospastic disorders sometimes are helped remarkably by the autonomic blocking agents. When these difficulties are severe and progressive and fail to respond to such measures they are best treated by sympathetic denervation. Altogether the results are quite good. They are excellent in the ill-defined group of vasospastic difficulties and in acrocyanosis. The discoloration and the associated vasoconstriction which are evident in patients with *livedo reticularis* are remarkably improved. The limbs remain warm and dry and the bluish mottling is replaced by a faint pink reticular mottling which is not nearly so conspicuous. Ulcerated areas tend to heal. The results are excellent in cases of acrocyanosis. Raynaud's attacks usually disappear or recur much less commonly and only after a more severe cold stimulus than was formerly required to produce them. Attacks never are initiated by emotional excitation in sympathectomized extremities. Digital ulcers tend to heal and not to recur. Rarely is any improvement noted in scleroderma and in some instances the scleroderma process continues to become progressively worse.

A very serious acute vasospastic difficulty is known as *arterial stupor* or segmental arterial spasm. In this condition a segment of a major arterial stem becomes so constricted as a result of spasm that it fails to transmit blood through its lumen. It occurs as a consequence of trauma in the vicinity of the artery. Sometimes the trauma is surgically induced. Varying degrees of ischemia result, depending upon the intensity of the spasm, the completeness of the obstruction of blood flow through the main artery and the location of the segment involved. Segmental spasm of the femoral artery may for example be associated with complete ischemia of the entire leg and foot almost up to the knee. If unrelieved gangrene may develop. Sometimes segmental arterial spasm responds dramatically to regional sympathetic procaine blockade or to spinal, caudal or general anesthesia. Unfortunately not all the patients respond to such measures. If the affected artery must be exposed under any circumstances because of the associated injury or if other measures have failed to relieve the spasm the affected portion of the artery should be stripped of its gross adventitia, bathed in procaine solution and irrigated with warm saline solution. If these measures do not relieve the spasm one may try the injection into the proximal artery of procaine solution, Prostigmin, or one of the autonomic blocking agents. In an occasional patient all efforts fail to relieve the spasm.

Organic Obstruction of Arteries. The most dramatic of the acute occlusive disorders is *arterial embolus*. The etiologic factors for such emboli are rheumatic valvular heart disease, myocardial infarction with mural thrombi and thrombi developing upon an arteriosclerotic plaque in the aorta or some large artery or within an aneurysmal sac. Thrombi in the heart are especially apt to become dislodged during episodes of atrial fibrillation or upon conversion of atrial fibrillation to normal sinus rhythm. The patient suddenly experiences pain, coldness, numbness and paresis. The ischemic part rapidly becomes pale, cyanotic, cold, hyperesthetic or anesthetic, paretic or paralyzed. Generally the diagnosis is easy. Occasionally it may be difficult to distinguish this condition from acute arterial thrombosis, rarely from segmental arterial spasm or acute venous thrombosis with marked vasospasm. The localization of the obstruction usually presents no problem. Emboli tend to lodge where the artery suddenly becomes reduced in caliber and ordinarily at points of bifurcation or

compression—the end of the aorta, the division of the common iliac into external iliac and hypogastric, the bifurcation of the common femoral artery the level at which the superficial femoral passes under the adductor tendon, the branching of the popliteal into anterior and posterior tibial arteries. If one observes the most distal pulse palpable, the level of coldness discoloration and numbness, and the parts paralyzed or weak, he can determine the level of occlusion with remarkable accuracy. In patients with saddle embolus to the terminal aorta for example, the iliac and femoral pulses are absent. Hip movements are present, but knee ankle and toe movements are weak or absent. The level of coldness pallor and sensory loss is near the groin. The findings are essentially the same but limited to one side when the common iliac artery is suddenly obstructed. The cold level may be a little more distal. When the common femoral artery is occluded, the last palpable pulse is just below the inguinal ligament. Knee movements are present, but not ankle or toe motion. The cold level is below the knee. In distal femoral or popliteal occlusion, pulsation is felt down to the adductor tendon or to the lower part of the popliteal space. The cold and sensory-change level is above the ankle. Only toe movements are lost. Unusually good collateral circulation or collateral circulation impaired by pre-existing occlusive disease may make the affected level lower or higher than one would ordinarily anticipate.

Effective therapy depends upon prompt recognition and the earliest possible treatment. As soon as the diagnosis is made heparin should be administered, the ischemic extremity made dependent and protected from injury and preparations begun for definitive treatment. If an artery important in the nourishment of the limb is occluded, embolectomy should be carried out immediately except in the most unusual circumstances. This is a simple procedure. Most embolectomies, for example those in the femoral artery can be accomplished painlessly with the patient under local anesthesia. Little or no risk is entailed. In contrast, the risk of amputation in neglected patients is relatively high. Once the embolus is removed and the small arteriotomy incision closed, the patient experiences relief of symptoms and good circulation is evident in the previously ischemic extremity.

The key to success is the performance of embolectomy before thrombosis has occurred distal to the site of the embolus. Prior to em-

bolectomy everything possible should be done to decrease the likelihood of such distal arterial thrombosis. In general, the chance for a good outcome is directly related to the shortness of the interval between occlusion by the embolism and embolectomy. Time is important, however primarily in view of the fact that the briefer the interval the more likely the distal arterial tree is to be patent and the longer the interval the more likely it is to be obstructed by thrombosis. Occasionally the entire distal arterial tree is clotted within a few hours. In contrast, it may remain open and successful embolectomy may be performed as long as forty-eight hours or more after the embolus has become lodged in the vessel. The better preserved the circulation in the affected extremity the more certain one can be of a good result from embolectomy. Hence preservation of some circulation should not influence one to elect nonsurgical treatment but should, on the other hand, prompt one to proceed with operative removal of the obstruction. After embolism has been treated, everything possible should be done to prevent recurrence. All too often a second or third embolus has proved fatal. If no specific therapy directed at the primary cause is applicable, prolonged anticoagulant treatment may be helpful. In patients with mitral stenosis, commissurotomy should be carried out. If the patient has had fibrillation, conversion should later be attempted.

Acute arterial thrombosis is similar in symptoms and signs but often of more gradual onset. The signs are not so consistently clear cut for any given level of obstruction, though the site of occlusion is generally evident from careful examination. It is hoped that such patients treated early will prove suitable for thrombectomy or by-pass grafting procedures. Often sympathetic denervation brings about remarkable improvement when the thrombosis is believed to be of too long duration to justify immediate direct operative attack upon the thrombosed artery. Not infrequently patients having acute thrombosis who survive without gangrene can be treated later by by-passing procedures (Fig. 17).

The commonest of all arterial disorders are the chronic obliterative arterial diseases and by far the commonest of these is *arteriosclerosis obliterans*. This occurs more commonly in men than in women and in the elderly rather than in the young. Though it is usually seen in individuals forty years or more in age, it is occasionally observed in younger people particu-¹ in those with

diabetes. Among diabetics females are affected about as commonly as males. In many individuals, the arteriosclerotic process is a diffuse one affecting arteries throughout the body. In other individuals, it may be a very localized disease affecting only a relatively small segment of some arterial stem. Superficial migratory phlebitis such as occurs in thromboangiitis obliterans is not observed in arteriosclerosis. The lower extremities are affected predominantly and only rarely the upper extremities.

Thrombotic obstruction may take place in the terminal aorta, producing *Leriche's syndrome*. In this condition the patient complains of intermittent claudication in the hips and frequently of impotence. When the iliac arteries are obstructed the intermittent claudication is in the hips and thighs. When the femoral or popliteal arteries are occluded intermittent claudication is felt in the calves. In rare circumstances in which the occlusive process affects the smaller arteries of the leg, the intermittent claudication may be noted in the feet. In addition to intermittent claudication, which is a very common complaint, rest pain may be present. This may be quite severe. It is usually experienced when the patient is in bed at night and is relieved to a certain extent by making the feet dependent. In patients with marked ischemia, the feet may be cold and numb. Ulceration of the toes or foot and superficial or deep gangrene may be present. In rare patients with occlusion of only the small arteries of the foot ul-

ceration or even gangrene of toes may be present with good pulses in the major arteries of the lower extremity and no intermittent claudication.

On examination one usually observes absent or weak pulsations in one or more of the major arteries of the extremity. The foot is apt to be cool and somewhat pale. The skin may have an atrophic glossy appearance. Growth of hair may be poor and the nails may be deformed. The foot is likely to show pallor on elevation and rubor in the dependent position. Venous filling may be slow and incomplete. A ray study of the soft tissues may reveal calcification of arteries.

Thromboangiitis obliterans occurs far less commonly than obliterative arteriosclerosis. Indeed in most communities thromboangiitis obliterans is a rare disease. The diagnosis is made far more commonly than the incidence of the disease warrants. It occurs predominantly in young individuals under the age of forty and almost exclusively in males. Though the lower extremities are primarily affected the upper extremities may be involved as well and are diseased in a higher percentage of patients than is true of arteriosclerosis. There is a history of superficial migratory phlebitis in a substantial percentage of patients with thromboangiitis obliterans. Vessels in any part of the body may be affected, but the occlusive inflammatory process tends to be segmental. Microscopically there is evidence of nonsuppurative panarteritis or panphlebitis associated with prolifera-

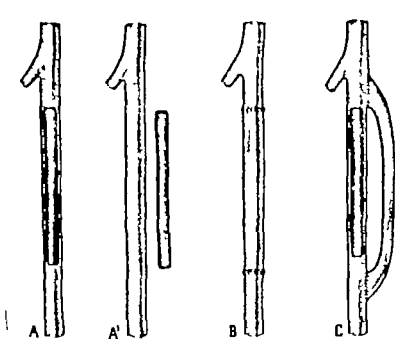


Figure 17 Methods for dealing with segmental arterial occlusion. A, By thromboendarterectomy. B, Excision and end-to-end interpolation of graft. C, End-to-side by-pass with graft.

tion of intimal cells and cellular infiltration, particularly about the vasa vasorum in the adventitial layer and a highly organized obstructing thrombus. As is true in patients with obliterative arteriosclerosis, patients with thromboangiitis obliterans tend to have intermittent claudication, may have severe rest pain and are subject to ulceration of the toes and feet and to superficial and deep gangrene. In general, ulcerations due to thromboangiitis obliterans are more painful than comparable lesions due to arteriosclerosis. In general too, the collateral circulation about obstructed lesions in patients with thromboangiitis obliterans is better than around similarly obstructed areas in patients having arteriosclerosis. Nevertheless collateral circulation is not infrequently totally deficient, leading to extensive gangrene and necessitating amputation.

The chronic obliterative arterial diseases demand very careful treatment. Abstinence from tobacco should be advised and this is absolutely mandatory in patients with thromboangiitis obliterans. Patients should be taught to take extraordinarily good care of the skin of their feet, to trim their nails carefully and in such a way as to minimize the hazard of developing ingrowing toenails. They should consult physicians for minor difficulties rather than apply home remedies. Seemingly trivial lesions such as calluses, fissures, vesicles and superficial ulcers may lead to loss of limb. Patients with intermittent claudication should be advised to cease walking and rest for a moment just short of the distance which brings on pain or fatigue, rather than to stop and rest after the onset of such claudication. They should understand that the walking distance can be increased by slowing the speed of walking. Existing diabetes should be kept under rigid control. Whenever annoying or disabling claudication is present, special study may be warranted in order to determine whether one is dealing with a segmental occlusion of a major arterial stem.

Distal arteriography is especially helpful. One can almost always determine the level of proximal obstruction. It is important to find out the extent of the obstruction and whether the distal arterial tree is open and adequate or whether it is diseased and insufficient. When a segmental occlusion is present with an open distal pathway there is an excellent possibility of being able to restore relatively normal circulation to the extremity and to eliminate intermittent claudication by thromboendarterectomy or a grafting procedure.

In general, by-pass grafting procedures are the most effectual. The likelihood of a segmental block is suggested by the presence of intermittent claudication with relatively good circulation at rest. It is more apt to be present in the young than in the old, in individuals who have unilateral rather than bilateral difficulty in the absence of demonstrable extensive calcification of vessels and in instances in which the arterial block is proximally rather than distally situated. Unfortunately all too often, when the occlusive process begins in the femoral artery it may extend down through the entire popliteal artery and involve its branches in the calf. Under such circumstances, there is no effective way of reopening the occluded arteries.

In these patients, the best method for improving the circulation is by means of sympathectomy. This may bring about complete relief of intermittent claudication in those rare instances in which it is felt solely in the small muscles of the foot. In the vast majority of patients, in whom the claudication is felt in the calf thigh or hips, sympathectomy cannot be counted upon to bring about any significant increase in walking distance. Many patients cannot walk any further after sympathectomy has been performed than they could before. When some improvement is noted, it is usually of small degree. Sympathetic denervation does, however, often bring about remarkable improvement in the general circulation to the extremity. A previously cold foot may become quite warm. The patient may be relieved of agonizing rest pain, numbness of the foot and toes may disappear and areas of superficial ulceration may heal.

If well-developed deep gangrene is present in a patient with obliterative arteriosclerosis the chances of avoiding amputation are slight, even though the gangrene may be limited to a toe. Toe amputations are rarely followed by healing when performed in elderly individuals with arteriosclerosis. Occasionally transmetatarsal amputations may be successful. Quite commonly good healing follows amputation carried out below the knee even when the popliteal, femoral and iliac pulses are not palpable. The more conservative amputations are more commonly successful in patients with thromboangiitis obliterans than in those having arteriosclerosis obliterans.

If pain limited to the distal portions of the foot persists after sympathetic denervation, it can sometimes be abolished by crushing appropriate sensory nerves in the leg. Such procedures are usually effectual if

the pain is diffuse and at best are not entirely satisfactory since the patient trades a painful foot for a numb one. On occasion it may be necessary to perform chordotomy in order to relieve pain. This is a justifiable procedure if severe pain persists in spite of more conservative measures such as sympathetic denervation, and if the circulation is sufficiently good so that there is no real threat of loss of limb.

Aneurysms. An aneurysm is a hollow sac filled with blood and connected directly with the lumen of an artery. Aneurysms are classified into two types. *True aneurysms* are those which result from expansion of the arterial wall and in which the wall of the parent artery enters into the composition of the sac. *False aneurysms* in contrast result from the organization of pulsating hematomas which follow traumatic or infectious disruption of the wall of the artery. The sac of such aneurysms is formed entirely anew from perivascular tissues. Aneurysms may be further classified into traumatic mycotic erosion and congenital aneurysms aneurysms which develop distal to areas of partial constriction or stenosis of arteries and aneurysms resulting from disease of the arterial wall. A special variety of the latter type of aneurysm is the dissecting aneurysm. Aneurysms are further classified according to shape as fusiform or saccular lesions. True aneurysms may be fusiform or saccular. Most traumatic, mycotic, erosion and congenital aneurysms are saccular.

When an aneurysm develops as a consequence of traumatic perforation or rupture of an artery the first stage is that of the pulsating hematoma. Blood extravasates through the opening in the arterial wall into adjacent tissues. The size and shape of the pulsating hematoma are determined by the nature of the surrounding tissues, the caliber of the injured artery and of the rent in its wall and by such factors as arterial blood pressure and the efficiency of the clotting mechanism. The major portion of the blood tends to clot quickly but as time passes the thrombus becomes compressed thinned out in its peripheral portions and a sac of dense fibrous tissue forms. The central space communicating freely with the lumen of the artery, becomes lined with a smooth endothelial like layer. Mycotic and erosion aneurysms develop in a similar fashion, except that the causative factor is infection rather than injury. In some instances mycotic aneurysms develop from the lodging of an infected embolus in the lumen of the artery. Presumably

the infection spreads into the wall of the artery and finally brings about thrombosis of its blood supply and ischemic erosion. In other instances mycotic aneurysms result from the plugging up by small infected emboli of the vasa vasorum with resultant ischemic necrosis of the arterial wall. An infection outside the arterial wall may lead to infection ischemia and disruption of the wall of the artery. These are erosion aneurysms.

Aneurysms may develop in arteries just beyond an area of partial constriction. One example is the aneurysm which develops in the subclavian artery distal to a point of compression by a tight anterior scalene muscle or a cervical rib. Another example is the aneurysm which develops beyond an area of coarctation of the aorta. The causative mechanism is thought to be the action of hydraulic forces. Blood passes through the constricted area with increased velocity and distal to the point of stenosis meets a mass of more slowly moving blood. Here there is eddying of blood and increased lateral pressure. The clash of opposing streams and the increased lateral pressure result in structural fatigue and distention of the wall.

Congenital aneurysms result from intrinsic weakness of the wall of the artery. The weak wall gradually or suddenly gives way and expands into a saccular aneurysm.

Aneurysms due to arterial disease such as arteriosclerosis or syphilis also result from the gradual or sudden giving way of the arterial wall. This may be the result of a progressive dilatation and stretching of the diseased and weakened wall or a more or less localized disruption of the inner coats with ballooning out of the weaker portion of the wall. If the entire circumference of the artery is involved, a fusiform lesion develops. If only a portion of the wall is involved, a saccular aneurysm arises. Dissecting aneurysms develop as the result of medionecrosis. The rent occurs in the intima and media and blood extravasates into the potential space in the medial portion of the arterial wall. The dissection may extend a variable distance along the course of the vessel and may rupture back into the lumen of the artery at some other point or perforate through the outer layer with resultant hemorrhage.

The majority of aneurysms of the ascending aorta are syphilitic in origin, although some develop as a result of arteriosclerosis and a few from trauma. Most of the aneurysms of the arch and descending thoracic aorta and almost all aneurysms of the abdominal aorta are arteriosclerotic. Peripheral ar-

terial aneurysms may be traumatic, syphilitic, arteriosclerotic or the result of medionecrosis. The peripheral and visceral arteries are the common sites for mycotic and erosion aneurysms. Intracranial aneurysms are thought to be primarily congenital, although some develop from inflammatory arteritis.

Symptoms caused by aneurysms vary with the location, size and structure of the lesion. Sometimes patients are unaware of the presence of aneurysms. In other instances a recognized mass or abnormal pulsation directs the patient's attention to the aneurysm. The chief symptoms arise from compression of adjacent structures, from rupture or from thrombosis. Aneurysms in the extremities may cause a sense of fullness or throbbing. They may be associated with paralysis of nerves or occlusion of veins as the result of compression. Sometimes they interfere with joint function. The thrombus within the sac may increase in size and bring about obstruction of blood flow into the distal artery with resultant ischemia. On occasion, a portion of the intraluminal thrombus may become dislodged and pass into the distal artery as an embolus. Aneurysms within the thorax may cause pain from vertebral erosion or compression of nerve roots. They may bring about partial or complete obstruction of the trachea, bronchi or esophagus and they may be associated with vagal paralysis. Intra-abdominal aneurysms may be similarly associated with root pain. They sometimes cause gastrointestinal dysfunction and, on occasion, obstruction of the ureters or vesicle neck. In the cranial cavity aneurysms often cause headache and localized neurologic signs such as ophthalmoplegia. Regardless of their situation, aneurysms may perforate with serious consequences. An aneurysm in the extremity may rupture through the overlying skin particularly if there is a thin, poorly healed wound, and fatal exsanguination may take place. When an aneurysm of an extremity perforates without external bleeding, adjacent blood vessels may be occluded by compression, with resultant ischemia. Intracranial rupture of an aneurysm near the surface of the brain results in the classical picture of subarachnoid hemorrhage. Rupture of aneurysms situated within the substance of the brain brings about variable signs and symptoms, depending upon their location and the extent of the hemorrhage. The rupture of an intrathoracic or abdominal aortic aneurysm usually leads to death, though prompt treatment may avert this disaster.

Aneurysms which are accessible to palpa-



Figure 18 Photograph showing thrombosed aortic aneurysm which required excision because of pressure paralysis of radial, ulnar and median nerves (Shumacker, H. B., and Wayson, E. E.: *Am. J. Surg.* vol. 79)

tion and auscultation can be recognized by the presence of a mass with intrinsic pulsation, a systolic thrill and a systolic bruit. A systolic bruit is heard in most aneurysms of the extremities but is not infrequently absent in intrathoracic and intra-abdominal lesions. Intra-abdominal aneurysms can usually be recognized with relative ease by the presence of a pulsating mass. Those within the thoracic cavity can be identified by fluoroscopic and x-ray examination. The commonest mistake in diagnosis is made when one erroneously interprets a transmitted pulsation of a cystic or solid mass lying adjacent to a large artery as intrinsic pulsation of the mass itself.

Aneurysms rarely become obliterated by progressive thrombosis. Sometimes, when thrombosis occurs within an aneurysm, there is simultaneous occlusion of the distal artery and ischemia of the part. Even if an aneurysm becomes solidly thrombosed without obstruction of blood flow through the distal artery the firm, clotted sac may persist as a tumor mass and require operative treatment because of its size or because it compresses adjacent structures, such as nerves (Fig. 18). Since aneurysms rarely undergo a satisfactory "spontaneous cure" and are associated with very real danger they should be treated surgically.

Treatment of aneurysms depends upon their size and location. If the aneurysm is accessible to palpation it is wise to carry out a test of the collateral circulation before undertaking operative treatment. Under all circumstances, aneurysms involving main arterial stems should be

— some way

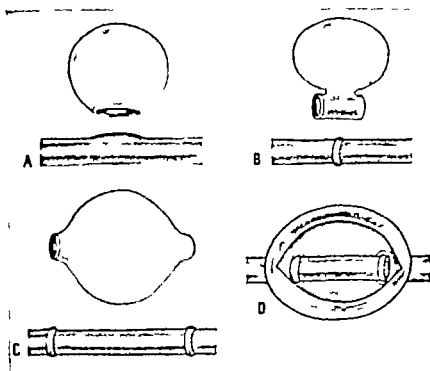


Figure 19 Drawings indicating methods for maintaining or restoring continuity of affected artery in patients with aneurysms. A By excision and lateral suture. B by excision and end-to-end anastomosis. C, by excision and grafting. D by intraneurysmal suture-interpolation of graft. (Shumacker H. B., In *Cyclopedia of Medicine, Surgery, Specialties*, F. A. Davis Company.)

so as to preserve or restore circulation through the affected artery (Figs. 10 and 20). In certain sacular aneurysms this can be accomplished by extirpating the lesion and closing the opening from the artery into the sac by lateral suture. In other instances it is possible to remove the aneurysm and unite the mobilized ends of the artery by direct anastomosis. Frequently excision of an aneurysm necessitates interpolation of a graft. Preserved homologous aortic grafts

and grafts fashioned of certain plastic materials are suitable for aortic replacement. Plastic prostheses are more reliable than homografts and are therefore preferred. In the extremities, fresh autogenous veins also serve satisfactorily for bridging defects of arteries. Whenever aneurysms are treated surgically it is important that adequate exposure be obtained and that one gain control of the affected artery proximally and distally. Large aneurysms in the extremity are sometimes approached best by opening the sac directly under tourniquet control. One can then deal with the openings of the artery into the sac by suture or transfixion according to the obliterative aneurysmorrhaphy technique of Matas. If a large artery is involved however it is preferable to bridge the defect by suture interpolation of a graft between the ends of the artery. Once this is accomplished, the major portion of the sac wall can usually be excised without damage to the collateral vessels. In recent years, a great advance has been made in the treatment of aneurysms by demonstration that the same principles which have been used for years in the surgical management of peripheral aneurysms can be utilized for the excisional cure of aneurysms of the aorta. When an aneurysm of a main-stem peripheral artery is not associated with adequate collateral circulation, the collateral circulation can often be improved by prolonged use

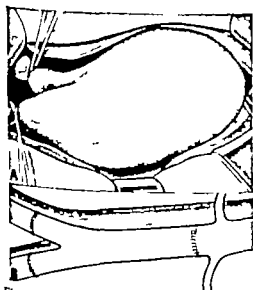


Figure 20 A, Large aneurysm of abdominal aorta and its bifurcation. B Graft interpolated after excision of aneurysm shown in A. (Shumacker H. B., and King, H. *Surg. Gynec. & Obst.*, vol. 99)

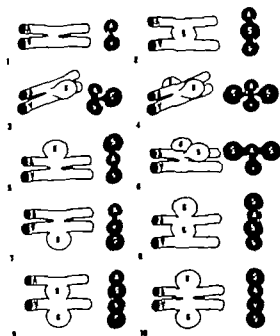


Figure 21. Diagrammatic representation of various types of arteriovenous fistulae and associated aneurysms. Symbols: A, artery V vein, S saccular aneurysm (Shumacker H. B., and Wayson, E. E.: *Am. J. Surg.*, vol. 70)

of intermittent proximal occlusion of the artery. Sympathetic denervation is a valuable adjunct in bringing about the development of good collateral circulation since, with sympathetic denervation, maximal blood flow through the collateral vessels is assured.

Arteriovenous Fistulae. Arteriovenous fistulae are abnormal, direct connections between arteries and veins. Most arteriovenous fistulae result from trauma, some are the result of a congenital derangement. Arteriovenous fistulae may be classified into several groups (Fig. 21) direct fistulous communications between arteries and veins, arteriovenous fistulae associated with aneurysms circled aneurysms in which there are multiple communications and marked dilatation of arteries and veins in the vicinity of the lesion and arteriovenous fistulae in vascular tumors such as hemangiomas and hemangio-endotheliomas.

Arteriovenous fistulae are not always associated with symptoms. Sometimes the lesion is first called to the attention of the patient by its discovery during the course of a physical examination. Occasionally the patients are aware of some difficulty because they hear or feel a buzzing sensation in the region of the fistula or because they palpate a thrill in its vicinity. Some patients seek help because they notice dilated veins in the region of the lesion or alteration of warmth

	Ballistocardiogram	Pulse Rate	Stroke Volume	Cardiac Index
Fistula Open		86	140	6.2
Fistula Occluded		62	110	3.5
After Atropine, Fistula Open		120	151	9.3
After Atropine, Fistula Occluded		120	94	5.8

Figure 22. Ballistocardiographic tracings, pulse rate and estimated stroke volume and cardiac index changes with occlusion of human arteriovenous fistula before and after atropinization (Elkin, D. C., and Warren, J. V.: *J.A.M.A.*, vol. 184)

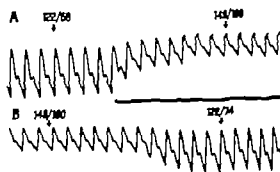


Figure 23. Optically recorded Hamilton manometric measurements of change in blood pressure (A) upon occlusion and (B) upon release of pressure from experimentally produced femoral arteriovenous fistula in dog with total excision of extrinsic nerve supply to heart. Period of occlusion indicated by heavy line (Shumacker H. B., et al.: *J. Thoracic Surg.*, vol. 20)

and color of the affected hand or foot. Sometimes medical aid is sought only after the development of cardiac symptoms, such as palpitation, tachycardia, dyspnea or frank heart failure. In rare circumstances, the patient may first seek help because of chills and fever resulting from bacterial endarteritis in the fistula.

The diagnosis usually is simple. Characteristically in the region of the lesion a continuous bruit is present, with a systolic accentuation and a continuous thrill. If the fistula is located so that it can be temporarily occluded by digital compression, one can demonstrate a slowing of the pulse rate during compression—the Nicoladoni-Brannham phenomenon. Usually at the same time, the systolic and diastolic pressures rise and the pulse pressure decreases (Figs. 22 and 23). Such changes are pathognomonic of arteriovenous fistulae. If one withdraws venous blood from the neighborhood of the fistula, he can demonstrate arterial oxygen—

saturation as compared with blood drawn from a similarly treated vein in the contralateral extremity. One will commonly note evidences suggesting increased circulation. The hand or foot may be unusually warm and well colored. If the arteriovenous fistula has been present before completion of epiphyseal growth, some increase in the length of the extremity may have taken place. Not in all instances are arteriovenous fistulae associated with evidences of increased circulation to the extremity. Sometimes they are associated with coldness, pallor, cyanosis and hyperkiloosis and in a few instances ischemic ulceration may be present. Although the arterial pulsation is increased in the region of the arteriovenous communication, the pulses distally are characteristically weaker than in the contralateral extremity and the blood pressure distal to the lesion is reduced. The systemic blood pressure may be somewhat elevated and the pulse pressure is characteristically increased. If the pulse pressure is wide, a capillary pulsation may be demonstrated or a pistol-shot sound may be audible over large peripheral arteries. There may be evidence of cardiac enlargement or even of heart failure. In most instances of large arteriovenous fistulae one can demonstrate an increase in total circulating blood volume.

The circulatory changes which occur on sudden occlusion of an arteriovenous fistula are a very interesting physiologic response. The slowing of the pulse rate can be abolished by administration of large doses of vagal blocking agents. Vagal blocking, however, does not abolish the blood pressure responses to the occlusion. The cardiac output undergoes a precipitous and significant decrease upon closing of the fistula. All of these changes occur with the very first beat of the heart after the fistula is compressed. It has been demonstrated experimentally that blood pressure and cardiac output alterations upon occlusion of an arteriovenous fistula occur after depriving the heart completely of its vagal and sympathetic nerve supply. They constitute mechanical responses to shutting off abruptly the parasitic circulation through the fistula.

The increased work load on the heart in patients with large arteriovenous fistulae gradually leads to dilatation of the heart and eventually in some instances, to frank heart failure. This results from the increased blood volume and the recirculation of blood through the fistula and directly back to the heart. Cardiac strain can be judged in a

rough fashion by the magnitude of the pulse and blood pressure responses to digital occlusion of the fistula. Significant cardiac enlargement is likely to be found in patients with marked slowing of the pulse rate, considerable rise in systolic and diastolic pressure and marked decrease in pulse pressure. Cardiac enlargement is also directly related to the size of the artery involved, to the size of the fistulous communication and to the duration of the lesion. Fistulae in the abdomen, pelvis and lower extremities are more likely to result in significant cardiac enlargement than fistulae of the head, neck and upper extremities.

Arteriovenous fistulae rarely close spontaneously. They tend, with the passage of time, to be associated with progressive dilatation of the veins in the neighborhood of the lesion and with dilatation of the proximal artery. The blood volume increases and cardiac dilatation becomes more marked as time passes. Ultimately heart failure may occur. In rare instances, subacute bacterial endocarditis may develop in the fistula, manifested by bacteremia, chills and fever. Because of these consequences arteriovenous fistulae should be treated by surgical extirpation.

As in patients with peripheral aneurysms, it is wise before undertaking operation upon an arteriovenous fistula, to determine if possible the status of the collateral circulation. Ample exposure of the arteriovenous fistula should be obtained and the main artery and vein should be isolated proximally and distally before approaching the region of the fistula itself. One should never carry out a proximal arterial ligation for an arteriovenous fistula except in the case of internal carotid-cavernous sinus fistula. In this instance the internal carotid artery has no branches between the point of origin and the point of the fistula and ligation of the carotid artery, preferably with ligation of the concomitant vein, often results in cure. In general, however, proximal arterial ligation does not effect a cure and is quite likely to result in gangrene. With the proximal artery ligated, the collateral vessels tend to drain into the distal artery and back through the fistulous communication into the vein and, thus, to deprive the distal portions of the extremity of adequate circulation. Arteriovenous fistulae should never be treated by simple ligation of the fistulous tract, for recurrence takes place far too commonly after such procedures. The fistula must be divided or excised. If the lesion affects a

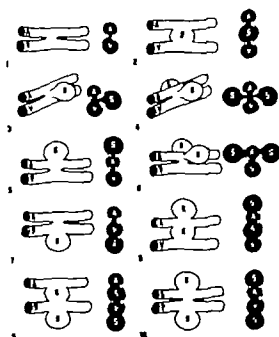


Figure 21 Diagrammatic representation of various types of arteriovenous fistulae and associated aneurysms. Symbols: A, artery; V, vein; S, sacculi aneurysm (Shumacker H. B. and Wayson, E. E. *Am. J. Surg.* vol. 70)

of intermittent proximal occlusion of the artery. Sympathetic denervation is a valuable adjunct in bringing about the development of good collateral circulation since, with sympathetic denervation, maximal blood flow through the collateral vessels is assured.

Arteriovenous Fistulae Arteriovenous fistulae are abnormal, direct connections between arteries and veins. Most arteriovenous fistulae result from trauma, some are the result of a congenital derangement. Arteriovenous fistulae may be classified into several groups (Fig. 21) direct fistulous communications between arteries and veins, arteriovenous fistulae associated with aneurysms, circled aneurysms in which there are multiple communications and marked dilatation of arteries and veins in the vicinity of the lesion and arteriovenous fistulae in vascular tumors such as hemangiomas and hemangioendotheliomas.

Arteriovenous fistulae are not always associated with symptoms. Sometimes the lesion is first called to the attention of the patient by its discovery during the course of a physical examination. Occasionally the patients are aware of some difficulty because they hear or feel a buzzing sensation in the region of the fistula or because they palpate a thrill in its vicinity. Some patients seek help because they notice dilated veins in the region of the lesion or alteration of warmth

	Ballistocardiogram	Pulse Rate	Stroke Volume	Cardiac Index
Fistula Open		86	140	6.2
Fistula Occluded		62	110	3.5
After Atropine, Fistula Open		120	151	9.3
After Atropine, Fistula Occluded		120	94	5.8

Figure 22 Ballistocardiographic tracings, pulse rate and estimated stroke volume and cardiac index changes with occlusion of human arteriovenous fistula before and after atropinization (Eldin, D. C., and Warren, J. V.: *J.A.M.A.*, vol. 134)

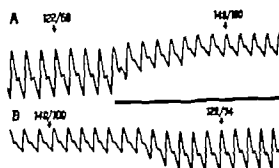


Figure 23 Optically recorded Hamilton manometric measurements of change in blood pressure (A) upon occlusion and (B) upon release of pressure from experimentally produced femoral arteriovenous fistula in dog with total excision of extrinsic nerve supply to heart. Period of occlusion indicated by heavy line (Shumacker H. B., et al.: *J. Thoracic Surg.*, vol. 20)

and color of the affected hand or foot. Sometimes medical aid is sought only after the development of cardiac symptoms, such as palpitation, tachycardia, dyspnea or frank heart failure. In rare circumstances, the patient may first seek help because of chills and fever resulting from bacterial endarteritis in the fistula.

The diagnosis usually is simple. Characteristically in the region of the lesion a continuous bruit is present, with a systolic accentuation and a continuous thrill. If the fistula is located so that it can be temporarily occluded by digital compression, one can demonstrate a slowing of the pulse rate during compression—the Nicoladoni-Branham phenomenon. Usually at the same time, the systolic and diastolic pressures rise and the pulse pressure decreases (Figs. 22 and 23). Such changes are pathognomonic of arteriovenous fistulae. If one withdraws venous blood from the neighborhood of the fistula, he can demonstrate

saturation as compared with blood drawn from a similarly located vein in the contra lateral extremity. One will commonly note evidences suggesting increased circulation. The hand or foot may be unusually warm and well colored. If the arteriovenous fistula has been present before completion of epiphyseal growth some increase in the length of the extremity may have taken place. Not in all instances are arteriovenous fistulae associated with evidences of increased circulation to the extremity. Some times they are associated with coldness, pallor, cyanosis and hyperhidrosis and in a few instances ischemic ulceration may be present. Although the arterial pulsation is increased in the region of the arteriovenous communication, the pulses distally are characteristically weaker than in the contra lateral extremity and the blood pressure distal to the lesion is reduced. The systemic blood pressure may be somewhat elevated and the pulse pressure is characteristically increased. If the pulse pressure is wide a capillary pulsation may be demonstrated or a pistol-shot sound may be audible over large peripheral arteries. There may be evidence of cardiac enlargement or even of heart failure. In most instances of large arteriovenous fistulae, one can demonstrate an increase in total circulating blood volume.

The circulatory changes which occur on sudden occlusion of an arteriovenous fistula are a very interesting physiologic response. The slowing of the pulse rate can be abolished by administration of large doses of vagal blocking agents. Vagal blocking, however, does not abolish the blood pressure responses to the occlusion. The cardiac output undergoes a precipitous and significant decrease upon closing of the fistula. All of these changes occur with the very first beat of the heart after the fistula is compressed. It has been demonstrated experimentally that blood pressure and cardiac output alterations upon occlusion of an arteriovenous fistula occur after depriving the heart completely of its vagal and sympathetic nerve supply. They constitute mechanical responses to shutting off abruptly the parasitic circulation through the fistula.

The increased work load on the heart in patients with large arteriovenous fistulae gradually leads to dilatation of the heart and eventually in some instances, to frank heart failure. This results from the increased blood volume and the recirculation of blood through the fistula and directly back to the heart. Cardiac strain can be judged in a

rough fashion by the magnitude of the pulse and blood pressure responses to digital occlusion of the fistula. Significant cardiac enlargement is likely to be found in patients with marked slowing of the pulse rate, considerable rise in systolic and diastolic pressure and marked decrease in pulse pressure. Cardiac enlargement is also directly related to the size of the artery involved to the size of the fistulous communication and to the duration of the lesion. Fistulae in the abdomen, pelvis and lower extremities are more likely to result in significant cardiac enlargement than fistulae of the head, neck and upper extremities.

Arteriovenous fistulae rarely close spontaneously. They tend with the passage of time to be associated with progressive dilatation of the veins in the neighborhood of the lesion and with dilatation of the proximal artery. The blood volume increases and cardiac dilatation becomes more marked as time passes. Ultimately heart failure may occur. In rare instances subacute bacterial endarteritis may develop in the fistula, manifested by bacteremia, chills and fever. Because of these consequences, arteriovenous fistulae should be treated by surgical extirpation.

As in patients with peripheral aneurysms it is wise before undertaking operation upon an arteriovenous fistula, to determine, if possible the status of the collateral circulation. Ample exposure of the arteriovenous fistula should be obtained and the main artery and vein should be isolated proximally and distally before approaching the region of the fistula itself. One should never carry out a proximal arterial ligation for an arteriovenous fistula except in the case of internal carotid-cavernous sinus fistula. In this instance, the internal carotid artery has no branches between the point of origin and the point of the fistula and ligation of the carotid artery preferably with ligation of the concomitant vein, often results in cure. In general, however proximal arterial ligation does not effect a cure and is quite likely to result in gangrene. With the proximal artery ligated, the collateral vessels tend to drain into the distal artery and back through the fistulous communication into the vein and, thus to deprive the distal portions of the extremity of adequate circulation. Arteriovenous fistulae should never be treated by simple ligation of the fistulous tract, for recurrence takes place far too commonly after such procedures. The fistula must be divided or excised. If the lesion affects a

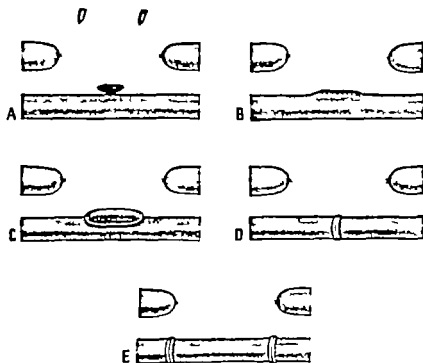


Figure 24 Drawings illustrating operative procedures permitting preservation or restoration of continuity of artery in patients with arteriovenous fistulae. A, By ligation and transfixion of divided fistula, B by lateral arteriorrhaphy; C, by transvenous lateral suture, D by end-to-end anastomosis, E, by interpolation of graft (Shumacker H. B. in *Cyclopedia of Medicine, Surgery Specialties*. F. A. Davis Company)

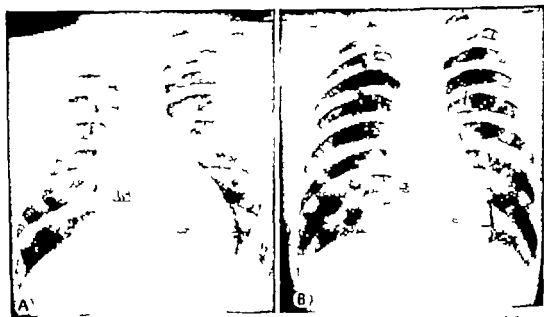


Figure 25 Chest roentgenograms (A) before and (B) two weeks after excision of external iliac arteriovenous fistula of two and one-third years duration in a young man without cardiac symptoms (Shumacker H. B. in *Cyclopedia of Medicine, Surgery Specialties*. F. A. Davis Company)

main-stem artery important in the nutrition of the extremity every effort should be made to preserve or restore its continuity. This can ordinarily be accomplished, either by carefully dissecting out the fistula, dividing it and closing the defect in the artery by

lateral suture or more commonly by excising the fistulous tract, together with the affected portion of the artery and vein, and reconstituting the artery by end-to-end anastomosis or by interpolation of a vascular graft (Fig. 24). The results of treatment are

very satisfactory (Fig 25). Any enditic dilatation which may be present tends to disappear shortly after operation. Cardiac failure if present is dramatically relieved. The increased blood volume returns to normal.

READING REFERENCES

- Allen, E. V., Barker, N. W., and Hines, E. A. *Peripheral Vascular Diseases*, 2nd ed. Philadelphia: W. B. Saunders Company, 1933.
- Allen, E. V., and Brown, C. I. Raynaud Disease: Critical Review of Minimal Hospital's for Diagnosis. *Am. J. M. Sc.* 153:187, 1932.
- Babson, H. T.: Considerations in the Location of Aortic Aneurysms. *Ann. Surg.* 134:77, 1953.
- Berger, L. Thromb. anginitis Obliterans. *Am. J. M. Sc.* 196:567, 1909.
- Cannon, J. A., and Barker, W. I. Successful Management of Obstructive Femoral Arteriosclerosis by Endarterectomy: Experience with a Semiknosed Technique in Selected Cases. *Surgery* 35:15, 1953.
- DeBakey, M. E., Cooley, D. A., and Creech, O. J., Jr. Surgical Considerations of Dissecting Aneurysm of the Aorta. *Ann. Surg.* 142:540, 1955.
- DeBakey, M. E., and Simone, I. A. Battle Injuries of the Arteries in World War II. *Ann. Surg.* 143:523, 1946.
- Ellis, D. C. Exposure of Blood Vessels. *J. A. M. A.* 12:441, 1910.
- Ellis, D. C. Traumatic Aneurysm: Matas Operation—5 Years After. *Surg. Gynec. & Obst.* 62:1, 1916.
- Freeman, N. E., and Storck, A. H. Successful Suture of Abdominal Aorta for Arteriovenous Fistula. *Surgery* 21:623, 1917.
- Harris, E. J., and others. Pliable Plastic Aortic Grafts: Experimental Comparison of a Number of Materials. *Arch. Surg.* 71:419, 1935.
- Holman, E. *Arteriovenous Aneurysm*. New York: The Macmillan Company, 1917.
- Hughes, C. W. *The Primary Repair of Wounds of Major Arteries: An Analysis of Experience in Korea*. In 10:3 *Ann. Surg.* 141:297, 1955.
- Lerkhe, B. The Syndrome of Thrombotic Obliteration of the Aortic Bifurcation. *Ann. Surg.* 127:103, 1917.
- Lewis, T. *Vascular Disorders of the Limbs*, 2nd ed. New York: The Macmillan Company, 1916.
- Lewis, T., and Lerkhe, B. M. Observations upon Vascular Mechanism in Arteriovenous Heart. *Surg. Gynec. & Obst.* 15:229, 1930.
- Maddock, W. C., and Collier, I. A. Peripheral Vascular Distortion by Tobacco and Its Relation to Thromboangiitis Obliterans. *Ann. Surg.* 98:70, 1933.
- Matas, R. Endoaneurysmorrhaphy. *Surg. Gynec. & Obst.* 70:470, 1920.
- Raynaud, M. A Local Aplysia and Symmetrical Gangrene of the Extremities (translated by Thomas Barlow). In *Selected Monographs*. London: The New Sydenham Society, 1898.
- Shumacker, H. B., Jr. Incisions in Surgery of Aneurysms, with Special Reference to Exploration in Antihubital and Iliofemoral Forae. *Ann. Surg.* 121:550, 1916.
- Shumacker, H. B., Jr. Sympathectomy as an Adjuvant in Operative Treatment of Aneurysms and Arteriovenous Fistulas: Sympathectomy Performed before or at Time of Operation. *Surgery* 22:571, 1917.
- Shumacker, H. B., Jr., and King, H. Surgical Treatment of Ruptured Aortic Aneurysms. *Arch. Surg.* 71:784, 1915.
- Shumacker, H. B., Jr., and Moore, T. C. Leg and Thigh Amputations in Obstructive Arterial Disease. *Arch. Surg.* 63:458, 1951.
- Ziperman, H. H. Aortic Arterial Injuries in the Korean War: A Statistical Study. *Ann. Surg.* 139:1, 1954.

The Lymphatics

By GERALD H. PRATT, M.D.

GERALD HILARY PRATT was educated at the Universities of Minnesota and Iowa and trained in surgery in the South and on the eastern seaboard. He had experience in both the Army and Navy Medical Corps during World War II and there developed his interest in the diseases of the vascular and lymphatic systems.

Definition. The lymphatic system is complex and consists of massive capillary combinations which carry the lymph from all parts of the body to a large system of col-

lecting vessels. These latter graduate in size and eventually transmit the lymph substance into the veins. Its passageway is filtered at various points by lymph glands which also

contribute lymphocytes to the fluid. Lymph itself is a transparent white or yellow fluid of alkaline type which may be pink when it contains red blood corpuscles. A common fat content may make it opalescent. The lymph corpuscles are granular similar to white blood cells and measure approximately $1/2500$ inch in diameter. The lymphatic vessels of the intestine are similar but during digestion they contain chyle, a milky substance, and they are designated as lacteals. Certain organs are of lymphocytic origin. Examples of these are the spleen, bone marrow, thymus gland, mucous membrane follicles and the tonsils.

The blood circulatory system carries oxygenated blood to the organs and periphery from the central pump, the heart. This blood passes through the capillary bed and tissue spaces and is reaccumulated in the venule end of the capillaries and returned to the heart by the venous system. The circulatory system has another component—the lymphatic system. This system is a closed one, with its own conduits, its capillaries, collecting minute lymphatic vessels, and larger transporting channels to its final vessels: the thoracic duct and the right lymphatic duct. The lymph drainage carrying certain products of metabolism from the bowel joins these final channels through the cisterna chyli (receptaculum chyli). These empty into the venous system at the junction of the subclavian and jugular veins. This lymphatic system is an integral and important part of the circulation and its anatomy and function are complex.

Embryology The lymphatic system develops embryologically like the rest of the arterial and vascular system. The lymphatic endothelium arises from the venous endothelium. There is no reason to believe that it arises from mesenchymal cells. The lymphatic endothelium forms paired and unpaired sacs. Of the former the jugular lymph sacs are derived from the jugular veins and then extend to the head, neck, thorax and arms. The other paired sacs arise from the iliac veins, where they unite with the cardinal veins. These spread to the abdominal wall, the pelvis and the legs. These lymphatic vessels join the cisterna chyli and vary from others in that they are not connected with veins. The unpaired lymphatics develop from the vena cava and mesonephric veins to form the retroperitoneal lymph sac. These lose their vein connections and join the cisterna chyli. Veins from the Wolffian

bodies form sprouts to develop as the cisterna chyli.

The lymph nodes primarily develop in capillary plexuses at the third month of fetal life. The lymphocytes are already present and these probably form lymph masses and the lymphoid nodules. The mesenchyma forms a connecting tissue capsule. Traube's nodules grow from this. The capillaries arise first, from which larger vessels lead to other vessels or nodes. The deeper plexuses develop first, both throughout the body and in the intestines and gradually the superficial thoracic vessels are formed. All the lymph sacs except the cisterna chyli are divided by connective tissue bridges, which help to form the lymph glands. The cisterna chyli in the lower part becomes a gland, but its upper portion remains as a conduit or cisterna.

Anatomy For descriptive purposes the lymphatic system may be divided into four parts: the lymphatic fluid, the capillaries, the collecting vessels including the thoracic ducts, and the lymph glands (Fig. 26).

Lymph is a clear yellow substance, similar to but more dilute than blood plasma. It has a specific gravity of approximately 1.015. Its contents are lymphocytes and red blood corpuscles. The lymph probably accumulates by an osmosis-diffusion-filtration process from the tissue fluids. It is not the same as tissue fluid, however.

The *capillaries* are lined by flat endothelial cells, which lie in connective tissue regions throughout the body in areas bathed by intracellular tissue fluids. These lymphatic capillaries anastomose widely and they do not have a set size or form. Many of these capillaries have blind ends. The plexuses have both superficial and deep layers, the superficial one being the smaller. In the skin lymphatic capillaries are found in the dermis. The epidermis is without lymphatic capillaries, which is of surgical interest. The subcutaneous tissue normally does not have any capillaries. Muscles, tendons, their sheaths, the periosteum and the joints all have lymphatic capillaries in abundance. The periosteum, the peritoneum, the pericardium and the pleura also have rich lymphatic components. In the digestive tract, just below the epithelium, are both superficial and deep lymphatic plexuses. The papillae appear to extend in blind-end formations into the villi of the small intestine. The lacteals arise in the lymphatic spaces in the intestinal villi.

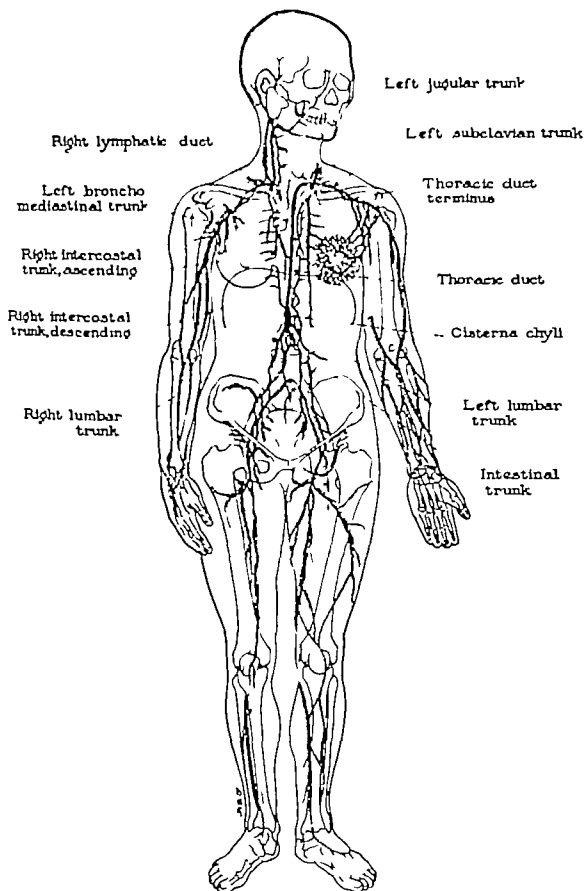


Figure 26 Diagrammatic drawing of the lymphatic drainage of the body

contribute lymphocytes to the fluid. Lymph itself is a transparent white or yellow fluid of alkaline type which may be pink when it contains red blood corpuscles. A common fat content may make it opalescent. The lymph corpuscles are granular similar to white blood cells and measure approximately $1/2500$ inch in diameter. The lymphatic vessels of the intestine are similar but during digestion they contain chyle a milky substance and they are designated as lacteals. Certain organs are of lymphocytic origin. Examples of these are the spleen bone marrow thymus gland mucous membrane follicles and the tonsils.

The blood circulatory system carries oxygenated blood to the organs and periphery from the central pump, the heart. This blood passes through the capillary bed and tissue spaces and is reaccumulated in the venule end of the capillaries and returned to the heart by the venous system. The circulatory system has another component—the lymphatic system. This system is a closed one, with its own conduits its capillaries collecting minute lymphatic vessels and larger transporting channels to its final vessels the thoracic duct and the right lymphatic duct. The lymph drainage carrying certain products of metabolism from the bowel joins these final channels through the cisterna chyli (receptaculum chyli). These empty into the venous system at the junction of the subclavian and jugular veins. This lymphatic system is an integral and important part of the circulation and its anatomy and function are complex.

Embryology The lymphatic system develops embryologically like the rest of the arterial and vascular system. The lymphatic endothelium arises from the venous endothelium. There is no reason to believe that it arises from mesenchymal cells. The lymphatic endothelium forms paired and unpaired sacs. Of the former the jugular lymph sacs are derived from the jugular veins and then extend to the head, neck, thorax and arms. The other paired sacs arise from the iliac veins where they unite with the cardinal veins. These spread to the abdominal wall, the pelvis and the legs. These lymphatic vessels join the cisterna chyli and vary from others in that they are not connected with veins. The unpaired lymphatics develop from the vena cava and mesonephric veins to form the retroperitoneal lymph sac. These lose their vein connections and join the cisterna chyli. Veins from the wolffian

bodies form sprouts to develop as the cisterna chyli.

The lymph nodes primarily develop in capillary plexuses at the third month of fetal life. The lymphocytes are already present and these probably form lymph masses and the lymphoid nodules. The mesenchyma forms a connecting tissue capsule. Trabeculae grow from this. The capillaries arise first from which larger vessels lead to other vessels or nodes. The deeper plexuses develop first, both throughout the body and in the intestines and gradually the superficial thoracic vessels are formed. All the lymph sacs except the cisterna chyli are divided by connective tissue bridges, which help to form the lymph glands. The cisterna chyli in the lower part becomes a gland, but its upper portion remains as a conduit or cisterna.

Anatomy For descriptive purposes, the lymphatic system may be divided into four parts: the lymphatic fluid, the capillaries, the collecting vessels including the thoracic ducts and the lymph glands (Fig. 28).

Lymph is a clear yellow substance, similar to but more dilute than blood plasma. It has a specific gravity of approximately 1.015. Its contents are lymphocytes and red blood corpuscles. The lymph probably accumulates by an osmotic-diffusion-filtration process from the tissue fluids. It is not the same as tissue fluid however.

The capillaries are lined by flat endothelial cells which lie in connective tissue regions throughout the body in areas bathed by intracellular tissue fluids. These lymphatic capillaries anastomose widely and they do not have a set size or form. Many of these capillaries have blind ends. The plexuses have both superficial and deep layers the superficial one being the smaller. In the skin, lymphatic capillaries are found in the dermis. The epidermis is without lymphatic capillaries, which is of surgical interest. The subcutaneous tissue normally does not have any capillaries. Muscles, tendons, their sheaths, the perosteum and the joints all have lymphatic capillaries in abundance. The perosteum, the peritoneum, the pericardium and the pleura also have rich lymphatic components. In the digestive tract, just below the epithelium, are both superficial and deep lymphatic plexuses. The papillae appear to extend in blind-end formations into the villi of the small intestine. The lacteals arise in the lymphatic spaces in the intestinal villi.

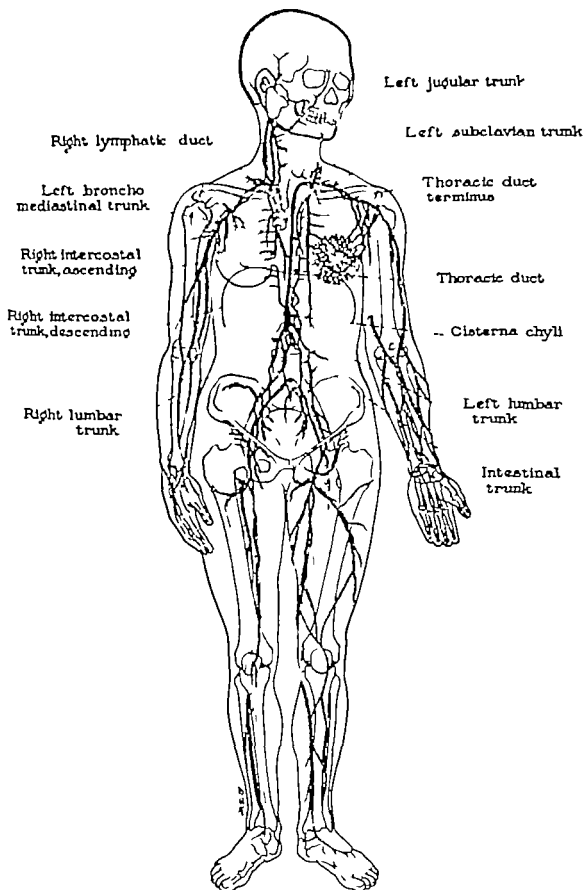


Figure 96 Diagrammatic drawing of the lymphatic drainage of the body

contribute lymphocytes to the fluid. Lymph itself is a transparent white or yellow fluid of alkaline type which may be pink when it contains red blood corpuscles. A common fat content may make it opalescent. The lymph corpuscles are granular similar to white blood cells and measure approximately 1/2500 inch in diameter. The lymphatic vessels of the intestine are similar but during digestion they contain chyle, a milky substance and they are designated as lacteals. Certain organs are of lymphocytic origin. Examples of these are the spleen, bone marrow thymus gland, mucous membrane follicles and the tonsils.

The blood circulatory system carries oxygenated blood to the organs and periphery from the central pump, the heart. This blood passes through the capillary bed and tissue spaces and is reaccumulated in the venule end of the capillaries and returned to the heart by the venous system. The circulatory system has another component—the lymphatic system. This system is a closed one, with its own conduits, its capillaries, collecting minute lymphatic vessels, and larger transporting channels to its final vessels, the thoracic duct and the right jugular duct. The lymph drainage carrying certain products of metabolism from the bowel joins these final channels through the cisterna chyli (receptaculum chyli). These empty into the venous system at the junction of the subclavian and jugular veins. This lymphatic system is an integral and important part of the circulation and its anatomy and function are complex.

Embryology The lymphatic system develops embryologically like the rest of the arterial and vascular system. The lymphatic endothelium arises from the venous endothelium. There is no reason to believe that it arises from mesenchymal cells. The lymphatic endothelium forms paired and unpaired sacs. Of the former the jugular lymph sacs are derived from the jugular veins and then extend to the head, neck, thorax and arms. The other paired sacs arise from the iliac veins, where they unite with the cardinal veins. These spread to the abdominal wall, the pelvis and the legs. These lymphatic vessels join the cisterna chyli and vary from others in that they are not connected with veins. The unpaired lymphatics develop from the vena cava and mesonephric veins to form the retroperitoneal lymph sac. These lose their vein connections and join the cisterna chyli. Veins from the wolffian

bodies form sprouts to develop as the cisterna chyli.

The lymph nodes primarily develop in capillary plexuses at the third month of fetal life. The lymphocytes are already present and these probably form lymph masses and the lymphoid nodules. The mesenchyma forms a connecting tissue capsule. Trabeculae grow from this. The capillaries arise first, from which larger vessels lead to other vessels or nodes. The deeper plexuses develop first, both throughout the body and in the intestines, and gradually the superficial thoracic vessels are formed. All the lymph sacs except the cisterna chyli are divided by connective tissue bridges, which help to form the lymph glands. The cisterna chyli in the lower part becomes a gland, but its upper portion remains as a conduit or cisterna.

Anatomy For descriptive purposes, the lymphatic system may be divided into four parts: the lymphatic fluid, the capillaries, the collecting vessels including the thoracic ducts, and the lymph glands (Fig. 26).

Lymph is a clear yellow substance, similar to but more dilute than blood plasma. It has a specific gravity of approximately 1.015. Its contents are lymphocytes and red blood corpuscles. The lymph probably accumulates by an osmosis-diffusion-filtration process from the tissue fluids. It is not the same as tissue fluid, however.

The *capillaries* are lined by flat endothelial cells, which lie in connective tissue regions throughout the body in areas bathed by intracellular tissue fluids. These lymphatic capillaries anastomose widely and they do not have a set size or form. Many of these capillaries have blind ends. The plexuses have both superficial and deep layers, the superficial one being the smaller. In the skin, lymphatic capillaries are found in the dermis. The epidermis is without lymphatic capillaries which is of surgical interest. The subcutaneous tissue normally does not have any capillaries. Muscles, tendons, their sheaths, the perosteum and the joints all have lymphatic capillaries in abundance. The periosteum, the peritoneum, the pericardium and the pleura also have rich lymphatic components. In the digestive tract, just below the epithelium, are both superficial and deep lymphatic plexuses. The papillae appear to extend in blind-end formations into the villi of the small intestine. The lacteals arise in the lymphatic spaces in the intestinal villi.

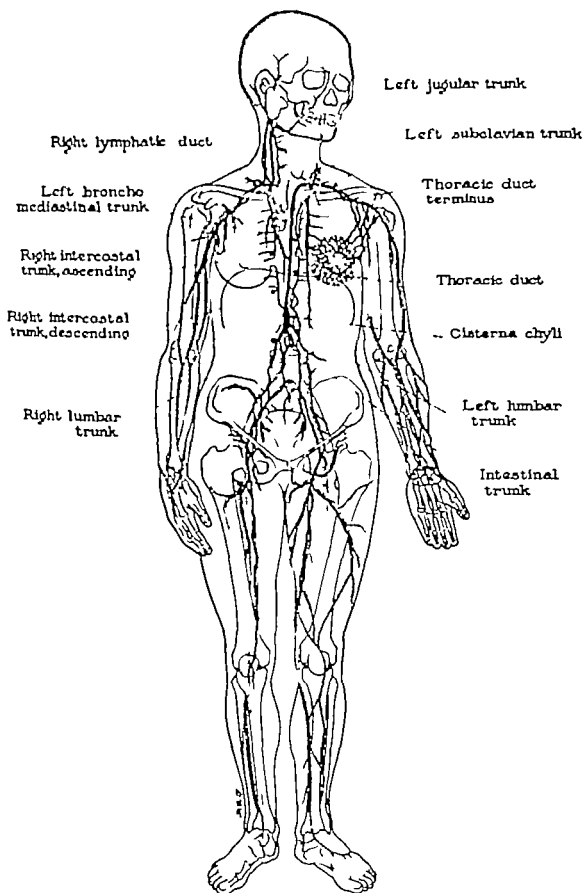


Figure 20 Diagrammatic drawing of the lymphatic drainage of the body

The salivary glands, the liver the spleen the adrenals and the kidney have large supplies of lymphatic capillaries. The nasal, tracheal and bronchial trees have a wide distribution of lymphatics within them. The bladder the prostate testes, ovaries and uterus all have a wealth of plexuses. The heart is also abundantly supplied with lymphatics which lie beneath the endocardium and throughout the myocardium. Lymphatics are not seen in the bone marrow or in cartilage. The central nervous system is not supplied with lymphatics. Other tissues without lymphatic capillaries are the liver lobule the meninges, the eye, the internal ear most striated muscle, the spleen pulp, kidney bone marrow cartilage and epidermis.

The lymphatic vessels collect the fluid from the lymphatic capillaries. All parts of the body with the exception of the non-vascular areas contain lymph vessels. These exceptions are the hair nails, cuticles and cartilages. The lymph vessels have an internal, a middle and an external coat. The internal coat is a layer of elongated endothelial cells, supported by an elastic membrane. The middle coat is of smooth muscle, except in the smaller vessels. The external coat has some muscle fibers but there is mainly a connective tissue covering which becomes firmer as the vessels increase in size. In construction, the thoracic duct, with a subendothelial layer of muscle and elastic fibers is more like a blood than a lymphatic vessel. The blood and nerve supplies to the lymphatics enter into the outer and middle coats.

The valves of the lymphatic vessels originate from the fibrous tissues that have an endothelial covering similar to that of veins. These valves are of a semilunar type and they are placed much closer together than are the valves of the vein. The number of valves increases as one approaches a lymph gland. They are fewer in number in the lower extremity as compared to the upper extremity or the head and neck. The vessels pouch above the valves and when distended they appear knotted in shape.

The superficial lymph vessels accompany the veins, with which they perforate the deep fascia. They lie in the submucosal fatty tissue throughout the digestive, respiratory and genitourinary tracts and in the thoracic and abdominal walls just below the serosa.

The lymphatic vessels are plexiform networks, these networks being larger than those of the capillary plexus. They form

small vessels which pass to a neighboring gland or from a larger lymphatic trunk. The deep lymphatic vessels are larger but fewer in number and they also accompany the blood vessels. The lymphatic vessels are more numerous than veins but are smaller in size. The vessels with which they anastomose are frequently of the same size.

The *cisterna chyli* is formed from the intestinal trunk which drains the lymphatics from the stomach and intestine, the pancreas, spleen and from the lower and forward part of the liver. It is joined by the right and left lumbar lymphatic trunks formed by the vessels from the aortic lymph nodes and the lower limbs and the walls of the pelvis kidneys and adrenal glands. The lymphatics of the abdominal wall also open in this area.

Through the *thoracic duct* the lymph and chyle are carried from the tissues and organs into the blood stream. It is a final collecting place for all of the lymph in the body except that from the right side of the head, neck and thorax, from the right arm, right lung, right side of the heart and the convex side of the liver. It begins at the second lumbar vertebra, to the right of and behind the aorta. There is a descending trunk from the posterior intercostal lymph glands, a trunk which drains the upper lumbar lymphatic glands, and a trunk from the posterior mediastinal lymph glands and the intercostal lymph glands of the lower and upper intercostal spaces which join the thoracic duct just above the *cisterna chyli*. The thoracic duct passes through the diaphragm with the aorta and ascends between the aorta and the azygos vein. At the level of the fifth thoracic vertebra, it passes to the left and ascends behind the aortic arch and the left subclavian artery between the esophagus and the left pleura. The left jugular and left subclavian trunks also join the thoracic duct, as may the left bronchomediastinal trunks. In the neck, the duct arches to approximately 3 cm. above the clavicle and crosses in front of the subclavian artery the vertebral artery and vein and the thyrocervical trunk and opens into the left subclavian vein at its junction with the left internal jugular vein.

The right lymphatic duct drains the areas on the right already described and opens into the right subclavian vein at its junction with the right internal jugular vein.

The lymph nodes or glands are oval or round structures spaced in the channels of the lymph vessels. Their size varies between that of a bean and that of an olive. The afferent lymph vessels enter at various parts

of the periphery. The efferent vessels leave at the hilum which is the same site through which blood vessels and nerves enter and leave. The gland is made of packed lymphocytes massed between trabeculae from the capsule. Reticular fibers from the trabeculae further divide the gland into sinuses. The afferent vessels open into the subcapsular sinuses and the efferent arise from the medullary sinuses. The germ centers where the lymphocytes increase by division are in the center of the cortical sinuses.

Modern surgery in its effort to control infection and malignant disease requires the knowledge of the lymphatic drainage from specific parts and organs. Upon such knowledge depend the possibilities of cure and sometimes life or death.

Lymphatic supply of upper extremity. The lymphatics of the upper extremity are both superficial and deep and in general follow the course of the veins. The superficial lymph glands normally are few in

number. There are some supratrochlear glands above the medial epicondyle of the humerus which drain the third, fourth and fifth fingers, the medial side of the hand and ulnar side of the forearm afferently to the deeper vessels. There are some deltoid pectoral nodes near the cephalic nodes which also drain superficially.

The deep glands may have a few scattered nodes along the radial, ulnar and brachial arteries. The main deep nodes are the axillary nodes which are large and are twenty to thirty in number. They consist of five groups: (1) The anterior or pectoral consists of four or five nodes around the lateral thoracic artery at the lower border of the pectoralis minor muscle. They drain the anterior and lateral chest wall and the lateral and middle part of the breast efferently to the subclavian group. (2) The posterior or subscapular glands follow the subscapular artery. They drain the skin and muscles of the back of the neck and thorax

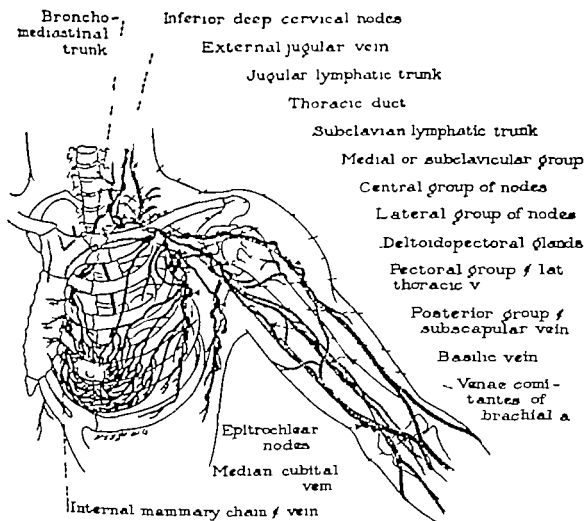


Figure 27. Lymph drainage of the axilla. Arrows indicate direction of flow. Note the five groups of lymph nodes and their vessels. Drainage to the lymphatic ducts to the neck and mediastinum makes eradication of malignant disease difficult once it has extended to the glands.

The salivary glands the liver the spleen the adrenals and the kidney have large supplies of lymphatic capillaries. The nasal tracheal and bronchial trees have a wide distribution of lymphatics within them. The bladder the prostate testes ovaries and uterus all have a wealth of plexuses. The heart is also abundantly supplied with lymphatics, which lie beneath the endocardium and throughout the myocardium. Lymphatics are not seen in the bone marrow or in cartilage. The central nervous system is not supplied with lymphatics. Other tissues without lymphatic capillaries are the liver lobule, the meninges, the eye, the internal ear, most striated muscle, the spleen pulp, kidney, bone marrow, cartilage and epidermis.

The lymphatic vessels collect the fluid from the lymphatic capillaries. All parts of the body with the exception of the non-vascular areas contain lymph vessels. These exceptions are the hair, nails, cuticles and cartilages. The lymph vessels have an internal, a middle and an external coat. The internal coat is a layer of elongated endothelial cells supported by an elastic membrane. The middle coat is of smooth muscle, except in the smaller vessels. The external coat has some muscle fibers, but there is mainly a connective tissue covering which becomes firmer as the vessels increase in size. In construction, the thoracic duct, with a subendothelial layer of muscle and elastic fibers is more like a blood than a lymphatic vessel. The blood and nerve supplies to the lymphatics enter into the outer and middle coats.

The valves of the lymphatic vessels originate from the fibrous tissues that have an endothelial covering similar to that of veins. These valves are of a semilunar type and they are placed much closer together than are the valves of the vein. The number of valves increases as one approaches a lymph gland. They are fewer in number in the lower extremity as compared to the upper extremity or the head and neck. The vessels pouch above the valves and when distended they appear knotted in shape.

The superficial lymph vessels accompany the veins, with which they perforate the deep fascia. They lie in the submucosal fatty tissue throughout the digestive, respiratory and genitourinary tracts, and in the thoracic and abdominal walls just below the serosa.

The lymphatic vessels are plexiform networks, these networks being larger than those of the capillary plexus. They form

small vessels which pass to a neighboring gland or from a larger lymphatic trunk. The deep lymphatic vessels are larger but fewer in number and they also accompany the blood vessels. The lymphatic vessels are more numerous than veins, but are smaller in size. The vessels with which they anastomose are frequently of the same size.

The *cisterna chyli* is formed from the intestinal trunk which drains the lymphatics from the stomach and intestine, the pancreas, spleen and from the lower and forward part of the liver. It is joined by the right and left lumbar lymphatic trunks formed by the vessels from the aortic lymph nodes and the lower limbs and the walls of the pelvis, kidneys and adrenal glands. The lymphatics of the abdominal wall also open in this area.

Through the *thoracic duct* the lymph and chyle are carried from the tissues and organs into the blood stream. It is a final collecting place for all of the lymph in the body except that from the right side of the head, neck and thorax, from the right arm, right lung, right side of the heart and the convex side of the liver. It begins at the second lumbar vertebra, to the right of and behind the aorta. There is a descending trunk from the posterior intercostal lymph glands, a trunk which drains the upper lumbar lymphatic glands, and a trunk from the posterior mediastinal lymph glands and the intercostal lymph glands of the lower and upper intercostal spaces which join the thoracic duct just above the *cisterna chyli*. The thoracic duct passes through the diaphragm with the aorta and ascends between the aorta and the azygos vein. At the level of the fifth thoracic vertebra, it passes to the left and ascends behind the aortic arch and the left subclavian artery between the esophagus and the left pleura. The left jugular and left subclavian trunks also join the thoracic duct as may the left bronchomediastinal trunk. In the neck, the duct arches to approximately 3 cm. above the clavicle and crosses in front of the subclavian artery, the vertebral artery, and vein and the thyrocervical trunk and opens into the left subclavian vein at its junction with the left internal jugular vein.

The *right lymphatic duct* drains the areas on the right already described and opens into the right subclavian vein at its junction with the right internal jugular vein.

The *lymph nodes* or *glands* are oval or round structures spaced in the channels of the lymph vessels. Their size varies between that of a bean and that of an olive. The afferent lymph vessels enter various parts

perior gastric nodes have upper nodes at the base of the left gastric artery a lower part accompanying the branches of this artery along the lesser curvature between the layers of the omentum and a paracardial group which resembles a chain of beads around the neck of the stomach. The inferior gastric nodes number from four to eight and lie between the layers of the greater omentum on the greater curvature near the pylorus. The hepatic glands have a hepatic group at the base of the hepatic artery and extend along the common bile duct in the lesser omentum. One node called the cystic gland, is at the neck of the gallbladder. The subpyloric hepatic nodes number about five and are located at the superior and descending parts of the duodenum at the bifurcation of the gastroduodenal artery. These nodes drain the stomach duodenum liver and pancreas and drain into the preaortic

nodes. The pancreaticolienal glands are on the upper and posterior border of the pancreas in relation to the splenic artery. They drain the stomach spleen and pancreas.

The *superior mesenteric glands* drain into the mesenteric ileocolic and mesocolic lymph nodes. The mesenteric glands number from 100 to 150 and lie in three areas near the terminal branches of the mesenteric artery at the secondary vascular loops and also along the main artery supply.

The *ileocolic nodes* which number from ten to twenty surround the ileocolic artery. They are grouped near the duodenum and also at the trunk of the artery. They divide into those called ileal glands which are located at the ileal branch of the artery; the anterior is in the ileocolic fold by the wall of the cecum and the posterior glands lie behind the cecum near the ascending colon. There is a single or double node in the

Celiac glands

Biliary nodes

Paracardial glands

Superior gastric glands

Suprapancreatic gl.

Splenic glands

Gastroepiploic gl.

Superior mesenteric glands

Preaortic glands

Mesocolic glands

Inferior mesenteric glands

Superior hemorrhoidal glands

Superficial inguinal nodes

Deep sub-inguinal nodes

Superficial sub-inguinal nodes

Cystic gland

Subpyloric glands

Ileocolic glands

Paracolic nodes

Appendicular gland

Hypogastric glands

External iliac nodes

Vesical glands

Figure 28. Lymphatics of the pelvis and abdomen. The lymphatic drainage from both the male and female genitalia and pelvis is superimposed. Note drainage of external genitalia through inguinal glands. The stomach, liver and colon have been divided to show the drainage of these and the other intestinal organs to the lymphatic glands which follow the major vessels. Arrows indicate direction of inguinal drainage. The irregularity of the lymphatic drainage illustrates the difficulty of the cure of malignant disease.

to the central axillary nodes (3) The five glands of the lateral group are located medially and behind the axillary vein. They drain the whole arm except the cephalic side and empty into the subclavicular axillary nodes and the inferior deep cervical nodes (4) The central or intermediate three or four nodes are deep in the base of the axilla. These drain all of the other groups to the subclavicular nodes (5) The subclavicular or medial nodes number from five to ten and are above and behind the pectoralis minor muscle. These glands drain all of the other axillary nodes. This makes them of utmost anatomic importance in cancer surgery, particularly of the breast. These glands drain the lymph conduits accompanying the cephalic vein and the upper and outer part of the breast. They unite to form a subclavian afferent vessel. This trunk may open directly into the subclavian and jugular veins or the jugular lymphatic trunk or on the left, the thoracic duct (Fig. 27)

The lymphatic vessels of the upper extremity are of both the superficial and deep types. The superficial vessels form trunks which accompany the cephalic, median and basilic veins. A few vessels on the ulnar side drain to the supratrochlear nodes. The others drain to the lateral groups of axillary nodes. A few empty to the subclavicular axillary nodes or inferior cervical nodes by way of the deltoid-pectoral nodes. The deep lymphatic vessels of the upper extremity follow the deep blood vessels and, in general, drain to the lateral axillary glands.

Lymphatic supply of lower extremity
The glands are the anterior tibial, popliteal and inguinal nodes. The anterior lie in the interosseous membrane and are surgically unimportant. The popliteal glands are five to eight glands in close approximation to the popliteal blood vessels; they drain the part of the leg corresponding to the lesser saphenous and popliteal veins and also the knee joint. Most of these glands are on the sides of the popliteal vessels and drain the tissues supplied by the anterior and posterior tibial vessels. The inguinal glands number from twelve to twenty and are in the femoral triangle. The superficial inguinal glands are just below the inguinal ligament, above the saphenous vein, and they drain the integument of the penis, scrotum, perineum, buttock and abdominal wall below the umbilicus. The subinguinal glands lie below the saphenous vein and have superficial and deep components. The superficial subinguinal glands drain the superficial tissue of

the leg and some of the genital areas. The deep subinguinal glands are under the fascia lata medial to the femoral vein, and they drain the penis, the clitoris and the superficial subinguinal glands.

The lymphatic vessels of the lower extremity are of surgical importance. They also are of both superficial and deep types. The superficial lymphatic vessels are composed of a medial larger group in which the vessels pass up with the great saphenous vein and into the subinguinal group of superficial glands. The lateral or fibular group of vessels arise from the lateral side of the foot and accompany the small saphenous vein to enter the popliteal glands.

The deep lymphatic vessels are few in number and accompany the corresponding deep blood vessels to the popliteal glands. The gluteal and ischial regions are drained by lymphatics which follow the blood vessels of the same name.

Lymphatic drainage of abdomen and pelvis (Fig. 28) The lymphatics of the abdomen and pelvis are of extreme surgical importance in the care of diseases or new growths of this area. The glands consist of the parietal and visceral nodes. The parietal glands which lie behind the peritoneum have as their subdivisions the external and common iliac, epigastric, iliac circumflex, hypogastric, sacral and the lumbar or aortic glands. The iliac, the epigastric, the circumflex and the hypogastric glands drain the areas supplied by the arteries of the same name. These glands are lateral to in front of and behind the aorta. The lateral aortic glands are in close approximation on the right to the inferior vena cava, the renal vein, the psoas muscle and the right crus of the diaphragm. On the left they follow the abdominal aorta and drain to the cisterna chyli. The pre-aortic glands, so important in the eradication of malignant disease, are the celiac and the superior and inferior mesenteric glands. These and the retro-aortic glands likewise drain into the cisterna chyli. These glands are named for the vessels around which they drain and their names indicate their anatomic location. They drain the viscera supplied by the celiac, superior and inferior mesenteric vessels. The visceral glands are related to the branches of these vessels.

The celiac glands are the gastric, the hepatic and the pancreaticocolic groups. The gastric glands are divided into the superior and inferior groups and they drain to the celiac part of the pre-aortic. The su

prior gastric nodes have upper nodes at the base of the left gastric artery a lower part accompanying the branches of this artery along the lesser curvature between the layers of the omentum and a paracardial group which resembles a chain of beads around the neck of the stomach. The inferior gastric nodes number from four to eight and are between the layers of the greater omentum on the greater curvature near the pylorus. The hepatic glands have a hepatic group at the base of the hepatic artery and extend along the common bile duct in the lesser omentum. One node called the cystic gland, is at the neck of the gallbladder. The subpyloric hepatic nodes number about five and are located at the superior and descending parts of the duodenum at the bifurcation of the gastroduodenal artery. These nodes drain the stomach, duodenum, liver and pancreas and drain into the pre-aortic

nodes. The pancreaticocolic glands are on the upper and posterior border of the pancreas in relation to the splenic artery. They drain the stomach, spleen and pancreas.

The superior mesenteric glands drain into the mesenteric ileocolic and mesocolic lymph nodes. The mesenteric glands number from 100 to 150 and lie in three areas near the terminal branches of the mesenteric artery at the secondary vascular loops and also along the main artery supply.

The ileocolic nodes which number from ten to twenty surround the ileocolic artery. They are grouped near the duodenum and also at the trunk of the artery. They divide into those called ileal glands which are located at the ileal branch of the artery the anterior is in the ileocolic fold by the wall of the cecum and the posterior glands lie behind the cecum near the ascending colon. There is a single or double node in the

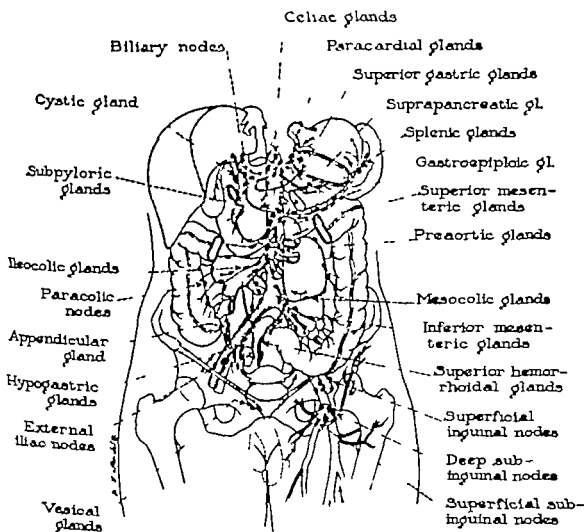


Figure 23. Lymphatics of the pelvis and abdomen. The lymphatic drainage from both the male and female genitalia and pelvis is superimposed. Note drainage of external genitalia through inguinal glands. The stomach, liver and colon have been divided to show the drainage of these and the other intestinal organs to the lymphatic glands which follow the major vessels. Arrows indicate direction of inguinal drainage. The irregularity of the lymphatic drainage illustrates the difficulty of the cure of malignant disease.

to the central axillary nodes (3) The five glands of the *lateral group* are located medially and behind the axillary vein. They drain the whole arm except the cephalic side and empty into the subclavicular axillary nodes and the inferior deep cervical nodes. (4) The *central or intermediate* three or four nodes are deep in the base of the axilla. These drain all of the other groups to the subclavicular nodes. (5) The *subclavicular or medial nodes* number from five to ten and are above and behind the pectoralis minor muscle. These glands drain all of the other axillary nodes. This makes them of utmost anatomic importance in cancer surgery, particularly of the breast. These glands drain the lymph conduits accompanying the cephalic vein and the upper and outer part of the breast. They unite to form a subclavian afferent vessel. This trunk may open directly into the subclavian and jugular veins or the jugular lymphatic trunk or on the left, the thoracic duct (Fig. 27).

The *lymphatic vessels* of the upper extremity are of both the superficial and deep types. The superficial vessels form trunks which accompany the cephalic, median and basilic veins. A few vessels on the ulnar side drain to the supraclavicular nodes. The others drain to the lateral groups of axillary nodes. A few empty to the subclavicular axillary nodes or inferior cervical nodes by way of the deltoid pectoral nodes. The deep lymphatic vessels of the upper extremity follow the deep blood vessels and, in general, drain to the lateral axillary glands.

Lymphatic supply of lower extremity. The glands are the anterior tibial, popliteal and inguinal nodes. The *anterior* lie in the interosseous membrane and are surgically unimportant. The *popliteal* glands are five to eight glands in close approximation to the popliteal blood vessels; they drain the part of the leg corresponding to the lesser saphenous and popliteal veins and also the knee joint. Most of these glands are on the sides of the popliteal vessels and drain the tissues supplied by the anterior and posterior tibial vessels. The *inguinal glands* number from twelve to twenty and are in the femoral triangle. The *superficial inguinal glands* are just below the inguinal ligament, above the saphenous vein, and they drain the integument of the penis, scrotum, perineum, buttock and abdominal wall below the umbilicus. The subinguinal glands lie below the saphenous vein and have superficial and deep components. The *superficial subinguinal glands* drain the superficial tissue of

the leg and some of the genital areas. The *deep subinguinal glands* are under the fascia lata, medial to the femoral vein, and they drain the penis, the clitoris and the superficial subinguinal glands.

The *lymphatic vessels* of the lower extremity are of surgical importance. They also are of both superficial and deep types. The superficial lymphatic vessels are composed of a medial larger group, in which the vessels pass up with the great saphenous vein and into the subinguinal group of superficial glands. The *lateral or fibular* group of vessels arise from the lateral side of the foot and accompany the small saphenous vein to enter the popliteal glands.

The deep lymphatic vessels are few in number and accompany the corresponding deep blood vessels to the popliteal glands. The gluteal and ischial regions are drained by lymphatics which follow the blood vessels of the same name.

Lymphatic drainage of abdomen and pelvis (Fig. 28) The lymphatics of the abdomen and pelvis are of extreme surgical importance in the cure of diseases or new growths of this area. The glands consist of the parietal and visceral nodes. The *parietal glands* which lie behind the peritoneum have as their subdivisions the external and common iliac, epigastric, iliac circumflex, hypogastric, sacral and the lumbar or aortic glands. The iliac, the epigastric, the circumflex and the hypogastric glands drain the areas supplied by the arteries of the same name. These glands are lateral to, in front of and behind the aorta. The *lateral aortic glands* are in close approximation on the right to the inferior vena cava, the renal vein, the psoas muscle and the right crus of the diaphragm. On the left they follow the abdominal aorta and drain to the cisterna chyli. The *paraortic glands*, so important in the eradication of malignant disease, are the celiac and the superior and inferior mesenteric glands. These and the retroaortic glands likewise drain into the cisterna chyli. These glands are named for the vessels around which they drain and their names indicate their anatomic location. They drain the viscera supplied by the celiac superior and inferior mesenteric vessels. The *visceral glands* are related to the branches of these vessels.

The celiac glands are the gastric, the hepatic and the pancreaticocolic groups. The *gastric glands* are divided into the superior and inferior groups and they drain to the celiac part of the 1- The su-

Lymph vessels of the *omentum* are large and have a complex anastomosis and many valves. They vary in size depending upon the diseases to which the patient has been subject. The lymph vessels follow the arteries and veins.

The lymphatic vessels of the *urinary organs* are divided into those of the kidney, ureter, bladder, prostate and urethra. The lymphatics of the *kidney* are in three plexuses, one in the substance of the kidney, a second below its capsule and a third in the surrounding fat. The vessels from the kidney substance converge into trunks which pass through the hilum. They join the lymph vessels from the subcapsule plexus and follow the course of the renal vein to end in the lateral aortic glands. The perinephric lymph vessels also drain to these same glands. The direction of the lymphatic vessels of the ureter depends on the part of the ureter involved. The upper *ureter* drains into the efferent vessels of the kidney and also into the aortic glands. Those vessels from the lower portion but above the brim of the pelvis, drain into the common iliac glands. The lymphatics from the lower or intrapelvic portion drain mainly into the hypogastric glands.

The lymphatic vessels of the *bladder* drain the lymph from the intramural and extra-muscular tissues. The mucosa apparently does not have a lymphatic supply. The lymphatic vessels drain from the anterior surface of the bladder into the external iliac glands. These lymphatics have minute glands situated in their course which are called the anterior and lateral vesical glands. The lymphatic vessels from the back of the bladder pass to the hypogastric and external and common iliac glands.

The lymphatics of the *prostate* drain into the hypogastric and sacral glands. There is one vessel from the back of the prostate which drains to the external iliac nodes and one from the anterior part drains to the lymph vessel from the membranous urethra. The drainage in part is similar to that of the urethra. The lymph vessels of the *urethra* are accompanied by those from the glans penis and drain to the subinguinal as well as the external iliac glands. The membranous and prostatic portions of the urethra in the male, and the entire urethra in the female are drained to the hypogastric glands.

The lymphatic vessels of the *testis* and *ovary* consist of both superficial and deep parts and are anatomically similar. These drain the tunica vaginalis and epididymis in

the male and the ovary in the female. The vessels form trunks which accompany the spermatic vein in the spermatic cord in the male and the ovarian vessels in the female to the lateral and pre-aortic lumbar glands. The lymphatic vessels of the *uterine tube* drain partly with the ovarian and partly with the uterine vessels.

The lymphatic vessels of the *uterus* consist of a superficial vessel under the peritoneum and a deeper one in the uterus itself. These lymphatics become greatly enlarged during pregnancy. They drain through the broad ligaments and are continued up to the ovarian vessels and then to the lateral and pre-aortic glands. A few vessels run to the external iliac and superficial inguinal glands. The lymphatics of the *cervix* drain to the external iliac glands, hypogastric glands and posteriorly to the common iliac glands.

The lymphatics of the *vagina* drain according to the part of the vagina involved. The vessels of the upper vagina lead to the external iliac glands, those of the middle part to the hypogastric glands and those of the lower part to the common iliac glands. These lymphatic vessels anastomose with those of the cervix, the vulva and the rectum, but not with those of the bladder, a point which is important at times surgically. The lymphatics of the *vulva* drain to the superficial inguinal glands.

Lymphatic drainage of the thorax (Fig. 29). There are two types of lymphatic nodes in the thorax—the *parietal* and the *visceral*.

The *parietal glands* are composed of the sternal, intercostal and diaphragmatic nodes. The sternal glands are in the intercostal spaces in close approximation to the internal mammary artery. They drain, to some extent, the breast, the deep structures of the anterior upper abdominal wall and, in part, the upper surface of the liver. These glands may drain directly to the subclavian or internal jugular veins or the thoracic ducts. The intercostal glands are in intimate association with the intercostal vessels. They drain the deep lymphatics from the sides of the chest and their lymph channels form a descending trunk to the cisterna chyli or thoracic duct. The upper glands drain to the thoracic duct on one side and to the right lymphatic duct on the other side. The diaphragmatic glands have medial anterior and posterior components. The anterior nodes are behind the base of the xiphoid. They drain the vessels from the upper part of the liver and the front part of the diaphragm and pass effer-

mesentery of the appendix as well as a few nodes called the right colic glands which lie medial to the ascending colon.

The *mesocolic nodes* are numerous and lie in the layers of the transverse mesocolon near the hepatic and splenic flexures. They drain into the preaortic nodes.

The *inferior mesenteric nodes* form one group on the branches of the left colic and sigmoid arteries. A second collection of nodes surrounds the superior hemorrhoidal artery in the sigmoid mesocolon. A third or perirectal group is in and around the muscular coat of the rectum. These glands drain the descending and sigmoid colons as well as the upper rectum and drain to the lower preaortic nodes.

Lymph vessels of the abdominal and pelvic viscera. The lymphatic vessels of the digestive organs below the diaphragm drain to the preaortic glands, following the course of the blood vessels which supply these organs. They are important because of the part they play in the spread of cancer or infection.

The lymphatic vessels of the stomach join above with the vessels of the esophagus and below with those of the duodenum. Those near the left gastric artery drain to the superior gastric nodes. The fundus and body of the stomach to the left of a line projected by the esophageal junction drain to the pancreaticocolic nodes. The vessels to the right of this point as far as the pylorus drain to the inferior gastric nodes and thence to the subpyloric nodes. The pylorus is drained by lymph vessels to the superior gastric nodes by way of the hepatic and subpyloric nodes. From this complex lymph node and vessel distribution the difficulty of eradicating malignant disease of the stomach, once it has extended, is evident.

The *duodenal lymph vessels* drain anteriorly and posteriorly to the pancreaticoduodenal nodes between the head of the pancreas and the duodenum and then to the hepatic and preaortic nodes.

The lymphatic vessels of the *jejunum* and *ileum* are familiar to all surgeons. During intestinal digestion they become distended with milky lymph fluid. These vessels are termed lacteals. They arise in the lymphatic spaces in the intestinal villi. They then course between the layers of the mesentery of the small bowel and converge to the appropriate mesenteric nodes. These small bowel nodes drain efferently to the preaortic nodes and then to the cisterna chyli.

The *appendix* and *cecum* are drained by

multiple lymph vessels which end in the ileocolic chain. There may be some lymph nodes at the peritoneal fold. These vessels form an anterior and posterior group, the anterior going to the ileocolic nodes and the posterior passing over the back of the cecum.

The *right and transverse colon lymph vessels* pass through the right and transverse mesocolon to the mesocolic nodes. The *descending and sigmoid colon vessels* drain to the left colic and sigmoid nodes. They end in the preaortic lymph nodes.

The lymph vessels from the *rectum* pass through the perirectal glands to the sigmoid and mesocolon glands. From here they go to those preaortic nodes located near the inferior mesenteric artery. The *anal lymphatic vessels* drain to the superficial inguinal nodes. The *anal canal lymphatic vessels*, however, follow the middle and inferior hemorrhoidal arteries and terminate in the hypogastric nodes.

The *liver* is drained by both superficial and deep lymphatics. The superficial lymphatic vessel drainage from the liver divides itself naturally into a superior and inferior part corresponding to the convex and lower surfaces of the liver. Some lymph vessels on the upper surface of the right and left lobes form a trunk which passes through the diaphragm with the vena cava and ends in glands near that organ. A few vessels go through the esophageal opening in the diaphragm to the paracardial glands in the superior gastric nodes. Some vessels from this side end in the preaortic nodes near the celiac artery. Another trunk from the upper right and left lobes ends in the hepatic nodes. Some lymph vessels from the under surface of the liver accompany the deep lymphatics to the hepatic nodes while one vessel goes through the diaphragm to the vena cava nodes. The deep lymphatic vessels form ascending and descending trunks, the former draining to the inferior vena cava area and the latter to the hepatic nodes. The *gallbladder* drains to the hepatic nodes as does the bile duct. The latter may drain also to the pancreaticoduodenal nodes.

The lymph vessels of the *pancreas* drain mainly to the pancreaticocolic nodes, but a few end in pancreaticoduodenal and preaortic nodes.

The lymphatic vessels of the *spleen* pass to the pancreaticocolic glands. The *suprarenals* accompany the suprarenal veins to the lateral aortic nodes, but occasionally some vessels drain to the posterior mediastinum above the diaphragm.

The *superficial lymphatic vessels* of the wall of the thorax are situated just below the skin and converge at the axillary glands. Those over the muscles drain to the subscapular, the pectoral and the sternal glands. A few of the lymphatic vessels in the pectoral area drain to the supraclavicular cervical glands.

The lymphatic vessels of the *breast* arise in the interlobular spaces and on the walls of the milk ducts. A plexus is formed for those in the central part directly below the areola and nipple, and these join and pass to the pectoral group of nodes in the axillary glands. The medial breast vessels pierce the chest wall and drain to the sternal glands or occasionally to the subclavicular glands. The lymph vessels of the lateral and medial parts of the breast drain to the subclavicular axillary glands. The upper and outer part of the breast also is drained to the subclavicular lymph nodes.

The *deep lymphatic vessels* of the thoracic wall are the lymphatics of the muscles on the ribs, the intercostal lymphatic vessels and the lymphatic vessels of the diaphragm. The lymphatics of the muscles on the ribs drain to the axillary glands and a few to the sternal glands. The intercostal lymphatic vessels drain both the intercostal glands and parietal pleura and empty into the intercostal glands. The internal intercostal lymph vessels and those from the pleura drain to the sternal glands. The lymphatic vessels of the diaphragm form a thoracic and an abdominal plexus. The plexus on the thoracic side drains to the glands near the junction of the seventh rib and its costal cartilage. They then pass to the glands on the esophagus, to those nodes around the termination of the inferior vena cava and to the glands which surround the aorta. The lymph vessel plexus on the abdominal surface of the diaphragm is drained in two ways. The vessels from the right half of the diaphragm drain to the glands near the inferior phrenic artery and the right lateral aortic glands. Those from the left half of the plexus pass to the pre-aortic and lateral aortic glands and to glands at the terminal portion of the esophagus.

The lymphatic vessels of the *heart* are of the deep and superficial types, the deep being under the endocardium and the superficial close to the visceral pericardium. The deep and superficial plexuses form a right and left collecting trunk. The *left trunk* drains both ventricles and rises in the anterior longitudinal sulcus. It receives a diaphragmatic trunk at the coronary sulcus and

the single lymph vessel thus formed passes to the tracheobronchial nodes. The *right trunk* drains the right atrium and the diaphragmatic side of the right ventricle. It ascends the posterior longitudinal sulcus and the coronary sulcus to the tracheobronchial nodes.

The *lungs* similarly have a superficial and a deep lymph plexus and unlike other plexuses, they do not anastomose to any great extent. This is of surgical importance. The superficial plexus is beneath the pleura and drains to the glands at the hilus of the lung. The deep plexus accompanies branches of the pulmonary vessels and the bronchi, either beneath the mucosa or around the bronchi. These vessels converge at the glands at the hilus and end efferently in the tracheobronchial glands. The alveolar walls of the lung have no lymph vessels.

The lymphatic vessels of the *pleura* drain the visceral part and end in the superficial efferents of the lung. The parietal pleura drains to the sternal glands, while the diaphragmatic part of the pleura is drained by the diaphragmatic chain. The lymph vessels of the mediastinal portion end in the posterior mediastinal glands.

The *thymus* lymph drains to the anterior mediastinal tracheobronchial and sternal glands.

The lymphatic vessels of the *esophagus* form a plexus around this organ and drain into the posterior mediastinal glands.

Lymphatic supply of the head and neck.

The *lymph glands of the neck* are in association with the salivary glands and the deep and superficial lymph plexuses. The *submaxillary nodes* vary in number from four to six. They are located in the submaxillary triangle and lie on the salivary glands of the same name. The middle node of Stahr is directly over the external maxillary artery at the mandible. The submaxillary nodes drain the cheek, the side of the nose, upper lip, side of the lower lip, the gums, the side of the tongue and the medial palpebral commissure. Lymph from the facial and submental nodes also drains to these glands. Efferently the lymph nodes drain to the upper deep cervical nodes. The *submental (suprahyoid) nodes* lie between the anterior bellies of the digastric muscles. They drain the lower lip, the floor of the mouth and the apex of the tongue. They drain to the submaxillary and deep cervical nodes. The *superficial cervical nodes* follow the external jugular vein. They drain the ear and parotid area to the superior deep cervical nodes.

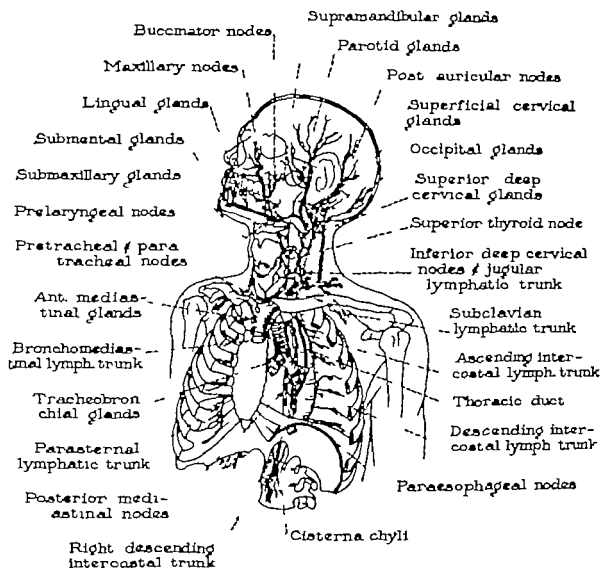


Figure 29 Lymph drainage of the thorax, thoracic duct, neck and head. The early involvement of the thoracic glands by cancer of the lungs reduces the possibility of cure in this disease. The large thoracic duct may be injured in its abdominal or thoracic portions. This results in chyle peritonæum or chyle thorax.

ently to the sternal glands. The medial glands are alongside the phrenic opening in the diaphragm and on the right may be in the sac of the pericardium. They drain the lymph from the middle diaphragm and, on the right, from the upper part of the liver. The lymph drains from these to the posterior mediastinal glands. The posterior glands are on the back of the crux of the diaphragm and drain the vessels from the lumbar nodes and efferently go to the posterior mediastinal nodes.

The *visceral lymph nodes* are named for their sites. They are the anterior mediastinal, posterior mediastinal and tracheobronchial glands. The anterior mediastinal nodes lie in front of the aortic arch and are in close relation to the innominate veins. They drain the lymph from the thymus, pericardium and

the sternal glands. Their vessels form, with the tracheobronchial ones, the right and left bronchomediastinal trunks. The posterior mediastinal glands are behind the pericardium close to the esophagus and the descending aorta. They drain efferently the esophagus, the posterior part of the pericardium, the diaphragm and the convex surface of the liver. Efferently they end in the thoracic duct or in the tracheomediastinal trunk. The tracheobronchial glands drain the lungs, the bronchi, the trachea and the heart and receive the drainage from the posterior mediastinal glands as well. Their lymph vessels join the internal mammary vessels and the anterior mediastinal glands to form the right and left bronchomediastinal trunks. They may drain into the thoracic ducts or directly into the subclavian or jugular veins.

the lymphatics and the part it plays in the spread or control of infection and malignant disease. In addition specific diseases of the lymphatic system and obstruction of the lymphatic flow become surgically important. Functional failure of this lymph drainage creates lesions which are disabling and disfiguring. Many of these diseases are amenable to surgery.

The lymphatics from the ileum and jejunum, called lacteals, collect the products of fat digestion and with the other intestinal lymphatic drainage carry these into the receptaculum chyli. This flow with the lymph from the rest of the body then goes to the subclavian veins at their junction with the jugular veins, by means of the thoracic and right lymphatic ducts. This lymph presents a surgical problem when obstruction, new growth or injury interrupts it. An injury may occur to the main conduits as the result of trauma or as a complication of a surgical operation. Most often serious injuries occur in the lower part of the neck and chest.

INJURIES TO THE LYMPH SYSTEM

The injuries to the lymph system may involve the glands or the lymph vessels. The latter injuries are the important ones. The large lymph ducts or the small lymphatic vessels may be the site of the injury.

Injuries to Thoracic and Lymphatic Ducts. The thoracic duct, which measures between 36 and 48 cm., runs from the second lumbar vertebra to the root of the neck. It is in intimate association with the vertebrae and from the fifth thoracic vertebra runs to the left side.

The duct may be injured by an open wound, an indirect bruising wound or most often, by a crushing wound. In the latter wounds, the other associated injuries take precedence in treatment and the lymph duct injury may be masked. The duct may be severed in part or entirely during a surgical operation. An extensive lymph node excision may cause severe duct lacerations. Burns and rays may cause duct injuries. The anatomic position demonstrates well the possibility of injury from trauma and in operations on the pleura and lungs, esophagus, sympathetic and large vessels of the neck.

If the lymph wound is an open one, a lymph fistula occurs with lymphorrhea (lymphorrhagia). The chyle will escape freely with each respiration.

The diagnosis of chyle thorax is made from the history of trauma, the x-ray appearance

of fluid and pressure or displacement and is confirmed by thoracentesis. Simple aspiration often will relieve the pressure and the laceration in the duct may close spontaneously.

The thoracic duct or the right lymphatic duct if injured at the time of an operation may be reanastomosed. In the case of the right duct this repair may not be necessary and a simple ligature or suture will resolve the process. Many times a ligation has stopped the lymph flow from the thoracic duct itself the duct continuing to carry the chyle through collateral lymph vessels or through a spontaneously created anastomosis. In crushing injuries, the treatment is directed to that of the other injuries. For the accumulation in the chest or abdomen thoracentesis or paracentesis will relieve the chyle pressure. The damaged duct usually repairs itself as the other wounds heal. The chyle thorax may require operative intervention because of pressure on or displacement of the chest or mediastinal contents. If the fluid continues to reaccumulate or body depletion from its loss is threatened, it can be repaired surgically after thoracotomy. If this is impossible technically, the duct should be ligated. The collateral circulation usually will carry the chyle through the opposite duct. Most injuries to the abdominal portion of the duct will heal spontaneously but if it continues to drain the duct may be ligated. One cannot overemphasize the damage done by the pressure of lymph in the thorax if unrelieved by aspiration.

Injuries to Small Lymphatic Vessels. The lymphatic vessels are injured with any laceration or incision. Usually extensive operations and especially lymph node dissections divide larger lymph vessels. Lymphedema occurs in these patients if a tissue space or cavity remains for lymph accumulation. External pressure will control most of these accumulations unless they are complicated by sepsis or unless the wound is drained. There is evidence that lymphatic collections and obstructions occur more often in drained than in undrained wounds.

Injuries to lymph vessels occur also from burns and rays from irradiation. The atomic explosions at Hiroshima and Nagasaki during World War II caused widespread damage to the lymphatics and the healing of the burns resulted in lymphedema and heavy keloids.

The fistulae caused by laceration of lymphatic channels of smaller size than the thoracic duct often close spontaneously. Lym-

The anterior cervical nodes have a superficial set which is on the anterior deep jugular vein and a deeper group which is in front of the larynx and trachea. These glands drain the larynx thyroid node and upper trachea to the deep cervical nodes.

The large and numerous deep cervical glands follow the carotid sheath from the skull to the thorax, hugging the pharynx, trachea and esophagus. The superior group lies under the sternocleidomastoid muscle around the accessory nerve and the internal jugular vein. The superior deep cervical nodes drain the occipital scalp the ear the back of the neck, part of the tongue the larynx, thyroid, trachea the nasal cavities, the nasal pharynx, the palate and esophagus. They also drain all the other glands of the head and neck except the lower deep cervical nodes. Efferently they form a trunk with the inferior deep cervical nodes and so-called jugular trunk. The inferior deep cervical nodes are in the supraclavicular triangle lateral to the sternocleidomastoid muscle. They are close to the brachial plexus and the subclavian vein. These nodes drain the back of the head and neck, the pectoral region and part of the arm. They also occasionally drain the upper surface of the liver. They receive drainage from the superior deep nodes and drain to join with a trunk from the superior nodes to form the jugular trunk. This trunk ends on the right side in the subclavian vein and on the left in the thoracic duct.

The skin and muscles of the neck drain to the deep cervical nodes. The pharynx drains to the retropharyngeal and the lower deep cervical nodes. The larynx in its upper part drains through the hyothyroid membrane to the superior deep cervical nodes. The lower laryngeal vessels go to the pretracheal and prelaryngeal nodes and a few of them pass between the cricoid and the first tracheal ring to the lower deep cervical nodes. The thyroid node drains to both the upper deep cervical nodes and to the paratracheal nodes.

Physiology The lymphatic system has two functions the first of which aids in digestion by the collection of chyle from the intestines and the transportation of this chyle through the lymphatic system to the veins. Its second function is to collect and return lymph from the other organs and the extremities. The lymphatic system also is a part of the body's defense. It contains and retains infection and new growth for a certain time. When its defense nature is overwhelmed, it appears to pass on or even to accelerate the spread of sepsis or malignant disease. In this way it

simulates a sponge, absorbing until it no longer can hold more at which time it appears to squeeze itself or to overflow. This saturation is not just a quantitative one but is qualitative as well, depending on the type of organism or cell its virulence or rapidity of growth or spread and the local defensive power of the lymphatics in the area involved.

The lymph system also collects foreign material. These substances are admitted to this closed system between the endothelial cells of the capillaries when these objects are pressed against the vessel surfaces. The muscular contractions motivate the lymph and this action is aided by the valvular system of the vessels. The degree of motion of the lymph stream varies with the individual's activities. Lymph will coagulate but it clots only one-fourth as rapidly as blood because of the lack of platelets in the lymph substance. Thrombosis occurs in the lymphatic vessels similarly to the thrombosis that occurs in veins. The inflammation to which patients with lymphedema are subject destroys the valves of the lymphatics, again in a similar manner to the way in which the valves of the veins are destroyed in thrombophlebitis.

The lymph circulation consists of an elaborate system of collecting vessels which drain the lymphatic fluid from the capillary networks. This fluid is collected from nearly all the organs of the body and its tissues by lymph capillaries. The lymph then passes to ever-enlarging vessels until it eventually reaches the thoracic ducts, which carry it to the venous system. At various places in this dense network are located the lymph glands or lymph nodes. The number of these glands depends upon the local lymphatic function and changes due to disease in which the lymphatic system plays a unique part. These are spaced in the path of the collecting vessels and appear to do two things: they filter out pathologic cells and organisms, passing on lymph fluid in a more purified state, and they add lymphocytes to this circulating fluid.

The function of the lymphatic system is to return lymph fluids from the tissues, organs and serous cavities and chyle from the alimentary tract to the general circulation. The central depository is the circulating blood. This transfer is made by a convergence of the lymphatic conduits into the thoracic duct on the left and the lymphatic duct on the right. The fluid is isotonic with blood. A surgical problem is posed by the drainage of

nese during World War II certain staging areas, the Samoan and Wallis Islands for example used for final training prior to the invasion of the Islands had epidemic and endemic filariae-infected natives. The marines and sailors fraternized with the natives. In one year (1943) 6000 trained troops had to be evacuated because of filariasis of the legs or genitalia following lymphatic obstruction. Rigid quarantine dropped this number in one year to 10 per cent of that figure.

The tuberculosis and syphilitic organisms cause similar invasions, infections and reactions.

INFECTIOUS LYMPHEDEMA. The lymphatic system is both a block to and pathway for the spread of infection. The ordinary commensal organisms such as the streptococci and staphylococci may increase in virulence and the hosts defense may be overcome; these organisms then enter the lymph vessels. They locally inflame these vessels and spread to the regional lymph nodes. These nodes filter out the infection and retain it unless the sepsis overwhelms them. In the latter event, the organisms may pass on to other lymph vessels and eventually to the blood stream. Such infections usually cause localized lymphedema. If the infection is continued or recurrent, fibrosis and scarring within the lymphatics is the end result.

In their invasion, these organisms may cause sufficient inflammation to obstruct the lymphatics and destroy their valves.

Repeated infections invariably cause low grade changes in the glands. These glands, in themselves, may keep the infection active or recurrent, similar to the situation in tonsillitis.

ALLERGIC LYMPHEDEMA. Some individuals are sensitive to certain pollens, proteins or environmental changes which affect the lymphatics and precipitate lymph blocks. An example is the allergic reaction to exposure to cold. A type of temporary block is due to massive hives. Such diathesis rarely is the cause of permanent lymphedema but such reactions if extensive can produce lymphedema.

POST THROMBOTIC LYMPHEDEMA. Thrombosis of the deep or superficial veins causes edema which throws an overload on the lymphatic component. There may be a damming back of the lymphatics, with permanent lymphedema. When the major venous vessels are clotted, rarely are there sufficient collaterals to return the fluid. Unless gravity drainage is utilized, a massive swelling must



Figure 30 Typical post thrombotic lymphedema. Massive lymph stasis secondary to obstruction of the deep vein system. The original lymph stasis is a spastic obstruction, but after a time this becomes an organic obstruction with the destruction of the lymph vessel valves (Pratt, G. H. J.A.M.A., vol. 151)

occur (Fig. 30). This results in the post phlebotic leg, or milk leg. Even if the veins recanalize at a later date, their valves are defective and they do not function adequately again. The fluid which has accumulated has created a space between the skin and fascia which, if not obliterated, becomes permanent. Fibrous trabeculae form in this space and, in the late stage, while elevation may drain some of the fluid, the heavy thickened tissue space remains.

TRAUMATIC LYMPHEDEMA. Lacerations, thrombosis, burns, operation incisions, keloids and the scarring which results from roentgen radium and radioactive isotope rays may destroy the continuity of the lymph channels. If the injuries are extensive enough, massive lymph collections follow because of the inability of the lymphatics to carry away the lymph fluid. The development of the fluid may be delayed until the scars have contracted, this final scarring obliterating the remaining collateral lymphatics.

CONGENITAL LYMPH COLLECTIONS. Many abnormal lymph collections have been reported. These may be due to lymph rests, cystic hygroma, simple lymph cysts, cavern

phorrea may be stopped by a pressure dressing. This therapy applies to the lymph drainage after gland resections in the neck, groin or axilla. Often the accumulation is considered purulent erroneously and a surgeon may continue to drain this fluid thus keeping its fistulous nature active. The best treatment is compression in the form of a bandage.

LYMPHEDEMA (LYMPH STASIS, ELEPHANTIASIS OR LYMPH OBSTRUCTION)

Lymphedema in the Lower Extremity
Since the lymphatic system is a closed one with its own spaces, fluid and vessels which begin as the lymph capillaries and end in the venous system, any obstruction to this system causes lymph accumulation. Such a collection, by its size and its complications, will cause increasing disability. Lymph obstruction always is accompanied by dilatation of the venous drainage system, which makes an effort to take over part of the fluid return from the involved tissues. The lymphatic obstruction, by its massive pressure, causes deleterious skin changes and distention or destruction in the other tissues, often with atrophy or even displacement of the muscles. The lymph vessels involved in lymphedema are those in the dermis, but not the epidermal layer of the skin, since the epidermis does not have lymphatics. The fluid collection extends into the subcutaneous fat and deep fascial layers. The muscle lymphatics usually do not become involved in this process because of their protected position.

The lymphatic drainage channels may be closed by a congenital defect or they may become obstructed secondarily by inflammation or internal or external pressure. Lymphedema may be localized due to a block in the main lymphatics, or it may be generalized with all of the lymphatics of the part obstructed. When the extremity or part is elevated, the fluid will flow by gravity through the subcutaneous tissues to areas where there are no lymphatic blocks and the lymph then will be carried away. This method of drainage occurs only when there is elevation of the part. With dependency the lymph reaccumulates, since there are no physical pathways for its removal. With failure of lymph drainage, the part becomes susceptible to inflammation caused by the usual contaminant organisms. Each inflammation causes further scarring and more lymph vessel obstruction. The patho-

logic process thus becomes self-perpetuating.

Etiology Any disease or trauma which directly or indirectly obstructs the lymphatic canals or destroys their valves may cause lymphedema. The causes of this condition thus are innumerable. A congenital or developmental failure of the lymph vessels or their valves may be responsible for the lesion. In some patients the lymph stasis seems to be due to an endocrine dysfunction. A specific or nonspecific infection in the lymphatic system, an injury, a burn and a primary or secondary new growth are the usual acquired causes. Thrombophlebitis, in addition to producing tissue space fluid accumulation, also causes lymphedema. The originating or precipitating factors in the development of lymphedema have been classified into eight types: specific lymphedema, infectious lymphedema, allergic lymphedema, post-thrombotic lymphedema, traumatic lymphedema, congenital lymph collections, malignant lymphedema and essential lymphedema.

SPECIFIC LYMPHEDEMA. Certain organisms have a predilection for invasion and inflammation of the lymphatic channels. Of these, the filarial worm (*Wuchereria bancrofti*), the tuberculosis organism (*Mycobacterium tuberculosis*) and the *Treponema pallidum* which causes syphilis are examples.

A mosquito, the *Stegomyia pseudoscutellaris* or *Culex fatigans*, picks up the worm, the *Wuchereria bancrofti*. The larvae are deposited and infected by the penetration of the bite of the mosquito. Adult filariae live in the lymph channels and discharge embryos. The dead worms also may cause a lymphatic inflammation. Streptococci or other pyogens frequently cause an associated suppurative infection. The lymph vessels become filled by organisms and inflammatory cells and this causes secondary lymph collections in the tissues. The lymph glands are infected and enlarged. The filariae produce recurrent febrile attacks, each one resulting in an inflammatory reaction with more pathologic changes.

Lymphedema due to filarial worm invasion has had historical military implications. The Roman Legions fought natives in Africa who had these grotesque obstructions which caused disfigurement of the limbs and genitalia. The term "elephantiasis" originated in their reports on these natives. Not only the skin resembled that of the elephant with its thickening and wrinkling, but the affected parts were grotesquely large. In the American campaign in the Pacific against the Japa-

the dependent medial areas. In some patients the collections are most irregular with overhanging masses of tissue which may obscure the normal contour of the extremities. In the enlargements of the upper extremity the mass may have involved even the fingers. In some of the massive collections, the normal position of the bones may have been displaced.

The shiny skin may become thickened and roughened. It may resemble the skin of an elephant. The pores enlarge and develop a pitted appearance. There is usually a pigment deposit, particularly in the lower extremity. The color varies from light yellow to all degrees of brown, purple and black. Ulcerations may be present because of the poor vascularity of the overdistended tissue. The associated venous involvement may be evidenced by large veins, dermatitis and ulcers. Dermatitis, especially of the fungus type, is often associated. Often there are weeping and eczematoid changes particularly around the toes. The skin in such areas will be heaped up and frequently is

draining. The nails usually show curvature irregularity and atrophy with associated fungus infection. There may be signs of repeated phlebitis.

The patients are subject to frequent attacks of sepsis. These bouts are characterized by high fever, chills, malaise and general upsets. The local reactions are similar to those of cellulitis, lymphangitis or erysipeloid reactions. The patient often gives a history of having had many attacks of "erysipelas." These reactions are due to lack of resistance of the part to the common skin contaminants, the streptococci and staphylococci. The limbs appear to lose their power to resist infection. Some patients have regularly recurring febrile attacks. There may be skip periods of freedom from fever and some patients may go for years without any septic attack. Such periods usually are associated with a time of excellent general health. Why some patients will have innumerable attacks and others none is not explainable. Each recurrence of the reaction increases the pathologic process, closing off

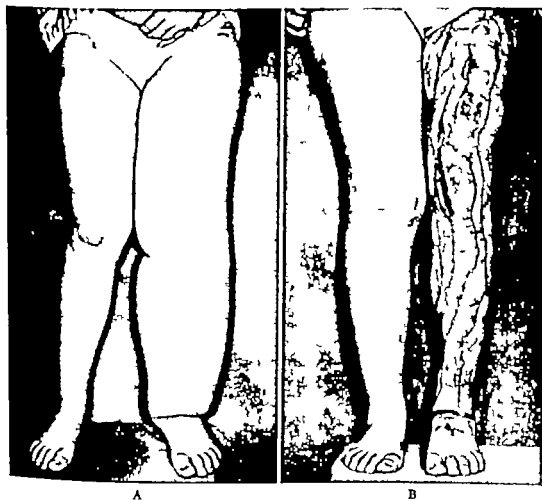


Figure 32. A, Extensive lymphedema of the Milroy type in a fifteen-year-old girl. Measurements: right calf 27.4 cm, thigh 56.0 cm; left calf 56.0 cm, thigh 66.6 cm. B, Postoperative view following operation. Scars can be covered by stocking or cosmetics.

ous lymph collections or lymphangiectasias. Abnormal lymph growths may interfere with obstetric delivery. Bizarre symptoms may develop, depending upon the location, size and extent of pressure.

MALIGNANT LYMPHEDEMA. Just as the lymphatics are the site of the spread of infection they are a pathway for the spread of malignant disease, particularly carcinoma. The rate of spread varies in different types of growths and in different individuals and there seems to be an inverse ratio of spread to age. The growth spreads along the lymphatics to the regional nodes which contain extensions for a time. The localization does not appear related to the size of the spread although it is related frequently to the length of time that the primary growth has existed. In some patients it appears that the regional nodes pass on the growth after their resistance has been overcome. The localization of growths in regional nodes varies greatly. The tumor may involve all of the nodes or only one or two. In some instances there is widespread dissemination with only minimal, or with no involvement of the regional nodes and some groups of nodes may be skipped with involvement of the others.

Sarcoma may spread by the lymphatic pathway although it most often extends by way of the blood stream. Melanoma frequently metastasizes by way of the blood stream. Sarcomas of the tonsil, thyroid or testicle are exceptions. The degree of involvement regionally often determines the size of the lymphedema. Secondary obstruction or overload of the venous drainage complicates the edema picture (Fig. 31).

Malignant tumors also may involve the lymphatics primarily: thus lymphosarcoma, Hodgkin's disease (malignant lymphoma) and lymphatic and leukocytic leukemia all may present lymphatic collections and secondary lymphedema.

ESSENTIAL LYMPHEDEMA. Some types of lymphedema are of unknown origin. These collections appear to be hereditary in nature, although most of them do not appear clinically until the time of puberty. The advent of the symptoms with this developmental changing time in life suggests that the maturing endocrine system, or some dysfunction of it, plays a part in precipitation of these lesions.

Some forms of congenital lymphedema also are known as Milroy's disease (Fig. 32) originally described as both a familial and congenital disease. Many of the patients in whom swelling of unknown origin

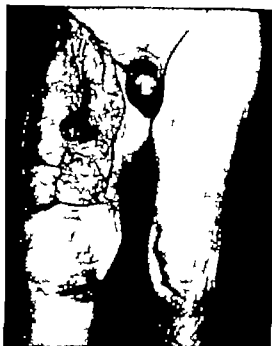


Figure 31 Malignant lymphedema secondary to liposarcoma of nine years duration.

appears at puberty do have a history of similar involvement occurring in other members of the family. Congenital lymphedema has been described as "simple" when the patient is the only one in the family so affected, and "hereditary" if other relatives are afflicted. There is some anatomic and physiologic defect in these patients from birth. It may be that a congenital swelling present from the day of birth is of such mild degree as to be not noticed until the time of more rapid growth. While the leg or legs are most often involved, the upper extremity may be affected as may the labia, scrotum and other male and female genitalia. The entire retroperitoneal space may be involved.

Symptoms The symptoms of lymphedema vary depending upon the degree of blockage, the age of the patient, the type of tissue involved, the secondary skin changes and whether the part has been subjected to repeated inflammations.

The usual picture is that of symmetrical enlargement of the extremity. In the leg, the swelling begins in the groin. It may be confined more to the upper or lower part of the limb but usually both portions are involved as are the foot and toes. In the congenital type, it most often involves one extremity both legs being implicated in only 25 per cent of the subjects. The skin is tight, tense and usually shiny. There may be wrinkled and lobular areas irregularly situated. The larger collections are in

months, years or indefinitely to maintain the control of the sepsis which is of great importance. This program of management of lymphedema due to infection is of help in the prevention of lymphedema due to other causes and has aided in controlling lymphedema seen after radical mastectomy in which infection and not malignant metastasis is the cause. Allergic lymphedema sometimes responds to desensitization. Antihistamines may be helpful. Patients with lymphedema due to cold exposure may be come conditioned by exposure to slowly lowered temperatures. Obstructions due to burns or scars require surgical correction. Lymphedema following irradiation in the absence of retained malignant disease is due to burns. Excision of the burned area and various plastic procedures may be helpful.

Lymphedema which follows the post thrombotic syndrome presents a complicated picture and its therapy is difficult. The treatment depends upon the stage of the disease which the patient presents. In the acute stage spasm is the most likely causative factor. For the patient with incompetent veins, ligation and stripping of these veins may be helpful. In all these patients venograms and lymphangiograms should be obtained routinely to determine the actual venous involvement and the adequacy of the remaining veins and lymphatics. For causalgia and the reflex spastic problems sympathectomy may be successful if paravertebral blocks produce a good clinical result.

In patients with mild residual lymph stasis, elevation of the part when the patient is not using it and good support when the part is dependent may be all that is necessary. Such therapy must be applied early and continued for as long as a year. Basically this therapy is planned to remove and keep the lymph fluid from the leg until such time as other lymph collaterals are developed or the lymph spaces are closed. This regimen consists of elevating the limb sufficiently high for gravitational drainage. This should be done each night and every time during the day that the limb begins to swell as shown by a tightness and discomfort of the bandage. Adequate support requires even application of a well fitted pressure bandage, reaching from the toes to as far as the swelling extends. The bandage must be removed and replaced each time that the part is elevated.

Massage, like exercise will cause lymph flow within the lymph channels. It will also

aid in the motivation and movement of extraluminal lymph. The massage should be from the distal to the proximal end of the swelling and should not be traumatic.

To the above measures must be added antibiotic therapy, care of fungus infection, treatment of any allied ulcers and sympathetic nerve blocks or sympathectomy for those in whom there is a large reflex spastic element.

In the moderate types of lymphedema, especially when the post thrombotic syndrome is an etiologic factor, sympathectomy may resolve the process. This operation should be selected as a treatment only if a sympathetic nerve block gives good laboratory and clinical evidence that some of the fluid collection results from a reflex spastic element. In some of the patients with venous involvement this can be combined with resection of the pathologic veins. In other patients in whom the lack of good early bandaging has left a residually enlarged leg, the surgical correction by wedge excision has been sufficient to give relief. In this operation a medial section of skin, subcutaneous tissue, lymph, fat and deep fascia is excised. The width of the skin segment removed is sufficient to reduce the part to normal size but a segment of subcutaneous and deep fascia wider than the skin segment is excised. The remaining superficial tissue is then mobilized and approximated with a plastic closure.

Surgical measures to develop lymph drainage to other nonblock areas have been tried for nearly as long as surgery has been performed. Originally Handley placed silk sutures subcutaneously in hopes of developing new lymph channels. Drainage tubes of all types, sizes and materials have been constructed. Tissue bridges with large pedicle grafts have been formed, testifying to the ingenuity of their originators if not to their surgical success. Techniques using glass needle tubes and various types of metal wires as well as tissue bridges are advocated by some surgeons. Our observed results have been poor.

A therapeutic technique based on the original observation of Kondoleon, that lymphedema is confined to the tissues external to the muscles has been employed. Kondoleon excised windows in the fascia, hoping that the lymph would flow internally and be absorbed by the muscle lymphatics. This procedure was extended by others and the technique consisted of undermining the skin and excising part, or all, of the subcu-



Figure 33. A, Photomicrograph of lymphedema showing avascular subepidermal layer and condensation; B vascular infiltration in the dermis (Pratt, G. H., Cardiovascular Surgery Lea & Febiger)

more lymph channels. The febrile reactions, while violent, will respond to the antibiotics.

Patients who have specific infections such as syphilis, filariasis or tuberculosis will have the symptoms of these diseases. The symptoms of lymph collections due to trauma, allergy and malignant disease are self-evident. The symptoms of lymphedema due to the post thrombotic syndrome include those of that syndrome. In addition to the enlargement of the part, there may be all the signs of thrombotic veins, depending upon the stage of the thrombosis and the recovery from it. Causalgia-like pain is not an uncommon symptom.

Pathology The epidermis of the skin per se, although thickened does not contain lymph stasis. The lymph accumulates in the dermis beginning at approximately $\frac{1}{16}$ inch in depth and shows great thickening. The papillae and sweat glands become obliterated. The edema is condensed and there may be avascularity due to pressure on the blood vessels from the fluid (Fig. 33). The lymph collections within the spaces become enlarged. The pressure of this lymph increases in the subcutaneous and fascial spaces. The lymph walls may be fragmented and gross lymph will be found in the extralymphatic spaces. Some of the structure of the subcutaneous tissues, fascia and collagens may be broken up. The fat is compressed and may become necrotic. Some of it is replaced by lymph spaces. The lymph channels are fibrosed, although some of them may be widely dilated. The muscles, tendons and even bone will show the effect of pressure and atrophy. The lymph may spread to the fascia and it often involves the fat below the fascia and within the muscle or around blood vessels. Lymph nodes are enlarged

and many of them become chronically inflamed and fibrosed.

In the stage of acute inflammation, the organisms may directly invade the lymphatics or pass through the cellular lining if there is no open lesion. The organisms multiply. The lymph fluid and the vessels react with an increase in the cellular elements and spread proximally. The edema is a hard, brawny type which indents or pits with difficulty. In the more advanced stage of the affection, suppuration may occur.

With elevation, the lymph flows gravitationally. With dependency it reaccumulates.

At a later stage, fibrous trabeculae form between the skin and deep layers. These form a skeleton-like framework which then maintains the space in which the lymph collects. The valves in the lymph channels are destroyed.

Treatment The aim of the treatment is twofold—elimination of the cause of the condition and relief of the residual lymph stasis.

Active therapy for the primary cause of the specific lymphedemas is indicated. In filarial infections, evacuation from the area of infection and avoidance of reinfection usually are all that are required. Lymphangiography should be performed prior to treatment in all cases of doubtful etiology. If the lymphedema is due to secondary infections, cultures should be made from material taken from the inflamed or infected area and the sensitivity of any organism to standard antibiotics should be determined. Antibiotic therapy should be thorough. When febrile attacks have been recurrent, the time pattern of these attacks should be determined and antibiotics given prophylactically prior to the anticipated time of the fever. Such therapy may have to be continued for

Grafts of skin, or skin and subcutaneous tissues, have been pedicled so that their base is proximal to the lymphatic block. The results have not been good. The technical difficulties attending use of these large grafts and the discomfort entailed in their production, and subsequently have made this type of operation obsolete.

Certain tissues of the body do not contain lymph. Among these is the epidermis. With the modern electric dermatomes one can remove skin so thin that only the epidermis is in the graft. All of the skin from the involved part is removed in long strips. Our best results have been obtained with skin 0.015 inch thick. Repeated biopsies show that the abnormal lymph collections begin approximately $\frac{1}{32}$ inch below the skin surface. After this thin skin excision is completed all the rest of the skin, the lymph, the fat and subcutaneous and deep fascia are removed. Great care is exercised to remove all of the fatty tissue and fascia from the underlying muscles. After hemostasis is secured, the skin previously removed is placed directly onto the muscle. The operation is performed in stages, the front half of the leg from the groin to the toes being operated upon first and the back half being operated upon when healing of the front graft is complete approximately in three weeks (Figs 34 and 35).

SECONDARY KELOIDS AND FUNGUS INTERIORES. Since the skin is poor, often diseased and subject to fungus infection disfiguring keloids or fungus collections may appear. In such cases these should be surgically excised

down to red muscle, grafted and the area treated with x ray beginning immediately after surgery to prevent recurrence.

Lymphedema of the Upper Extremity. Any of the etiologic factors detailed as a cause of lymphedema of the lower extremity may cause such an accumulation in the upper extremity. Most cases of lymphedema of the upper arm, however, follow operations for carcinoma of the breast (Fig. 36). One of the earliest suspected causes for lymphedema of the arm after an operation namely sepsis, often is a factor.

One of the last papers written by William Halsted was about the problem of lymphedema following radical mastectomy. He believed that the streptococcus was the cause. Most of his patients with this problem who were followed died of malignant disease and appraisal of the part that sepsis plays in the picture was neglected for some time. Swelling of the arm after mastectomy should be considered of possible malignant origin until proved otherwise. There remains, however, a sizable group of patients in whom lymphedema develops and annoyingly persists in the absence of carcinoma. It has been demonstrated not only that lymphedema follows infection but that the elimination of this infection by antibiotics controls and sometimes corrects lymphedema.

If it is determined that there is no malignant or infectious cause for the lymphedema surgical eradication of the lymphedema may be performed. The surgical treatment is similar to that for lymphedema of the lower extremity.

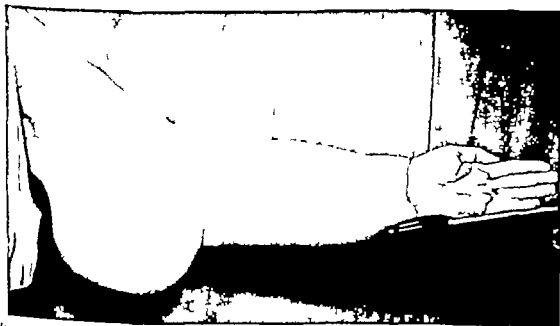


Figure 36 Lymphedema occurring seven years after radical mastectomy for carcinoma of the breast.



Figure 34. A, Massive lymphedema nine years after radical mastectomy and x-ray therapy. B, postoperative appearance. Patient had no recurrence of malignant disease thirteen years after original operation. Lymphedema has not recurred since the described operation was performed in 1954.

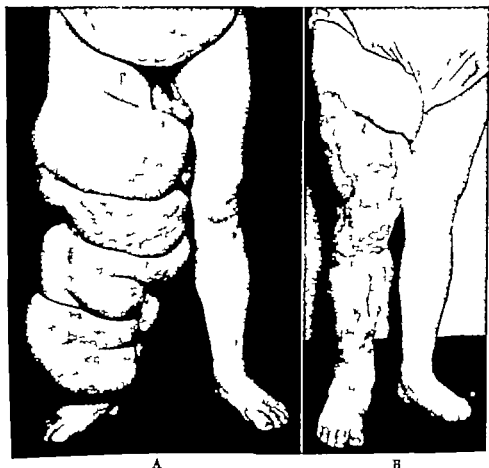


Figure 35 A, Massive lymphedema; B, postoperative appearance (Pratt, C. H. J.A.M.A., vol. 147)

taneous and deep fascia. The skin was replaced directly on the muscles. While reduction in size of the part resulted, lymphedema recurred in nearly all patients. The

value of the operation was temporary and consisted in plastic excisions of lymph swollen tissues. This operation and its modifications have limited value.

of the blood is not to replace red blood cells. The transfusions are given to replace some defensive factor lacking, at least temporarily, in the patient so afflicted. This defensive factor when supplied by blood is used up rapidly and must be replaced regularly approximately every two days. The regular repeated small blood transfusions have been decisive in overcoming infections of this type in innumerable cases. Abscesses should be drained wherever they occur. The original site if it is a blister, broken nail or callus, should be treated only if there is localized suppuration. Surgical intervention in the presence of localized pus is imperative, but it is important to remember that without such localization, the best treatment locally is surgical restraint. Such cutting in the absence of pus is bungling surgery and will be registered by increased morbidity and possibly mortality.

Chronic Lymphangitis. The usual septic organisms may cause chronic lymphangitis if their virulence is not too great or the defense of the host is high. Repeated attacks of acute lymphangitis may also result in chronic lymphangitis. In the absence of a septic cause for chronic lymphangitis the specific types of infection may be the cause of chronic lymphangitis. The various fungus infections such as epidermophytosis occasionally involve the lymphatics, usually in the form of the "tid." Most often the fungus infection is associated with a secondary invading organism. Sporotrichosis, a more rare mycotic disease caused by *Sporotrichum schenckii* may be the cause. Carcinoma may spread through the lymphatics and cause signs and symptoms not unlike those of an inflammation. The possibility of malignant disease should always be ruled out in any patient with nonspecific lymphangitis.

In nonspecific chronic lymphangitis, there may be recurrent signs of redness and swelling. The degree of lymph obstruction varies from a mild block to lymphedema. The glands are enlarged and their physical size often adds to the lymph obstruction. In many cases, thrombophlebitis is associated. The skin changes depend upon the degree of lymph obstruction and the venous involvement. Often there is pigskin appearance with enlarged pores. There may be secondary dermatitis or ulcer formation. A duskiness appears in the skin in the later stages of the disease. The edema is of the brawny type which pits with difficulty and persists longer than normal edema. In tuberculosis the lymphatic channels are cordlike nodules which later break down, forming abscesses and

may become secondarily infected. A similar type of nodular change occurs with syphilis but the lesions are less likely to suppurate. The epitrochlear glands may be involved by both of these lesions.

In chronic lymphangitis secondary to sepsis the initial site of infection may have to be excised. A small callus infected nail bed, foreign body or an area of osteomyelitis may be responsible for continuance of the lymphangitis infection until the source is eliminated. Antibiotic drugs should be used and if possible the specific one for the organism cultured should be selected. Tuberculous lymphangitis may require general therapy for the disease. In such cases, the newer drugs such as streptomycin with para-aminosalicylic acid and isoniazid should be utilized. X-ray therapy has had some beneficial results in some cases. Excision of the involved lymph nodes often is necessary. In each case the possibility of malignant disease should be considered and biopsies performed when doubt exists.

Lymphadenitis. The cause, course, symptoms and treatment of lymphadenitis in general are similar to those of lymphangitis, and the infections of the glands may be acute or chronic.

The glands enlarge and become tender when the invading organisms are septic in type. The degree of inflammation depends upon the virulence of the organism and the defense of the host. In tuberculosis, the glands enlarge rapidly, become firm and discrete nodules and then break down and caseate. The glands become fused together and suppuration and sinus formation take place. In syphilis, the nodes are universal but discrete. They are painless and do not tend to suppurate. In carcinoma, gland enlargement and secondary reaction depend upon the type of cell and extent of the spread as well as the therapy.

In the treatment of mild inflammation, correctly selected antibiotics may be all that is necessary. The organism should always be cultured and sensitivity to the routine antibiotics determined. Locally warm packs may help to resolve the process. If suppuration occurs surgical drainage is necessary. When doubt as to the cause of the enlarged gland exists biopsy or excision is necessary and should not be delayed.

The glands most often involved in tuberculous adenitis are the mediastinal nodes which drain the lungs. These nodes may be fibrotic and heal with surrounding calcium deposits. Most individuals have such calcified nodes in their lungs or in the medias-

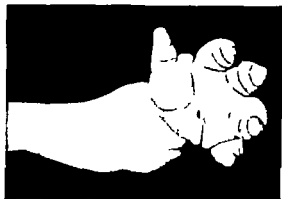


Figure 37 Massive lymphedema involving fingers, hand, forearm and part of the arm (Pratt, G. H.: Cardiovascular Surgery Lea & Febiger)

Congenital lymphedema of the upper extremity The cause for this condition is unknown. Some congenital rest, overactivity as in the Milroy's syndrome, or a glandular imbalance may be a factor. The lymphedema may involve the entire extremity or parts of it. Unfortunately it often involves the digits, causing not only an unsightly but a mechanical obstruction (Fig. 37)

The milder cases may respond to elevation and support. Some of the vasodilator drugs seem to be partially effective. Treatment of the patients with extensive lymphedema is unsatisfactory. The arm, forearm and hand can be treated effectively. The digits themselves pose a problem both from the general surgical and plastic standpoint. The viability of the graft must be obtained without sacrificing the mobility so necessary to tendon and joint motion. The fact that most of these patients are children adds to the difficulty since patient cooperation is required for the necessary active motion. Each patient's problem must be individually assessed and the treatment planned specifically for him.

Lymphedema Scrotum Lymphedema Labia. The cause for these lymphatic collections is unknown. The lymph stasis may be minimal in amount or it may develop into a tremendous and grotesque collection. The symptoms may vary from those in which the patient is aware of the enlargement only when tight fitting clothing is worn, to that typified by the textbook picture of the East Indian sitting on his scrotum. When lymphorrhea is a complication, the problem may become most severe. Some patients must wear towels or rubber devices to collect the lymph. Secondary sepsis and dermatitis are common. In the female, pregnancy complicates the symptoms markedly.

The treatment of these lymph collections depends upon the extent of the lymph stasis.

In the mild cases, antibiotic therapy hygienic care and elastic support to the genitalia may ameliorate the symptoms. In the extensive and advanced degree of lymphatic obstructions, surgical eradication may be necessary. In the patient with massive lymph labia partial or complete vulvectomy may be necessary and at times curative. In lymph scrotum, treatment must be individualized. The lymph area may be excised. In others, a new scrotal covering must be constructed by grafts and the skin and all subcutaneous and lymph-bearing tissues surgically excised.

LYMPHATIC INFECTIONS

Acute Lymphangitis. The most common causative organisms of acute lymphangitis are the staphylococcus and streptococcus. Usually the infection gains entrance through some skin break caused by an injury. The entry point may appear innocuous. A small paronychia or an ingrown nail, fungus infection, skin break or puncture wound often is the site of entry and source of infection.

The small lymph vessels or capillaries may be first involved. Usually there is redness, some heat and tenderness with edema. The condition spreads proximally involving first the lymph plexus and then the large lymph vessels. Once the main channels are involved, a red streak develops from the original site of infection to the local nodes. In the arm, these may be the epitrochlears. The infection then goes to the axillary nodes or it may go directly to the axilla. In the lower extremity the popliteal glands less often are involved. A red streak extending directly from the site of origin of the infection to the inguinal or axillary nodes is the most common. Often there are associated perilymphangitis and cellulitis. Once the glands are involved, there may be all degrees of sepsis from simple inflammation to suppuration and abscess formation. If the infection extends beyond the regional nodes, it invades the blood stream, producing bacteremia and septicopyemia. Abscesses then may occur in any part of the body.

An essential part of the treatment is rest and immobilization of the part. Local cultures and blood cultures should be obtained and the sensitivity of the causative organism to the antibiotics should be determined. The correct antibiotic should be given in massive doses. In the absence of a positive culture or until it is obtained, antibiotics can be given empirically. Massive wet dressings should be applied locally. Blood transfusions should augment the antibiotic therapy. The purpose

but there may be some discoloration due to venous pressure or venous involvement. Dilated blood vessels, especially the veins, often accompany these growths. They may occur at any place, but are often seen in the extremities. The head, face, neck, shoulders and chest are more unusual sites. They may develop a vesicular or keratotic appearance and in the latter instance they appear like warts. When this tumor occurs in combination with blood vessels it is called *lymphangioma cavernosum* (Fig. 35). Such a tumor is red to blue in color and is livid. Many of them are considered birthmarks. When they occur around the mouth they are called *macroGLOSSIA* and around the lips they are named *macroCHELIA*.

Gross these tumors vary markedly. The large ones are compressible. Histologically they are found to arise from epithelial cells. The *lymphangioma simplex* has vesicles, and these distend the epidermis. The proliferation of the epidermis over the vesicles forms keratosis. The vesicles contain coagulated lymph and the interstitial connective tissue is edematous. The *cavernosum* type shows much larger sinuses and in these the epithelial lining is well formed.

In the small growths electrocauterization has been effective. Carbon dioxide snow application will eradicate some of the superficial ones. The larger lesions should be excised surgically. These tumors do become malignant on occasion and this is a valid reason for their excision.

Lymphangioma Cysticum *Lymphangioma cysticum* appears to be caused by germ rests. In some of the tumors trauma or chronic inflammation is a contributing factor. They occur most often in the neck and sacrum. They have been seen frequently on the extremities and more rarely in the internal organs.

These cysts are large, not compressible and cause symptoms depending upon their site and size. They may cause distortion or pain by pressure. They are softer than xanthomas. Biopsies may be required for differentiation from other tumor masses.

The cysts are lined with endothelium. The fluid is serous or chylous. The surrounding sac is made of connective tissue and includes muscle fibers.

These tumors should be surgically excised. If they are removed without puncture a complete cure will be obtained.

Other Lymphangiomas. These small tumors also may be in the form of congenital lymphangectasia. They may be in the skin

(*nevus lymphaticus*) the lips (*macroCHELIA*) the tongue (*macroGLOSSIA*) the dorsum of the hand (*macroCHERIA*) or the neck (*cystic hygroma*) or they may be lymphatic cysts or hydrocele of the neck, axilla or floor of the mouth. In the neck the tumor is beneath the deep fascia and may protrude either anteriorly or posteriorly to the sternocleidomastoid muscle. The tumor may be of such size as to interfere with respiration or swallowing.

Surgical treatment is difficult and sometimes dangerous. If the cyst is not too large and does not interfere with swallowing or breathing, conservative therapy may be used. Sometimes the condition regresses spontaneously. X-ray therapy has been effective. Radium has been used. In some instances, a combination of radium and subsequent surgery has been effective with secondary plastic repair.

Lymphosarcoma. This is the most common of the malignant lymphomas. The tumor starts in the neck, the mediastinum or the axilla and grows rapidly with infiltration of the surrounding tissues. It also extends to and involves the skin. It may enlarge rapidly because of hemorrhage within it. The early signs may be those due to pressure on nerves or other vital structures. Secondary lymph stasis begins distal to the involved glands. Metastasis occurs by way of the blood stream particularly to the lung and the liver. The condition must be differentiated from other primary and secondary tumors of the glands, particularly from Hodgkin's disease. *Lymphosarcoma* grows and spreads more rapidly (Fig. 39) usually occurs on one side of the body and has ulceration whereas Hodgkin's disease rarely ulcerates and is slower in its extension.

Two distinct types of lymphosarcoma are recognized. These are a localized form and a generalized one called *lymphosarcomatosis*. In the localized form, the tumor may be composed of lymphoblasts (*lymphoblastoma*) which have large round cells. When the cells are small the tumor is called *lymphocytoma*. When the tumor cells originate from the reticular cells, the growth is called a *reticulum cell sarcoma*.

In the generalized form, the sarcoma is in many different parts of the lymphatic system and metastasizes early by way of the blood stream. The tumor may be in the blood vessels or in the blood stream itself (*leukosarcoma*).

Surgical excision usually is indicated only for biopsy or to relieve unusual pressure.

tinum At other times cold abscesses form with caseation. It is likely that from these latter abscesses develops miliary tuberculosis which is disseminated by the blood stream. Inflammation of these glands subsides when the primary disease abates.

Cervical tuberculous glands are visible and palpable and are important in differential diagnosis of cervical adenopathy. At the time of the biopsy surgical excision is indicated. Most of these glands require excision. Their chronic draining sinuses will lead to secondary suppuration.

Syphilitic adenitis requires specific therapy and operation is indicated for biopsy only.

Repetitive Septic Adenitis. Suppurative adenitis in itself may continue bouts of sepsis. These glands chronically subacutely and acutely reinfected become sources of the disease. Like repeated tonsillitis which results in frequent sore throat, these glands no longer are defensive. Their radical excision may help the patient. It must be recognized that these glands were placed by nature as a defense mechanism. When they have been reinvasion by organisms they are containers of but not defenders from, infections. While their excision removes a natural barrier this protection is no longer present and the patient often is better after radical surgery.

Lymphogranuloma Inguinale. This tropical disease also occurs in temperate climates. Adult male Negroes are most commonly affected. The etiologic factors are intracellular microorganisms called Donovan's bodies. They are passed from one person to another by contact.

Three to seven weeks after contact, the organisms cause a papule to develop on or near the genitalia. The involved area enlarges and breaks down and gives a thin white discharge which has a fetid odor. Other nodules may be secondarily affected. The regional lymph nodes are not enlarged in all cases but may be secondarily affected.

The diagnosis is made from the clinical appearance, the demonstration of the organisms, a typical pathologic section or reaction to the complement fixation test. There is no active surgical treatment, although surgery may be necessary for plastic repairs. Intravenous injections of tartar emetic have been effective and streptomycin may be of value.

TUMORS OF THE LYMPHATIC SYSTEM

Tumors of the lymphatic system are not too uncommon. Lymph divides the malignant



Figure 38. Lymphangioma cavernosum in a boy aged twelve years. There is massive involvement of the right lower extremity which includes both the lymphatics and the venous system.

ones into those having lymphocytic differentiation (lymphosarcomas, including lymphatic leukemia with lymph gland involvement) those with reticular cell characteristics those with mixed cell types, such as Hodgkins disease, and those with imperfect cell type differentiation. Benign lymphatic tumors may be primarily lymphatic or a mixture of lymphatic and blood vessel development.

Lymphangioma. Most lymphangiomas are of congenital origin. Some are noted in early life, but even these may have been present at birth but not noted. They are usually associated with other congenital anomalies. They have been seen in association with hemangiomas neurofibromas and undescended testicles.

This type of tumor may develop as an individual growth called *lymphangioma simplex*. It may be any size from that of a minute nodule to as large as a baseball. These tumors are usually the color of skin.

and excessive connective tissue. The spleen is enlarged and has gray areas within it resembling lymph nodes. It has been called the "hard baked spleen." Lymph nodes also are found in the liver and bone marrow. The blood will show secondary anemia and leukopenia with relative lymphocytosis. Leukocytosis may occur terminally.

The disease has been grouped into the localized the acute generalized the Pel-Ebstein syndrome characterized by alternate periods of fever and nonfever occurring at two-week intervals and the typhoid type because of its insidious onset remittent fe-

ver and enlargement of the retroperitoneal and mediastinal glands.

The main lesion from which Hodgkin's disease must be differentiated is lymphosarcoma. Lymphosarcoma occurs on one side of the body primarily grows more rapidly than does malignant lymphadenoma and tends to develop skin ulceration. Carcinoma normally has a primary source. Biopsy may be necessary to determine the correct diagnosis. Leukemia is differentiated by the blood count. In tuberculous lymphadenitis, the glands are irregularly nodular coalesce and caseate. In syphilis the gland enlargement is general



Figure 40 A, Hodgkin's disease invading the spleen. B, microscopic section showing giant cells and single-nucleus endothelial cells.

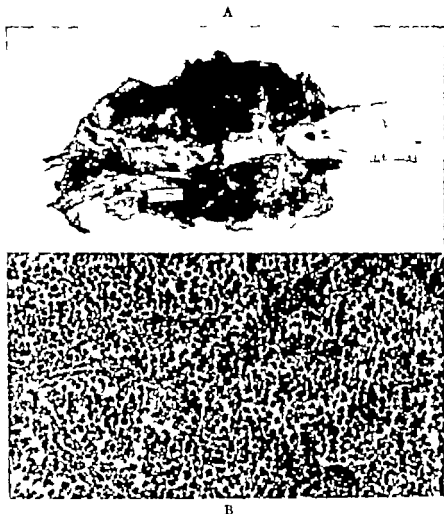


Figure 39 A, Lymphosarcoma arising from the periaortic lymph nodes. Note growth around and into the aorta; B, microscopic appearance of lymphosarcoma with massive infiltration of round cells (courtesy of Dr. A. Rottbo).

Radiation therapy has been used extensively and the results at first are good, but the tumor recurs. Regression can be maintained for some time with radiation therapy. Nitrogen mustard (mechlorethamine hydrochloride) and radiophosphorus have been effective in causing remissions. Mechlorethamine hydrochloride is thought to act by halting mitosis. This substance has been used in conjunction with radiation. If the lesion is localized, surgical excision may be tried and occasionally has been effective.

Hodgkin's Disease (Malignant Lymphadenoma) This disease is one of the malignant lymphomas and was described by Hodgkin in 1832. It was also called lymphadenoma without leukemia by Wunderlich, malignant lymphoma by Billroth and lymphogranuloma by Sternberg.

The cause of Hodgkin's disease is unknown. It attacks males more often than females and is seen in early adult life.

The lymph nodes rapidly enlarge without

pain. Often the enlargement begins in the glands of the neck and progresses in the direction of the lymph flow. The affected glands are discrete, variable in size and elastic. There is usually a high fever. The condition is progressive and fatal, but remissions have occurred.

Enlargement of the glands in the neck or thorax may cause coughing, shortness of breath and difficulty in swallowing. There may be inequality of the pupils and paralysis of the recurrent nerve. Edema of the arm or face with dilated veins occurs. Any type of neurologic symptoms may arise from intracranial or spinal cord pressure.

All of the lymphoid tissue of the body may become involved. The affected glands are round, not adherent and encapsulated. On cut section they appear gray and non-necrotic. Microscopic examination reveals giant cells with three or four nuclei (lymphadenoma cells) increased endothelial cells having single nuclei (Fig 40) an eosinophils

less fluid or are nearly empty so that their identification or injection made the problem of lymphangiography most complex. That the method has value in differentiation of the

causes and the treatment is obvious. Recently the technique has been standardized.

Lymphangiography of Leg and Thigh
The superficial lymphatics are medial to the



Figure 41 A, Lymphatics of lower extremity as shown by lymphogram. B Lymphatics in lymphedema. Notice obstruction due to valve failure (Gergely R.: Radiology vol. 71)

ized, but slight and positive reactions to serologic tests and a therapeutic test will differentiate this disease.

Patients with Hodgkins disease live from two to four years after the condition becomes manifest. Those having remissions may live from five to ten years.

Surgery is indicated for biopsy and occasionally for relief of pressure. The glands respond to radiotherapy but, unfortunately this is only temporarily effective. Methyl bis (β -chloroethyl) amine hydrochloride (mechlorethamine) has been used and there has been some fair response. This substance may be employed in conjunction with x ray therapy. The radioactive isotopes particularly phosphorus (P^{32}) are being used in some patients. Triethylenemelamine (TEM) is introduced in treatment in 1951. Rottino reported one patient with subsidence of massive nodes of the neck, axilla and mediastinum following this therapy. He also reported one patient with no recurrence nine years after excision of the nodes of the neck. Some patients have survived ten years after x ray therapy. It is apparent that no one type of treatment should be tried to the exclusion of all others. With supportive and other therapy the patient can be made more comfortable and months or years may be added to his life.

Supervoltage ray therapy. More recently tumors of the lymphatic system have been treated by supervoltage therapy. This can be given by x ray or cobalt. This latter substance can be given in large dosages and can reach the site of the lesion more readily than roentgen therapy. In addition the reaction of the patient to the treatment is much less. Both general and local response to supervoltage therapy as administered today is minimal.

Carcinoma. Since carcinoma is disseminated by the lymphatic stream it is a frequent cause for tumors of the lymph glands. The primary focus usually is apparent. In certain parts of the body such as the cervical nodes, the primary site may be difficult to demonstrate. Repeated biopsies of the tonsil, pharynx, thyroid gland or bronchus may be required when undifferentiated carcinoma is found in the cervical gland. The tendency of some carcinomas to skip certain local glands may add to the confusion.

Sarcoma. Sarcomas other than those specific for the lymphatic system, may involve the glands. Unlike carcinoma, however, most of these tumors spread by way of the blood stream. The rapid enlargement of a tumor

in a gland may make one suspicious of sarcoma, as these tumors tend to have internal hemorrhages and increase suddenly in size.

Endothelioma. Endotheliomas arise within the endothelial linings of the lymph sinuses and the lymph glands and may metastasize. These tumors are rare and are differentiated by biopsy particularly from secondary carcinoma.

Chloroma. Chloroma has been described as a sarcoma of the blood forming organs. It involves the lymph nodes and spleen. The greenish color indicated by its name is thought to be due to blood pigment or the products of fatty cell metabolism. The disease affects young males and is rapidly fatal.

Lymphatic Leukemia. This is a disease of the blood characterized by a marked increase in the number of circulating lymphocytes. The lymph glands as well as other lymphoid tissue throughout the body enlarge. Lymphatic leukemia may go through an aleukemic stage at which time diagnosis may be difficult. The relative lymphocytosis and the clinical course suggest the diagnosis even in this phase. Children are usually affected by the acute form, which is rapidly fatal. Their glands enlarge, but are soft and discrete. High fever and weakness as well as the blood picture are characteristic. The chronic form occurs in older persons. The glands are generally enlarged. The nodes are gray and homogeneous. On section the reticulum is seen to be packed with lymphocytes. Often the larger the lymphocytes the more rapidly growing is the tumor. There is no surgical treatment for leukemia. Biopsy is indicated. Nitrogen mustard administered intravenously urethan ACTH and cortisone have been tried. X-ray therapy will relieve some of the symptoms and P^{32} has given equivocal results.

LYMPHANGIOGRAPHY

Despite the fact that arteriography and venography have been in use for over twenty years, the application of similar techniques to the lymphatic system (Fig. 41) was difficult and was delayed until recent years. The early work was of an indirect type and began approximately in 1931 with efforts in this direction by dos Santos. Identification of both lymph vessels and nodes was made in dogs and rabbits. More recently Kinmonth has refined the technique sufficiently for it to be of clinical value and surgical use. To some extent, techniques used today are much like those described by Homans years ago.

Lymph vessels are so small, contain color

- and Elephantoid States Dependent upon Chronic Obstruction of the Lymphatic and Venous Channels. *Am. J. Trop. & Prev. Med.* 7:60 1913.
- Winn, W. F.: An Undescribed Variety of Hereditary Edema. *New York M. J.* 56:305 1813.
- Park, E. J., Barnes, S. H. and Ross, C. T.: New Operative Treatment for Elephantiasis. *Surg. Gynec. & Obst.* 81:812 1915.
- Patt, G. H.: Surgical Management of Lymphoedema. *J. A. S. A.* 147:1121 1951.
- Patt, G. H.: New Operative Technique for Surgical Correction of Lymphoedema. *J. A. M. A.* 151:555 1953.
- Patt, G. H.: Cardiovascular Surgery. Philadelphia: Lea & Febiger 1954.
- Patt, G. H. and Wright, I. S.: The Surgical Treatment of Chronic Lymphoedema (Elephantiasis). *Surg. Gynec. & Obst.* 71:211 1941.
- Robert, F. L.: The Regeneration of the Lymphatics. *Arch. Surg.* 13:871 1926.
- Robert, F. L.: The Recognition of Elephantiasis: the Problem of Experimental Lymphoedema. *Arch. Surg.* 20:513 1930.
- Rottino, A.: Hodgkins Disease. *New York J. Med.* 55:701 1955.
- Schobinger II., Cooper, P., and Rousselot, L. M.: Observations on the Systemic Venous Collateral Circulation in Portal Hypertension and Other Morbid States within the Thorax. *Ann. Surg.* 150:188, 1939.
- Servelle, M.: *Pathologie vasculaire médicale et chirurgicale*. Paris, Masson & Cie 1952.
- Sistrunk, W. E.: Lymphoedema Operation for Elephantiasis. *J. A. M. A.* 71:800 1918.
- Watson, W. L. and McCarthy, W. D.: Blood and Lymph Vessel Tumors; a Report of 1,056 Cases. *Surg. Gynec. & Obst.* 71:509 1910.

tibia and there is also a lateral group. A third group which follows the lesser saphenous is of less clinical significance. Since the superficial lymphatics are the ones involved most often in lymphedema, their delineation is most important. These lymphatics follow the greater and lesser saphenous veins to the superficial lymph nodes. Under normal circumstances they are thin (about 1 mm in size) wavy and parallel. Most of them are on the medial side of the leg and thigh.

Diffusible dye (patent blue, patent blue violet or patent blue V) is injected into the subcutaneous tissue and is picked up by the superficial lymphatics. This solution is made in an isotonic solution 11 per cent, in distilled water and is sterilized by autoclaving. This dye delineates the lymphatics. A total of 2 to 4 ml. is injected in the subcutaneous tissue between the toes (0.5 ml. in each toe web) and massaged with gauze for half a minute. It usually does not diffuse retrogradely. This dye is nontoxic but will run into the blood system and cause the patient to become blue.

In a bloodless field an incision is made in the foot proximal to the injection site. By means of pressure the lymph trunk is distended with dye. A radiopaque solution of 70 per cent iodopyracet or 50 per cent sodium diatrizoate is injected through a no. 18 needle. From 1 to 10 ml. is injected and radiographs of it are made as soon as possible. Nonscreen films of the extremities and screen films for the pelvis are suggested. Serial films give the best results and should be timed. Sites of blocks may be determined. Both benign and malignant causes of blocks may be indicated.

In lymphedema the lymph vessels become tortuous and dilated.

In lymphedema associated with iliofemoral venous thrombosis spasm of both the veins and lymphatics plays a part. In the acute stage permanent obstruction does not occur.

In chronic venous insufficiency thrombosis of the protein-rich lymphatics may cause a permanent edema state. Repeated bouts of febrile reactions of lymphangitis or cellulitis may cause fibrosis and scarring.

Recently a technique to demonstrate venograms has been done by direct bone marrow injection. It has been found that such venograms delineate lymph glands. This is of importance in malignancy and may indicate the site of metastasis better than any gross exploration. In addition, massive metastasis

may be demonstrated and indicate that any surgical procedure is contraindicated. Twenty cc. of 25 per cent iodopyracet or 76 per cent sodium methylglucamine diatrizoate is used.

READING REFERENCES

- Allen, E. V., Barker, N. W., and Hines, E. A., Jr.: *Peripheral Vascular Diseases*, 2nd ed. Philadelphia, W. B. Saunders Company 1933.
- Dos Santos, R., Lames, A. C., and Calkas, J. P.: *Arteriographie des membres et de l'aorte abdominale*. Paris, Masson & Cie, 1931.
- Foley, W. T.: *The Medical Management of Lymphedema*. *Mod. Concepts Cardiovas. Dis.* 24: 355, 1933.
- Gergely, R.: *The Roentgen Examination of Lymphatics in Man*. *Radiology* 71: 59, 1958.
- Gergely, R., and Zeebok, Z.: *De la Lymphangiographie*. *Progr. méd.* 64: 2300, 1956.
- Ghormley, R. L., and Overton, L. M.: *The Surgical Treatment of Severe Forms of Lymphedema (Elephantiasis) of the Extremities*. *Surg. Gynec. & Obst.* 61: 83, 1935.
- Halsted, W. S.: *Swelling of Arm after Operation for Cancer of the Breast—Elephantiasis Chirurgica*. *Bull. Johns Hopkins Hosp.* 32: 309, 1921.
- Handley, W. S.: *Lymphangioplasty: A New Method for the Relief of the Brawny Arm of Breast-Cancer and for Similar Conditions of Lymphatic Edema*. *Lancet* 1: 784, 1906.
- Holman, C., McSwain, B., and Beal, J. M.: *Swelling of the Upper Extremities following Mastectomy*. *Surgery* 15: 757, 1944.
- Homans, J.: *Phlegmasia Alba Dolens and Relation of Lymphatics to Thrombophlebitis*. *Am. Heart J.* 7: 415, 1932.
- Homans, J.: *Treatment of Elephantiasis of the Legs*. *New England J. Med.* 215: 1069, 1936.
- Homans, J.: *Circulatory Diseases of the Extremities*. New York, The Macmillan Company, 1939.
- Homans, J., Drinker, C. K., and Field, M. E.: *Elephantiasis and the Clinical Implications of Its Experimental Reproduction in Animals*. *Ann. Surg.* 100: 812, 1934.
- Hodack, S. S., and McMaster, P. D.: *Lymphatic Participation in Human Cutaneous Phenomena, Study of Minute Lymphatics of Living Skin*. *J. Exper. Med.* 57: 731, 1933.
- Kinmonth, J. B.: *Lymphangiography in Clinical Surgery and Particularly in Treatment of Lymphedema*. *Ann. Roy. Coll. Surgeons England* 15: 300, 1954.
- Kinmonth, J. B., and Taylor, G. W.: *Lymphatic Circulation in Lymphedema*. *Ann. Surg.* 139: 129, 1954.
- Kinmonth, J. B., Taylor, G. W., and Harper, R. A. K.: *Lymphangiography: Technique for Its Clinical Use in Lower Limb*. *Brit. M. J.* 1: 240, 1953.
- Kondoleon: *Die Lymphableitung, als heilmittel bei chronischen Oedemen nach Quetschung*. *Munchen-med. Wchnschr.* 59: 525, 1912.
- Lee, F. C.: *The Establishment of Collateral Circulation Following Ligation of the Thoracic Duct*. *Bull. Johns Hopkins Hosp.* 33: 21, 1932.
- Lamb, C.: *Tumors of Lymphoid Tissue*. Baltimore, Williams & Wilkins Company, 1934.
- Matas, R.: *The Surgical Treatment of Elephantiasis*

the closed cavity characteristics of the head.

Cranocerebral injuries may be caused by direct forces acting upon the head such as a moving object striking the head. Indirect forces may also act upon the head as when a moving body contacts a nonmoving or slower moving object. As a result of direct and indirect forces, the head may undergo acceleration, deceleration or compression (inbending of the skull). In many accidents, the sudden setting of the head into motion (acceleration) and compression coexists, also a sudden stoppage of the head (deceleration) and compression may coexist. Indirect forces include falls upon the lower portions of the body and the buttocks resulting in a downward thrust of the head toward the spine. Whip-lash injuries may cause a sudden lagging behind of the head in relation to the body which is projected forward as the neck is first hyperextended then flexed.

As a result of these various forces acting upon the head, various changes may occur—deformation of the skull, increase in intracranial pressure, mass movements of intracranial contents, distortion of the skull and dural septa, shearing of a portion of the head and contents without increase in intracranial pressure, shearing and severe increase in intracranial pressure such as with wounds by bullet or shell fragments, or a combination of two or more of these changes.

Deformation (compression) of the skull if sufficiently severe results in a fracture of the skull. A depressed or perforated fracture may occur if the injuring force is of high velocity whereas a linear fracture may be caused by a blow of lesser velocity.

A sudden increase in intracranial pressure occurs at the time of the impact. This may be due in part to the deformation of the skull and in part to the acceleration or deceleration of the head. If there is extensive tearing of the tissue, including scalp, skull and brain, the phenomenon of increased intracranial pressure may not involve the cranial contents since the closed cranial cavity state, under the circumstances of an open wound, may not then obtain. In experimental studies 15 to 100 pounds per square inch (450 to 5000 mm. Hg pressure) results in concussive effects. The time duration of the increase in intracranial pressure is directly proportional to the effects upon the brain. The records suggest positive and negative phases of pressure with elevations at the origin of the blow and negative pressures at an area opposite the region of the blow. In this connection it may be noted that the craniospinal junction

constitutes a region of pressure gradients between the cranial cavity and the spinal canal. Brain stem involvement is more possible if the closed cavity dynamics of the head obtains when the energy of a major blow is dissipated.

Mass movements of the intracranial contents also may occur. This has been shown by cinephotographic studies to take place with impact injuries. Mass movements result in tearing of tissues particularly when brain and irregularly placed cranial bony structures are apposed. This is true in the neighborhood of the frontotemporal junction and the anterior and middle fossae. Many contusions and lacerations of the brain surface occur in this region resulting from mass movements with abutment of the brain against the sharp edges of the lesser wing of the sphenoid and the irregular surfaces of the floors of the anterior fossae.

Distortion of the skull and the dural septa may occur in craniocerebral trauma with heavy forces acting upon the head with little or no velocity. This is particularly true in injuries to the newborn in the birth canal with forces being applied to the head by the contraction of the uterus and the resistance of the birth canal. Distortions may also occur in the adult as a result of low velocity heavy forces. Such distortions tear and bruise the brain, the dural sinuses and other intracranial blood vessels.

Shearing of a portion of the head and contents may occur without increase in the intracranial pressure at the time of impact, as illustrated by a puncture wound made by a sharp object.

As a result of these forces, fracture, concussion, contusion, laceration and hemorrhage may occur. Contusions and lacerations of the brain may occur as a result of mass movements of the brain against rough and sharp bony structures, such as the lesser wing of the sphenoid and the floors of the anterior fossae, inbending of the skull at the point of the blow with fracture and depression at this site, negative pressures in deceleration and acceleration injuries due to "cavitation," or the distortion of bony and dural prominences may tear the adjacent brain.

Small hemorrhages may occur in the path of the injuring force or larger hemorrhages may result from tears of larger vessels. There may be pressure gradients between the cranial cavity proper and the craniospinal junction, thus causing hemorrhages in the brain stem. Distortions may result in tears of connecting veins on the surface of the brain.

NERVOUS SYSTEM

Cranioerebral Injuries

By ELISHA S. GURDJIAN M.D.,
and JOHN E. WEBSTER M.D.

ELISHA STEPHENS GURDJIAN was born in Smyrna, Asia Minor now Turkey, and received his Doctor of Philosophy degree and medical education at the University of Michigan.

JOHN ELLIOTT WEBSTER, the son of a pharmacist received his education in Michigan where he was born. He is a product of the University of Michigan and Wayne State University College of Medicine.

The investigations of Doctors Gurdjian and Webster upon the forces of stress and strain produced in cranioerebral injuries have added considerable knowledge to an understanding of the many factors involved in these injuries.

In 1957 over 94,000 persons in the United States were killed as a result of accidents. Among these accidental deaths, head injuries played a prominent role. Thirty-eight thousand and five hundred deaths were the result of motor vehicle accidents. In and about the home 27,000 lives were lost as a result of injuries. Deaths caused by industry numbered 14,500 while public accidents other than those involving motor vehicles took about 16,500 lives. In individuals under the age of twenty-one years, accidents were an important cause of death. For the entire population, they were the fourth most common cause. Many accidents involving head injuries occur in and about the home—the result of falls down steps, off ladders and from

porches. Among the adult population, home and vehicular accidents are often associated with the consumption of alcoholic beverages, carelessness and fatigue.

Mechanism of Injury. For a good understanding of the mechanism of the head injury the importance of the structural characteristics of the skull and contents must be recognized. The skull is a spheroid structure containing semiliquid contents—brain, blood and cerebrospinal fluid. In injuries, the craniospinal junction presents an avenue for pressure gradients to occur between the head cavity and the spinal canal. The "closed cavity" feature of the head may be disturbed by certain types of trauma. In other instances, severe injuries may depend upon

the closed cavity characteristics of the head.

Cranio-cerebral injuries may be caused by direct forces acting upon the head such as a moving object striking the head. Indirect forces may also act upon the head as when a moving body contacts a nonmoving or slower moving object. As a result of direct and indirect forces the head may undergo acceleration, deceleration or compression (inbending of the skull). In many accidents, the sudden setting of the head into motion (acceleration) and compression coexists also a sudden stoppage of the head (deceleration) and compression may coexist. Indirect forces include falls upon the lower portions of the body and the buttocks resulting in a downward thrust of the head toward the spine. Whip-lash injuries may cause a sudden lagging behind of the head in relation to the body which is projected forward as the neck is first hyperextended then flexed.

As a result of these various forces acting upon the head various changes may occur—deformation of the skull, increase in intracranial pressure, mass movements of intracranial contents, distortion of the skull and dural septa, shearing of a portion of the head and contents, without increase in intracranial pressure, shearing and severe increased intracranial pressure, such as with wounds by bullet or shell fragments or a combination of two or more of these changes.

Deformation (compression) of the skull if sufficiently severe results in a fracture of the skull. A depressed or perforated fracture may occur if the injuring force is of high velocity whereas a linear fracture may be caused by a blow of lesser velocity.

A sudden increase in intracranial pressure occurs at the time of the impact. This may be due in part to the deformation of the skull and in part to the acceleration or deceleration of the head. If there is extensive tearing of the tissue, including scalp, skull and brain, the phenomenon of increased intracranial pressure may not involve the cranial contents, since the closed cranial cavity state, under the circumstances of an open wound, may not then obtain. In experimental studies 15 to 100 pounds per square inch (450 to 5000 mm. Hg pressure) results in concussive effects. The time duration of the increase in intracranial pressure is directly proportional to the effects upon the brain. The records suggest positive and negative phases of pressure with elevations at the origin of the blow and negative pressures at an area opposite the region of the blow. In this connection it may be noted that the craniospinal junction

constitutes a region of pressure gradients between the cranial cavity and the spinal canal. Brain stem involvement is more possible if the closed cavity dynamics of the head obtains when the energy of a major blow is dissipated.

Mass movements of the intracranial contents also may occur. This has been shown by cinephotographic studies to take place with impact injuries. Mass movements result in tearing of tissues particularly when brain and irregularly placed cranial bony structures are apposed. This is true in the neighborhood of the frontotemporal junction and the anterior and middle fossae. Many contusions and lacerations of the brain surface occur in this region resulting from mass movements with abutment of the brain against the sharp edges of the lesser wing of the sphenoid and the irregular surfaces of the floors of the anterior fossae.

Distortion of the skull and the dural septa may occur in cranio-cerebral trauma with heavy forces acting upon the head with little or no velocity. This is particularly true in injuries to the newborn in the birth canal with forces being applied to the head by the contraction of the uterus and the resistance of the birth canal. Distortions may also occur in the adult as a result of low velocity heavy forces. Such distortions tear and bruise the brain, the dural sinuses and other intracranial blood vessels.

Shearing of a portion of the head and contents may occur without increase in the intracranial pressure at the time of impact, as illustrated by a puncture wound made by a sharp object.

As a result of these forces, fracture, concussion, contusion, laceration and hemorrhage may occur. Contusions and lacerations of the brain may occur as a result of mass movements of the brain against rough and sharp bony structures such as the lesser wing of the sphenoid and the floors of the anterior fossae. Inbending of the skull at the point of the blow with fracture and depression at this site negative pressures in deceleration and acceleration injuries due to "cavitation," or the distortion of bony and dural prominences may tear the adjacent brain.

Small hemorrhages may occur in the path of the injuring force or larger hemorrhages may result from tears of larger vessels. There may be pressure gradients between the cranial cavity proper and the craniospinal junction, thus causing hemorrhages in the brain stem. Distortions may result in tears of connecting veins on the surface of the brain.



Figure 1 Contusions of the temporal and frontal tips in a patient with an occipital fracture.



Figure 2 Deep contusion of the left fronto-temporal region with petechial hemorrhages surrounding the area of contusion. If the patient survives for several days, such an area will soften and become infarcted and may constitute an intracerebral hematoma with severe tissue loss.

With fractures of the skull there may be tears of the dural lining with hemorrhages due to laceration of the meningeal vessels. Subdural hemorrhages and intracerebral hemorrhages may take place by the mechanical disruption of veins. Laceration of the arachnoid and of the brain may occur. The effect of sudden compression of the chest upon intracranial structures is not well understood. Indirect involvement of the brain by such forces may be produced when the chest is suddenly decelerated by a steering wheel.

Pathology The terms *concussion*, *contusion*, and *laceration* of the brain should not be used in a manner to suggest prognostic evaluation and the degree of severity of

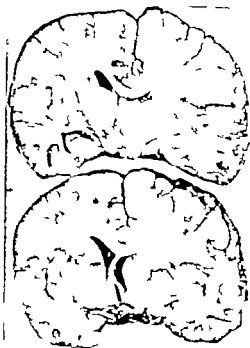


Figure 3 In the cross section of the brain above, compression of the upper brain stem by a left-sided uncus herniation is noted. Below marked swelling of the centrum ovale on the left side is seen. Such swelling of the white matter extends all the way into the brain stem.

injury Concussion is defined as a post traumatic state due to an involvement of the upper brain stem centers, resulting in unconsciousness with low pulse rate and a cold clammy skin associated with a shocklike state. Contusions (Figs 1 and 2) and lacerations of the brain may accompany concussion, but not necessarily. Lacerations and contusions may occur without associated involvement of brain stem centers causing a concussion.

Increased intracranial pressure and edema of the brain in acute head injury are important pathologic entities. Increased intracranial pressure may result from edema and swelling of the brain (Fig. 3) hemorrhage into the intracranial contents and increased intracranial blood volume due to trauma. It is agreed that hemorrhage into the intracranial contents causes increased intracranial pressure. This is due to an actual increase of the volume of intracranial contents caused by the escape of blood, particularly into the subarachnoid space. Blood in the subdural and subarachnoid spaces on the brain surface may result in a high osmotic pressure and consequently may attract a collection of tissue fluids and cerebrospinal fluid in the area of the bleeding. In the subarachnoid space, actual increase in the amount of fluid may occur because of the osmotic

pressure of the cerebrospinal fluid. Traumatic contusions of the brain, bloody extravasations, small thrombi and areas of necrosis may cause edema. Collections of fluid and swelling in these areas of tears and bruises are similar to edema about a traumatic lesion elsewhere in the body.

The presence of edema without an appreciable amount of bleeding into the tissues is difficult to explain. Many years ago, vasomotor paralysis of cerebral vessels by the trauma, with subsequent venous stasis and tissue waterlogging due to increased permeability of the capillaries, was thought to be a cause for the brain swelling. Recently the same opinion has been restated by neuropathologists.

Other explanations of increased intracranial pressure are increased activity of the choroid plexus due to irritation and stimulation by the injury, resulting in increased cerebrospinal fluid formation; a lowering of the rate of absorption of the cerebrospinal fluid due to venous stasis with venous pressure elevated well above that of the cerebrospinal fluid. Increased protein content in the cerebrospinal fluid due to blood retards its absorption and, since bloody cerebrospinal fluid is quite common in head injury, increased intracranial pressure may then be due in part to a deficient absorption of the cerebrospinal fluid.

That edema and swelling of the brain occur is unquestioned. That this can take place in the absence of large mass lesions in the cranial cavity is also well substantiated. Figure 4 shows a contusion of the left temporo-

parietal region of the brain associated with marked edema on the affected side with actual herniation of the cingulate gyrus across the midline. Swelling and edema of the brain may also result in tentorial herniation of the uncus and brain stem as well as herniation of the medulla and cerebellar tonsils into the foramen magnum. Swelling of the white matter under a mass lesion may extend into the brain stem.

Clinical Manifestations. The clinical manifestations of acute head injuries depend mainly upon the degree of involvement of the brain. The gravity is indicated, in part, by the length of unconsciousness following trauma. It should be pointed out, however, that a certain number of apparently serious open wounds of the cranial cavity may be unassociated with initial unconsciousness or may be associated with a very short period of unconsciousness. On the other hand, many closed injuries of the head may be associated with severe derangement of the conscious state for many days.

The older classifications of patients with head injury have emphasized the terms concussion, contusion and laceration, the first named representing a usually mild injury, the last two indicating injury of a more severe grade. Recent evidence indicates that some patients with brain laceration may not have an initial unconscious state, whereas others with no laceration or contusions may have a fatal outcome with only a so-called concussive effect and little evidence of microscopic damage to the nervous system. Consequently, a present-day classification of head injury recognizes the fact that patients who have initial unconsciousness following the trauma have brain stem involvement. However, patients without initial unconsciousness may have extensive brain damage without involvement of the brain stem. Patients with brain stem syndromes present post-traumatic unconsciousness, their pulse is slow and the pallor of their skin suggests a shocklike state. The brain stem dysfunction may be of varying intensity involving both reversible and irreversible states. The reversible state may result in complete recovery. Head injuries without a brain stem syndrome are common. Open wounds of the head without initial unconsciousness are examples. The utilization of energy resulting in the open wound leaves little or no energy to involve the brain stem centers.

Another group consists of patients having a combination of a brain stem syndrome with a severe wound of the nervous system. It



Figure 4. Left temporo-frontal contusion and swelling of the left hemisphere. Note cingulate gyrus herniation from left to right over the corpus callosum. Note also hemorrhages in the brain stem due to internal herniation. Note the ventricular shift from left to right in the absence of a mass lesion.

Table 1 Summary of Pathologic Conditions in Acute Cranioerebral Trauma

Scalp

Contusion
Laceration
Hematoma

Skull

Fracture—open—closed
Linear
Comminuted
Depressed
Expressed (pushed out)
Penetrating

Intracranial contents

Brain
Concussion
Contusion
Laceration
Edema and increased intracranial pressure
Subdural accumulation of cerebrospinal fluid
Hemorrhage
Epidural
Subdural
Acute
Subacute
Chronic
Subarachnoid—localized, generalized
Intraparenchymatous
Peteelial
Massive

Blood or cerebrospinal fluid discharge or discharge of brain tissue from

Ear
Nose

Infection

Osteomyelitis
Meningitis
Cerebritis and cortical ulceration
Brain abscess
Epidural
Subdural
Intraparenchymatous

Complications

Pneumocephalus
Spurious meningocele

Associated systemic disorders

Fractures
Epine injuries
Chest injuries
Abdominal injuries

should also be pointed out that in the course of time a patient who may begin with no evidences of brain stem dysfunction may through edema of the brain or intracranial hemorrhage develop evidences of involvement in this region. These secondary manifestations of brain stem derangement are, of course, quite common in head injury. If we are to use the term "concussion," it should be defined as post traumatic unconsciousness following impact, due to functional or anatomic derangement of the upper brain stem. Concussion may occur with or without brain contusion or laceration, or with or without a skull fracture and vice versa.

The clinical manifestations of head injury may be grouped as states of consciousness and their alterations, changes in the vital functions, headaches, dizziness, vomiting, the neurologic status of the patient, convulsions, the cerebrospinal fluid findings and the associated injuries elsewhere in the body.

States of consciousness and their alterations A normal state of consciousness is a favorable sign in the patient with an acute head injury. This state is a contraindication for any operative intervention except in patients having defects of the skull such as open wounds. Progressive return of consciousness is also a favorable sign and is almost always a contraindication for surgical treatment. On the other hand, progressive drowsiness and periods of consciousness alternating with periods of drowsiness and disorientation may signify that a dynamic lesion exists necessitating surgical intervention. A lucid or conscious interval followed by drowsiness and semiconsciousness or unconsciousness is an important syndrome, often, but not always, signifying an intracranial mass lesion. At times, localized areas of bruising and contusions of the brain with swelling of the tissue particularly over the convexity of the hemisphere, may be associated with this syndrome. Sometimes following simple lumbar puncture the patient's disturbed conscious state may return to normal. Middle meningeal hemorrhage subdural hemorrhage subdural accumulation of cerebrospinal fluid, intracerebral hematomas and edema of the brain may be associated with a lucid interval. In a certain number of head injury patients, extreme disorientation alternating with periods of semiconsciousness and psychotic manifestations are seen. In this group an injury in the temporal tip may be present, resulting in this agitated state. Area 44, which is at the junction of the frontal and temporal lobes, may be injured in severe cranioerebral trauma, the result of mass movements of the brain. Unilateral or bilateral contusions of this region may occur. Acute psychotic states usually tend to resolve, but personality derangements may exist for a long time.

In general, patients who remain unconscious for many days return to consciousness through a transitional state of semiconsciousness with disorientation and confusion. Occasionally the return to normalcy is quite prompt, but more often it is prolonged. Neurotic and psychotic manifestations associated with head injury are difficult to evaluate in respect to cause-and-effect relationship. Psy-

choneurotic manifestations seen several months or years after a head injury from which the patient apparently has made a satisfactory physical recovery are probably not related to the head injury per se but to the experience of stress upon an abnormal pretraumatic personality. A proper evaluation should include a careful consideration of the patient's psychiatric background, his ability to tolerate stress and his reactions to environmental experiences. In some patients mental deterioration following a severe head injury with no improvement in the intellect can undoubtedly be ascribed to the total devastating effect of the trauma upon the central nervous system. Among the patients with this class of injury adolescents and children rehabilitate better than do adults. The latter may remain in a vegetative state for weeks or months following a severe head injury. In these patients, the condition appears to be related to cortical brain damage with or without a major degree of brain stem injury.

Amnesia following head injury is quite common. Usually the patient remembers neither the accident nor activities immediately preceding the accident. Sometimes the amnesic state may be present in patients who appear normal. Such patients may have a short period of post-traumatic unconsciousness or may be merely dazed for several seconds. Nearly 80 per cent have complete amnesia for the experience of the injury itself and the period just before the impact. According to some observers, prolonged post-traumatic amnesia increases the likelihood of post-traumatic epilepsy but at the same time suggests that the interval between trauma and the development of epilepsy is going to be longer.

Vital functions in head injury Vital functions are judged by the heart or pulse rate, the respirations, the body temperature and the blood pressure. In general, these may be altered from the normal at the time of injury and as the results of the injury become evident.

In experimental head injury the pulse rate may be increased at impact. Vagal effect (bradycardia) with escape is frequently recorded in a kymographic study of the pulse or the blood pressure. Experimentally the initial result of a severe head injury is an increase in the pulse rate.

In the seriously injured patient, the pulse may be full, slow and bounding initially. As improvement takes place, the pulse may become a faster, less full and bound-

ing. If the patient fails to improve it may first become slower. A pulse rate of 40 to 55 per minute is quite common in the patient with an acute severe head injury. Eventually in the fatally injured patient the pulse may suddenly increase in rhythm with lowering of the amplitude. It may become elevated to 120 to 180 or higher eventually becoming thready in the patient who is failing. These changes occur in the course of ten to twenty-four hours or longer following the injury.

It is interesting to note that occasionally a low pulse rate may be noted without an associated abnormal conscious state. A pulse rate of 40 to 50 unassociated with abnormalities of the heart is seen in some patients following head injury who are conscious and are feeling quite well. A cause for such a low rate may be a period of vagal stimulation with resultant bradycardia. Such bradycardia usually disappears in the course of two or three days. In other patients the pulse may be within the range of normal to start with but over a period of several days may show a lowering in rate. Such a decrease should be carefully evaluated in conjunction with other signs of increase in intracranial pressure due to a dynamic lesion in the cranial cavity. In patients having fatal lesions, such a pulse will eventually increase in rate and become thready. A fast, thready pulse may be seen from the beginning and may be associated with a shocklike state, particularly if injuries to other parts of the body are also present.

The respirations observed in animals with experimental head injury may be somewhat increased in rate when the blows are of subconcussive force but with a moderate or severe head injury there is cessation of respirations for varying periods. In moderate injuries following the return of the respiratory rhythm, the animal's respirations continue at a somewhat slower rate without much subsequent change. In more severe injuries respirations may never return or if they do they may be gasping in type eventually becoming arrested. The animal may have apneic periods ranging from seconds to a minute.

In the human being, depending upon the degree of the injury respirations may be normal or variously altered. In a lethal injury the patient may take gasping breaths. In the less seriously injured individual, there may be deep stertorous breathing which may become accelerated. The hyperpnea may be associated with atony of the bronchiolar

and bronchial epithelium with collections of excretions in the upper respiratory passages resulting in typical noisy hyperpnea (pulmonary edema). In other patients, the respirations may be slow and labored with evidence of difficulty in obtaining a satisfactory exchange. In some following a normal respiratory rhythm there may be a lowering of the rate. Under these circumstances, the presence of a dynamic lesion may be considered likely. Rapid respirations associated with a high temperature and unconsciousness hold a poor prognosis. Rapid respirations in a conscious patient usually result from other than intracranial abnormalities; a chest lesion may be present.

Hyperpnea in patients with severe head injury is common. Its association with obstructive collections of tracheobronchial secretions is also common. The reason for this collection may be a poor exchange of air in the establishment of the vicious cycle with pulmonary edema, improper activity of the ciliated epithelium of the bronchial tree due to the severity of the injury or improper activity of the muscular layers of the smaller bronchioles resulting in poorer excretion. These abnormalities of the muscular activity and the ciliated epithelium may result from a central mechanism with paralysis of parasympathetic and sympathetic impulses. Cheyne-Stokes respirations are also seen occasionally in the head injury patient, although the slow gasping respirations are much more common. Cheyne-Stokes respirations also indicate a poor prognosis. In the experimental animal such hyperpneic states with pulmonary edema do not occur following severe head trauma.

Respiratory embarrassment with poor exchange may be improved by careful toilet of the upper respiratory passages and employment of a tracheotomy. The latter may be lifesaving in many patients by preventing obstruction. Less effort is required to ventilate the unobstructed lungs.

The temperature of the patient with an acute head injury may be subnormal or normal soon after trauma. It may be higher several hours or days after the accident. Particularly if there is associated injury elsewhere, early subnormal temperature, as a part of a shock state may be noted. Associated with such a low temperature there may be a peculiar yellowish gray discoloration of the skin, the extremities being quite cold. In many patients with severe head injury the blood pressure may be at a normal level.

In this respect the shock state in the cranio-cerebral trauma patient is different from shock in the patient having skeletal and bodily injury.

In the patient with severe contusions of the brain and with bloody spinal fluid, a slightly elevated temperature with a warm dry skin may continue for several hours and then hyperthermia may develop with a temperature of 104 to 107 F., associated with hyperpnea, pulmonary edema and a rapid pulse rate. If such a state is not attended with unconsciousness, other causes for the hyperpnea and the elevated temperature should be found. In patients with moderately severe head injury the temperature is elevated to 101 to 103 F. and it tends to lower as the patient shows improvement. Dehydration, absorption and repair processes and infection may cause some increase in temperature. A high level of temperature sustained for many days or weeks is unusual. Even though the cranio-cerebral trauma may have initiated some of the upward trend of the temperature, other causative factors such as pulmonary, genitourinary or meningeal infections should be sought. In the course of convalescence, a sudden rise of temperature should be carefully evaluated, particularly in patients upon whom an operative procedure has been performed. It may signify a complicating infection requiring re-exploration of the wound.

In the experimental animal the blood pressure usually increases following a concussive blow. In subconcussive blows, there may be a drop in pressure resulting from vagoglossopharyngeal stimulation. In the severely injured animal, the blood pressure after rising 100 mm. Hg or more begins to fall within seconds. As the initial pressure is then reached, the fall is followed by a secondary rise which is not as high as the initial rise. Still later the vasomotor tone is completely lost and the blood pressure falls to zero.

Obviously such changes in the human being are not seen in the hospital but may occur at the time of the injury. In the emergency room, the blood pressure readings of most patients with head injury are near normal. The blood pressure is well sustained even though the patient may present the appearance of a shocked state. It has been shown experimentally that in the seriously injured animal there is a central vascular dilatation with peripheral vasoconstriction. In the human being, essentially the same

changes probably take place with peripheral vasoconstriction resulting in cold limbs and a pale yellowish gray sweating face.

Occasionally the blood pressure may rise in association with increasing intracranial pressure as produced by a massive intracerebral hematoma. A slowing of the pulse and respirations may accompany such an increase in the blood pressure. In some patients there may be an increase in blood pressure and a high level of systolic and diastolic pressures may be sustained for several days but become normal as the patient improves. Most often the blood pressure readings are not of much help in assessing the level of the increase in intracranial pressure. Except for the fact that the pressure level records the general functional competency of the vascular system blood pressure readings are of minor help.

Headache. Headache is a common symptom in patients with head trauma. As the seriously injured patient improves from the initial disturbed conscious state he may complain of headaches. In other instances headache may be the result of an increase in intracranial pressure in patients without traumatic unconsciousness. In still others it is seen following minor head injuries unassociated with unconsciousness at the time of impact. These patients nevertheless, complain bitterly of headaches which in the main are caused neither by intracranial bleeding nor increased intracranial pressure.

The headaches are usually generalized, but they may be localized to one or the other side of the head and are frequently located in the vicinity of scalp lacerations and bruises. Some patients may have headaches only when in the upright position noted upon getting up and about from bed. These headaches disappear when the patient is in the recumbent or semi-Fowler's position. It has been postulated that such headaches may be due to generalized hypotension with traction upon the intracranial blood vessels.

The mechanism of headache in the patient with a head injury may be related to several factors. Recent information suggests that headache is usually due to changes in the blood vessel tone and caliber of intracranial and extracranial vessels. The vessels at the base of the brain and those of extracerebral structures may become dilated or constricted. Pain resulting from dural vessel involvement also is possible. Abnormalities of tone in the blood vessel walls may result from both mental and hypothalamic disturbances.

Such a central involvement may influence blood vessels not only of the head and scalp but also of other parts of the body such as the gastric or intestinal mucosa. The latter may be associated with ulceration erosion of vessels and gastrointestinal hemorrhage. In the gastric mucosa such an influence is probably through vasospasm and ischemia followed by erosion of the lining.

Traction upon the larger vessels at the base of the brain may develop from abnormal cerebrospinal fluid hydrodynamics related to the presence of blood in the subarachnoid space. In patients who obviously have no cerebrospinal fluid hypertension and yet complain of headaches, traction on the larger blood vessels of the circle of Willis may be a factor particularly with change of the position of the patient's head. An important cause for headaches in the post-traumatic patient involves an associated injury to neck structures, including muscles, ligaments and the cervical nerves. Since many deceleration and acceleration injuries result in traction of the head away from the neck and compression of the head toward it and the trunk, damage to the second and third cervical vertebrae with associated involvement of muscles and nerves may result in head and neck pain. Particularly in injuries of the second cervical area, painful conditions in the back of the head and neck on one or both sides may result and eventually generalized headaches may develop. Hunter and Mayfield have shown that there is extensive distribution of pain in the scalp from the cervical plexuses with involvement of nerves in the upper cervical region. Neural and muscular involvement at the cranio-cervical junction may cause abnormalities of head positioning with initiation of a vicious cycle which eventuates in diffuse headache.

When in the less seriously injured individual there is history of migraine or periodic headaches prior to the head injury the patient may complain that the headaches following the injury are aggravated. As to whether or not these complaints can be ascribed to a neurosis or a justifiable syndrome is a question. In a few instances patients have complained that migraine states which have been quiescent for three or four years have been reactivated following a head injury. It is difficult to evaluate such a relationship. The head injury in such patients probably is a precipitating factor. Other stresses involving psychic injury such as a divorce or financial loss, may have the same effect.

Headaches observed in the post-traumatic syndrome patients are noted usually to improve in a period of three weeks to three or four months. Occasionally a patient continues to complain, but often this is found to involve compensation factors. These patients continue to benefit financially as long as their complaints are validated. Dizziness usually disappears before the attacks of headache subside. Occasionally headaches are absent from the beginning while dizzy attacks predominate. In some, the attacks of dizziness are completely absent. In general, complaints of intermittent headache and dizziness favor an organic cause, while persistent headache or dizziness which never disappears completely suggests the presence of a neurosis or malingering.

It is interesting to note that often seriously injured individuals have no post-traumatic headaches. Others with a very mild head injury have severe complaints. About 80 per cent of patients with all types of head injury complain of headaches. In boxers, persistent post-traumatic headaches and vertigo following knockdowns and knockouts are quite infrequent.

Dizziness Dizziness is a common complaint during the convalescent period. When the extremely ill individual recovers, attacks of dizziness are not quite as common as in patients with less severe craniocerebral trauma. Many patients with a minor head injury with or without an initial period of unconsciousness at the time of impact may have residual dizziness. Two types are noted: first, the vertiginous type, or an actual feeling of rotation or whirling of the body or surrounding objects, is experienced and, second, giddiness with a feeling of unsteadiness and lightheadedness which is unassociated with feelings of whirling or vertigo. A change in position frequently initiates the dizziness. In the vertiginous type, certain positions of the head and the direction of gaze may result in symptoms. The patient becomes giddy, may feel nauseated and may vomit. Patients learn to refrain from the undesirable posturing of their body and their eyes.

Changing the body position, such as from a supine to a standing position, or vice versa, or bending forward, may result in a feeling of losing balance and meeting the floor. Many patients learn to move more deliberately in order to prevent the feeling of giddiness. If the patient is not careful, he may be felled by the attack. Patients frequently identify their spells by the term "blackout,"

but on questioning admit that unconsciousness does not accompany their spells. The giddiness or vertiginous attacks tend to disappear over a period of several weeks or a few months. The attacks first become less frequent and the patients feel quite well between attacks.

The cause for dizziness is not completely understood. Vertiginous attacks are due to an involvement of the vestibular nerves, the labyrinth peripherally or their central connections in the brain stem. Occasionally true vertigo will occur as a temporal lobe irritative phenomenon. Although giddy attacks may be due to an involvement of the cerebrum, no definite area has been related to their production. The disturbance in the peripheral balancing mechanism seems a more likely cause.

The combination of headaches and dizziness as post-traumatic sequelae, particularly in minor head injuries, is known to both the physician and the layman. The protraction of such complaints may be due in part to functional factors, but that they have an organic basis in the early cases is unquestioned. In part, these complaints are on an organic level, but psychoneurotic influences alter, lengthen or reinforce these sequelae.

The study of the labyrinthian mechanism in patients with head injury has not been fruitful. Those who complain of giddiness have shown no unusual degree of abnormality of the labyrinthian structures, excepting possibly for some decrease in the reaction to stimuli. In patients who have severe vertigo, caloric tests have shown some impairment of the labyrinth function in association with nerve deafness. Many of the latter group also complain of tinnitus, usually on the one side. A ringing in the ear may be associated with a history of bleeding from one or both ears.

Vomiting Vomiting is frequently associated with craniocerebral trauma. In a large series of patients having head injuries, it occurred in about 20 to 30 per cent of the patients. In the patient with a minor head injury nausea and vomiting may be the result of a reflex stimulation of the vagoglossopharyngeal system. In some individuals it may be due to the activation of this reflex through the presence of swallowed blood in the stomach. In the more seriously injured patient, nausea and vomiting may develop with recovery from an unconscious state. Sometimes vomiting and nausea are associated with changing of the position of the patient. When this occurs vestibular dam-

age with nuclear and peripheral involvement of the vestibular system may be the cause. Usually vomiting is a frequent manifestation in the less seriously injured individual. In the profoundly injured patient it does not occur as frequently. The vomitus is usually bloody or coffee-ground color either from the presence of swallowed blood from trauma to the oral cavity or from gastric bleeding from an abdominal injury. In some instances vomiting may be the result of hypothalamic abnormalities producing ulceration and hemorrhage of the gastric mucosa. Because of the possibility of aspiration of the vomitus, the management of patients with head injury may require the washing out of the stomach soon after admission to the hospital. Vomiting, along with dehydration and fluid imbalance may result in chemical changes including hyponatremia and hypokalemia which delay or prevent recovery.

Neurologic manifestations in cranio-cerebral injuries. In acute cranio-cerebral injuries neurologic manifestations may be disguised by abnormalities in the state of consciousness. Thus, an accurate evaluation of the patient's ability to cooperate normally, his visual acuity, difficulties of hearing, abnormalities of the sense of smell, speech disturbances and sensory abnormalities may be impossible. However important conclusions may be drawn from the neurologic examination. Repeated examinations are valuable and often mandatory. Minor changes in the neurologic status of the patient may signify abnormalities not suspected previously. In the presence of extensive intracranial damage, certain abnormalities in the sensory sphere also become evident as the patient recovers consciousness. Thus, visual defects, abnormalities in the power of thinking, sensory phenomena, and auditory, olfactory and speech disturbances may become evident.

In the early stages following an acute severe head injury the examiner is often limited in his evaluation by the lack of cooperation of the patient. Under these circumstances, an examination of the pupils and fundi, search for bleeding from body orifices and an evaluation of gross abnormalities of motor function may be of value. Any inequality of the pupils is noted. In over 90 per cent of the patients having inequality of the pupils, the dilated pupil is on the side of the intracranial lesion. A dilated pupil may indicate partial or complete involvement of the oculomotor nerve. There may be divergent-strabismus, or conjugate deviation

of the eyes to one or the other side. Usually in patients with cortical lesions the eyes are turned toward the lesion. In those with nuclear lesions the eyes are turned away from the lesion. Examination of the fundi may show the presence of hemorrhages in the aqueous or vitreous humor or in the retinas. Such hemorrhages when present soon after injury may indicate local forces acting directly on or about the globe. In other instances there may be evidence of venous engorgement, diffuse retinal edema, blurring of the disks or choked disks.

Nuchal rigidity may be present particularly in those patients who are resistant and uncooperative. Its absence does not preclude the presence of subarachnoid hemorrhage with bloody spinal fluid. There may be varying degrees of associated decerebrate rigidity. In some patients who are profoundly ill there may be limpness or flaccidity with a generalized areflexia, which is an ominous sign.

Weakness or paralysis of an extremity, facial weakness and the presence of certain abnormal reflexes may be identified in the unconscious or semiconscious patient. It may be evident that the patient moves one upper extremity more frequently than the other. On stimulation one may notice that one entire half of the body is paralyzed as compared with the other half. There may be facial distortion suggesting a weakness on one side of the face. In this connection the presence of a unilateral corneal reflex loss may be significant. Unilateral corneal reflex loss is probably due to the presence of central or peripheral facial weakness and consequently may be significant as a localizing finding. The presence of unilateral or bilateral pyramidal tract signs with confirmatory reflexes should be looked for and recorded.

Speech disturbances may occur in some patients with severe brain damage but with little or no evidence of brain stem involvement. Open depressed skull fractures in the left frontoparietal area may be associated with motor aphasia and, in some instances, with jargon speech. An inability to understand spoken language may be present. Although large amounts of brain may have to be sacrificed during débridement, a relatively good return of function eventually occurs and usually traumatic aphasia clears up. As a patient becomes more cooperative, evidences of sensory aphasia, alexia, agaphia and apraxia may be noted.

Visual disturbances, particularly involvement of the visual fields, may be noted in

the convalescent patient, although suspected by the extent and location of damage sustained at the time of the accident. Homonymous hemianopsia with occipital lobe involvement is occasionally noted. In temporal lobe lesions due to open depressed skull fractures there may be quadrant defects or homonymous defects. Some patients who are conscious on admission to the hospital may have a bilateral blindness which disappears after several minutes to hours. A history of a blow on the occiput may suggest the possibility of a contusional state with edema of the calcarine area bilaterally with resultant blindness. We have seen at least four or five such patients during the past fifteen years. In one of these, a residual homonymous defect was present for several days and eventually disappeared. Persisting unilateral blindness may occur in patients who are only briefly unconscious. The optic nerve of normal appearance at the first examination becomes atrophic in two to three weeks.

Sensory abnormalities of cortical origin are not common in patients with head injury. However, stereognosis may be noted. Some loss of tactile and vibratory sense on one side of the body as compared with the other may be found in some patients with extensive parietal damage. In the majority of patients, light touch and tactile sense are well preserved even though there may be known parietal lobe damage.

Certain neurologic syndromes have been described in patients with head injury involving specific areas of brain and intracranial contents. Among these are the syndrome of the superior longitudinal sinus, catatonic states, the syndrome of contusions and lacerations of the frontotemporal junction, plegic states with pseudobulbar paralytic phenomena, and the brain stem syndrome associated with temporal lobe and uncus herniations.

The syndrome of the superior longitudinal sinus was first described by Holmes and Sargent in patients with tangential fractures of the vault near the midline. Paresis of the lower limbs and one or both upper limbs was described. The pathologic anatomy was either that of contusions of the lower extremity area or a compression or thrombosis of the sagittal sinus. We have described four cases of this syndrome, which included one of a perforating wound of the sagittal sinus with an interhemispheric hematoma, two cases of depressed fracture of the interparietal region with rigidity of the lower extremities and one of an epidural hematoma

at the midline with bilateral pyramidal tract signs and triplegia. Such abnormalities may also occur as a result of brain stem herniation through the incisura.

Catatonic states in persons with head injuries almost always have left frontoparietal injuries with aphasia. The association of aphasia and catatonia was pointed out by Kleist, who thought that catatonia may be a pyramidal tract dysfunction. It is felt that a catatonic attitude suggests a left frontoparietal lesion in a right handed individual. These patients may be conscious or semiconscious. Frequently they are aphasic and when a limb is placed in a certain position this position is sustained until fatigue causes the limb to be lowered.

Contusions and lacerations of the frontotemporal junction may be associated with central facial weakness on the opposite side and jacksonian seizures of the face and the mouth on the one or both sides. These may become generalized seizures. Frontotemporal junction contusions are common in serious head injuries. When there are circumoral twitchings associated with jacksonian or generalized seizures, the possibility of frontotemporal junction contusions should be considered.

Plegic states with pseudobulbar paralytic phenomena are common in the more seriously injured individuals with extensive brain damage. Bilateral spasticity eventually disappearing, but associated with some unsteadiness and ataxia, is seen. This may result from bilateral pyramidal tract involvement in the posterior thalamus or bilateral contusions and lacerations of the motor cortex. Associated with this condition is a change in the patient's voice to a nasal tone as well as an early inability to swallow normally. Later swallowing may become normal, but the abnormal pseudobulbar speech may continue for many years.

An intracranial mass lesion, such as an extradural or subdural hematoma, as well as severe swelling of the brain may result in a brain stem herniation through the incisura or a temporal lobe herniation with the uncus extending into the posterior fossa through the incisura. Thus, the brain stem and certain cranial nerves, particularly the third cranial nerve, may be compressed. In some patients, involvement of the third cranial nerve, resulting in a dilated pupil, is due to hemorrhage from the middle meningeal artery in the middle cranial fossa. Pupillary inequality as a result of brain stem compression, bilateral pyramidal tract signs and

deceitful attitudes and rigidities are manifestations of temporal lobe or brain stem herniations. With compression of the brain stem the vessels about the structure may become compressed with resultant congestion and ischemia. Hemorrhages in the upper brain stem may occur by diapedesis. Such hemorrhages occur in patients with fatal subdural and epidural hematomas but it should be pointed out that similar lesions also may occur as a result of the initial impact. An initial impact resulting in brain stem hemorrhage may cause death within a matter of a few minutes to several hours.

Diagnosis of this condition is made by the observation of abnormalities of the pupils, body tone, the reflexes and deterioration of the conscious state. Its management in most instances consists in removal of the initial cause of the herniation. An epidural subdural or intracerebral hematoma should therefore be removed with dispatch. Prompt surgical treatment may be lifesaving. On the other hand if the temporal lobe or brain stem herniation has been present for too long a time an irreversible state may be present. Some of these patients may possibly be helped by temporal lobe excision with section of the incisural border.

Herniation of the medulla and cerebellar tonsils in acute head injury is uncommon but may occur in conjunction with a posterior fossa massive hemorrhage. Sudden stoppage of respirations with heart action continuing is the typical clinical result in the untreated patient.

The cranial nerves may be injured in cranio-cerebral trauma. The first cranial nerves are injured in association with anterior fossa fractures with injury in the neighborhood of the cribriform plates. Anosmia may result with improvement in olfactory function in eight to twelve months. If of longer duration, anosmia usually becomes permanent. In anomic patients the sense of taste is fairly well preserved at least for basic flavors.

Injury to the optic nerves locally may occur by impact of the fracture and by hemorrhage into the vaginal sheath of the nerve. Usually the blindness is permanent and the nerve head atrophies. We have seen only one instance of bilateral blindness due to optic nerve injury associated with a fracture extending to the optic foramen on one side.

The third, fourth and sixth cranial nerves are frequently injured in patients with extradural and subdural hematomas. The sixth nerve may be injured at the base because of

distortion of the brain at impact. Occasionally the third and fourth nerves are also injured in this manner. Diplopia due to paralysis of one or more ocular muscles is frequently the result of intraorbital damage. Third nerve paralysis on one side with contralateral paralysis of the body may suggest a midbrain injury but it is also seen in association with subdural and epidural hematomas in a basilar location.

Paralysis of ocular movements may occur in patients with head injuries complicated by an arteriovenous fistula between the carotid artery and the cavernous sinus. Proptosis of the eyeball develops often within forty-eight hours, chemosis occurs and a bruit may be heard over the globe. The ipsilateral common carotid artery should be promptly ligated in treating this condition.

The fifth cranial nerve may be injured in its supraorbital and infraorbital portions by fractures involving the foramina bearing the same names. Occasionally the ganglion and the entire fifth nerve are involved with fractures of the middle fossa.

The seventh and eighth cranial nerves are injured in transverse fractures of the petrous bone. Bleeding from the ear may accompany seventh nerve paralysis with longitudinal fractures invading the facial canal and the middle ear. Complete deafness with a dead labyrinth usually indicates a transverse fracture through the internal auditory meatus. Seventh nerve paralysis of the peripheral type involves paralysis of the forehead, orbicularis oculi and orbicularis oris as well as the platysma muscles. Seventh nerve paralysis may be due to injury of the nerve at the time of impact or it may result from edema and hemorrhage in the facial canal neighborhood. In the latter instance it makes a delayed appearance four to twelve days after the injury. In most of these patients peripheral facial paralysis improves. An occasional patient may need a spinal-accessory facial or hypoglossal-facial anastomosis to improve the tone of the facial musculature.

The ninth, tenth, eleventh and twelfth cranial nerves are injured in penetrating wounds of the head. They are almost never involved in the usual civilian type of head injuries. In one of our patients, the third, fourth, fifth, sixth, seventh, eighth, ninth, tenth, eleventh and twelfth cranial nerves were sheared on one side in a penetrating wound of the skull base. In a civilian patient with an occipital fracture, the twelfth cranial nerve was found to be paralyzed on one side.

the convalescent patient, although suspected by the extent and location of damage sustained at the time of the accident. Homonymous hemianopsia with occipital lobe involvement is occasionally noted. In temporal lobe lesions due to open depressed skull fractures there may be quadrant defects or homonymous defects. Some patients who are conscious on admission to the hospital may have a bilateral blindness which disappears after several minutes to hours. A history of a blow on the occiput may suggest the possibility of a contusional state with edema of the calcarine area bilaterally with resultant blindness. We have seen at least four or five such patients during the past fifteen years. In one of these a residual homonymous defect was present for several days and eventually disappeared. Persisting unilateral blindness may occur in patients who are only briefly unconscious. The optic nerve of normal appearance at the first examination becomes atrophic in two to three weeks.

Sensory abnormalities of cortical origin are not common in patients with head injury. However, stereognosis may be noted. Some loss of tactile and vibratory sense on one side of the body as compared with the other may be found in some patients with extensive parietal damage. In the majority of patients, light touch and tactile sense are well preserved even though there may be known parietal lobe damage.

Certain neurologic syndromes have been described in patients with head injury involving specific areas of brain and intracranial contents. Among these are the syndrome of the superior longitudinal sinus, catatonic states, the syndrome of contusions and lacerations of the frontotemporal junction, plegic states with pseudobulbar paralytic phenomena, and the brain stem syndrome associated with temporal lobe and uncus herniations.

The syndrome of the superior longitudinal sinus was first described by Holmes and Sargent in patients with tangential fractures of the vault near the midline. Paresis of the lower limbs and one or both upper limbs was described. The pathologic anatomy was either that of contusions of the lower extremity area or a compression or thrombosis of the sagittal sinus. We have described four cases of this syndrome which included one of a perforating wound of the sagittal sinus with an interhemispheric hematoma, two cases of depressed fracture of the interparietal region with rigidity of the lower extremities and one of an epidural hematoma

at the midline with bilateral pyramidal tract signs and triplegia. Such abnormalities may also occur as a result of brain stem herniation through the incisura.

Catatonic states in persons with head injuries almost always have left frontoparietal injuries with aphasia. The association of aphasia and catatonia was pointed out by Kleist, who thought that catatonia may be a pyramidal tract dysfunction. It is felt that a catatonic attitude suggests a left frontoparietal lesion in a right handed individual. These patients may be conscious or semiconscious. Frequently they are aphasic and when a limb is placed in a certain position this position is sustained until fatigue causes the limb to be lowered.

Contusions and lacerations of the frontotemporal junction may be associated with central facial weakness on the opposite side and Jacksonian seizures of the face and the mouth on the one or both sides. These may become generalized seizures. Frontotemporal junction contusions are common in serious head injuries. When there are circumoral twitchings associated with Jacksonian or generalized seizures the possibility of frontotemporal junction contusions should be considered.

Plegic states with pseudobulbar paralytic phenomena are common in the more seriously injured individuals with extensive brain damage. Bilateral spasticity eventually disappearing, but associated with some unsteadiness and ataxia, is seen. This may result from bilateral pyramidal tract involvement in the posterior thalamus or bilateral contusions and lacerations of the motor cortex. Associated with this condition is a change in the patient's voice to a nasal tone as well as an early inability to swallow normally. Later swallowing may become normal, but the abnormal pseudobulbar speech may continue for many years.

An intracranial mass lesion, such as an extradural or subdural hematoma, as well as severe swelling of the brain may result in a brain stem herniation through the incisura or a temporal lobe herniation with the uncus extending into the posterior fossa through the incisura. Thus the brain stem and certain cranial nerves, particularly the third cranial nerve, may be compressed. In some patients involvement of the third cranial nerve, resulting in a dilated pupil, is due to hemorrhage from the middle meningeal artery in the middle cranial fossa. Pupillary inequality as a result of brain stem compression, bilateral pyramidal tract signs and

for operative intervention. Epidural, subdural and intracerebral hematomas may be associated with focal seizures followed by focal neurologic signs.

Decerebrate states resulting as they frequently do from uncus or brain stem herniation, may be treated by temporal lobectomy or better still by section of the tentorium cerebelli, including the incisural border. In view of the serious condition of the patients and also because the procedures are undertaken in almost moribund subjects the results have not been too successful. The use of hypertonic solutions and lowering of the intracranial pressure by lumbar puncture have not been particularly helpful.

Cerebrospinal fluid findings. A lumbar puncture or a cisternal puncture if the former is not practical because of the presence of skeletal and spinal fractures, is worth while and should be carried out whenever needed. Routine use of a lumbar puncture is not suggested by most neurosurgeons at the present time. Early a lumbar puncture may upset the hydrodynamics in the cranial cavity sufficiently to influence also the signs and symptoms of a dynamic lesion. A forming epidural hematoma may be associated with lowering of the spinal fluid pressure even though the patient's mass lesion is expanding. He may expire because the mass lesion was not recognized and evacuated. Lumbar punctures may be for diagnostic or therapeutic purposes. Diagnostic lumbar punctures should be done to classify the degree of damage. The spinal fluid pressure should always be recorded. The blood content should be noted and this can be done by making a complete blood count of the cerebrospinal fluid. From previous studies and experiences with repeated lumbar punctures, it may be said that cerebrospinal fluid which is very bloody to start with may clear in about eight to ten days. Within two days, a very bloody spinal fluid may be faintly xanthochromic in some patients. The red blood cells hemolyze and disappear much more rapidly than do the white blood cells. At the end of about four or five days, a lumbar puncture may reveal xanthochromic fluid with a white cell count out of proportion to the red count in the same specimen. Usually the white cells in such a count are divided between polymorphonuclears and lymphocytes. In general, low spinal fluid pressure does not rule out the possibility of a massive lesion in the cranial cavity. However most dynamic lesions in the cra-

nial cavity are associated with fairly high cerebrospinal fluid pressures over 250 to 300 mm of water. Also bloody cerebrospinal fluid is often associated with elevated pressure.

Hypotension of the cerebrospinal fluid occurs and is often a grave sign. A seriously ill patient having a cerebrospinal fluid pressure of 75 mm or less may be in an extremely poor condition. Low cerebrospinal fluid pressure or hypotension may indicate a poor prognosis. Levels of intracranial pressure during anesthesia have been studied by Hyder, Evans and others and high levels of cerebrospinal fluid pressure were noted during such recordings. It has been suggested that increased intracranial pressure per se is not a dangerous condition. However it should be pointed out that brain swelling may be present with or without high cerebrospinal fluid pressure. In some instances the cerebrospinal fluid pressure gives an index of the degree of brain swelling. In other instances because of the presence of hydrodynamic abnormalities the level of cerebrospinal fluid pressure may not be high even though the edema of the brain is present either locally or generally.

The use of lumbar puncture for therapeutic purposes should be individualized. An occasional case of weakness of one side of the body or aphasia may clear up after a lumbar puncture. Its use as a means of removing blood in the spinal fluid to lower the incidence of pia arachnoiditis is held to be unnecessary. In autopsy subjects, bloody collections in the sulci of the hemispheres have been noted in the cranial cavity even though repeated lumbar punctures have been performed.

Concomitant injuries elsewhere in the body. In the usual high velocity deceleration injuries the possibility of damage not only to the head but to other parts of the body is great. Associated injury elsewhere in the body is often responsible for the morbidity as well as the fatal outcome. In a series of 184 consecutive head injuries studied at autopsy in sixteen instances there was major associated damage which undoubtedly influenced the outcome. Chest injuries were present in five patients, in two vertebral column injuries were noted, in three, chest and abdominal injuries and in four extremity and chest damage. Extremity injuries alone were present in six patients. Particularly in the unconscious patient, consideration must be given to the possibility of

in association with a fracture through the hypoglossal foramen

It may be said that abnormal neurologic findings may be of value in localizing the lesion and, depending upon their pattern of appearance may indicate the presence of a dynamic lesion. Weakness or paralysis of one-half the body seen soon after an injury may signify a laceration of the motor region whereas weakness or paralysis of one-half the body developing over a period of twenty-four to thirty-six hours may indicate the presence of a dynamic lesion such as a subdural, epidural or intra cerebral hematoma. The importance of repeated neurologic examinations cannot be overemphasized. Thereby with sound clinical judgment one may be able to detect the surgical type of lesion. The proper evaluation in the changes of the signs and symptoms may indicate the need for emergency intervention which may be lifesaving.

Convulsions In patients with acute head injury convulsive disorders are seen in less than 10 per cent. These include focal or jacksonian, generalized or grand mal, petit mal and psychomotor convulsions. Commonly noted are decerebrate rigidity patterns particularly in patients with serious head injuries. Less common are generalized convulsive seizures of the grand mal variety. Least common are jacksonian manifestations which may become generalized.

Decerebrate spells are seen in the seriously injured. Almost always these patients have bloody spinal fluids with elevated spinal fluid pressures. The patient may become rigid. At times the rigidity may involve one-half of the body and change from one side to the other. Usually both sides become rigid when the patient is moved or stimulated. The patient has bilateral pyramidal tract signs with Babinski reflexes present bilaterally. At times, particularly among the younger the Babinski signs may be continuous. Nuchal rigidity and opisthotonos may be seen. Usually there are no associated convulsive movements of the body but occasionally decerebrate rigidity may be followed by a generalized convulsive seizure. The patient has a set, rigid jaw and at times there may be some foaming at the mouth. The attacks usually last from a few minutes to several hours. Frequently short lasting attacks may continue to occur. As the patient fails, he may be less rigid and more flaccid. Usually decerebrate rigidity attacks in the adult mean a fatal outcome. In a child, such attacks may not be as serious and

recovery often occurs. Particularly is this true when the decerebrate state is caused by a mass lesion which can be evacuated.

Decerebrate rigidity may be due to brain stem involvement from the initial impact or it may result from brain stem and/or temporal lobe herniation resulting from brain swelling or a large epidural or subdural collection. If such subdural or epidural collections are not promptly treated, an irreversible state due to brain stem herniation may occur.

Grand mal seizures occur less frequently. Occasionally they are seen soon after injury as the patient recovers consciousness. They may be seen later in the course of a serious head injury. The spinal fluid is frequently bloody. The pressure may be high. There may or may not be a history of idiopathic epilepsy and this should be noted carefully in all patients with generalized convulsive seizures following head trauma. No definite localizing signs may be present. However in many instances there may be bilateral Babinski signs soon after a generalized convulsive seizure. At times generalized convulsive seizures occur as a sequel to jacksonian seizures or in some instances following decerebrate seizures. The attacks may become continuous, resulting in status epilepticus. This is not commonly seen in head injury patients since such individuals are energetically treated early. Patients of the younger age group, particularly infants and children, are more apt to develop convulsive seizures of a grand mal variety than are adults. At times, meningitis, traumatic cerebritis or a brain abscess may be initiated with a jacksonian or grand mal seizure.

Jacksonian seizures or focal seizures are important because they imply a discrete type of lesion of the brain commonly a mass lesion. Jacksonian seizures of the mouth and upper extremity area may be followed by generalized seizures and may signify contusions of the frontotemporal junction. More frequently localized compression of the motor areas by a subdural or epidural hematoma and contusions of the motor region may result in jacksonian seizures. These may eventuate in generalized convulsions in the untreated patient or the patient upon whom operation has not been performed.

The management of a patient with convulsive seizures requires a proper airway. In some instances, hypoxia may be the initiating factor and a good airway provided by an intratracheal tube or tracheotomy may be valuable. Focal signs may indicate the need

removal through a small incision will restore the scalp to its normal dimensions.

Open wounds of the head include linear fractures of the skull with an overlying laceration, depression of the skull with or without dural tear (Fig 5) and with or without brain laceration, cerebrospinal fluid rhinorrhea and cerebrospinal fluid otorrhea resulting from cranionasal and cranioaural communications. Penetrating wounds of the head by high velocity missiles, such as a bullet or shell fragment or by low velocity wounding, such as by sharp instruments, knife, stiletto or ax are classified as open wounds of the head.

Open fractures of the skull with no depression may be treated by closing the laceration overlying the fracture after careful cleansing and debridement of the wound. Open depressed fractures may be associated with dural lacerations in about 15 per cent of such patients admitted to large head injury services. Many patients with open depressions may have had no initial unconsciousness or may have been unconscious for a short time. The majority of depressions occur in the anterior half of the vault. Few occur in the posterior third in the occipital and the posterior parietal areas. Such depressions occur in the forward portions of the head since these portions are the most frequently injured. About 75 per cent of skull depressions have an overlying laceration making them open wounds. At times the laceration may be a small puncture wound not communicating with the underlying depression or comminution. At times the depression may be over functional centers of the brain so that the patient enters the hospital with weakness or paralysis of the opposite half of the body, aphasia or other clinical abnormalities. Depressions in the more forward portions of the head are attended with a low mortality. Depressions involving the frontal sinus may be associated with a high morbidity in view of the possibility of cranionasal communications with complicating intracranial infection.

The site of the depression may determine the underlying damage. Depressions may overlie the larger venous sinuses, such as the sagittal and lateral sinuses, producing a tear of the sinus which bleeds as the fragments of bone are removed at operation. The patient's preoperative roentgen ray studies should be carefully evaluated so that the surgeon may be prepared to meet such complications. In some depressions the middle meningeal vessels may be involved. De-

pressed fractures involving the temporal bone with a dynamic syndrome suggest the possibility of venous or arterial tears and a mass lesion in an epidural position.

A detailed roentgen ray delineation of depressed fractures is extremely important. When there is fragmentation of bone the number of fragments should be noted so that at operation one can account for their removal. This is particularly true in war wounds where fragments of bone and other organic matter left in the cranial cavity are of serious consequence in the causation of post traumatic infection.

Open wounds over the frontal sinuses and neighborhood may be associated with cranionasal communications. In many instances, such communications are established in closed injuries without evidence of a skull fracture demonstrable in roentgenograms. Such injuries in this neighborhood have to be considered as open wounds unless proved otherwise. Linear fractures extending into the base of the anterior fossa may be associated with tears of the dural lining in the region of the cribriform plate with resultant infection many months or even years later. Only at the post mortem examination may a short fracture be seen to extend into the anterior fossa and the cribriform plate. Sometimes there may be a discrete and separate skull fracture with an unsuspected dural tear because the fracture is far removed from the region of the visible damage such as in a bullet wound of the parietal or occipital region with cribriform plate fracture. Complicating meningeal infection may point to an injury in this region related to a patent cranionasal communication. However the most important causes of cranionasal fistulae are compound depressions of the frontal sinus region.

With cranionasal and cranioaural communications pneumocephalus may occur with the cerebrospinal fluid leakage. When pneumocephalus is seen in the patient with acute cranio cerebral trauma and is associated with an open depressed fracture and dural tear repair of the latter usually suffices in curing the pneumocephalic condition. On the other hand, sometimes pneumocephalus may appear several months or years after the acute injury because of the presence of a patent cranionasal communication. In some instances the patent cranionasal communication may exist even though cerebrospinal fluid rhinorrhea is not recognized or the patient shows no air in the cranial cavity upon roentgen ray examination. Under these cir-



Figure 5 A, Experimental depressed fracture in cadaver head. Note the difference between the amount of involvement on the external (A) and the internal (B) surfaces of the skull. Note that on the internal surface (B) there is extensive fragmentation of the inner table.

injuries to various portions of the body the chest and abdomen especially.

Clinical-Pathologic Correlations in Cranio-cerebral Trauma When the patient with a head injury is first seen in the emergency room of a hospital, he may appear profoundly moderately or slightly injured, as evidenced by the immediate clinical findings. He may present, upon examination, a closed head injury or an open wound of the head. A patient with a closed head injury may be conscious or deeply comatose. Patients with open wounds of the head may have varying degrees of involvement from a minor-appearing laceration of the scalp to an extensive wound with extrusion of brain tissue. Patients with open wounds and with extensive damage of nervous tissue may have little or no evidence of brain stem involvement, no initial unconsciousness or an unconscious state of a very short duration. In both types of closed and open head injuries, the condition of the patient may be influenced by later developments of the cranio-cerebral trauma, including swelling and edema of the brain, hemorrhages into the intracranial spaces and intracranial infection.

Contusions and lacerations of the scalp are the commonest types of head injury. Ecchymosis of the eyelids may rapidly develop with marked swelling or there may be small hemorrhagic discolorations, manifest several days after an injury surrounding the upper and lower eyelids as well as the conjunctiva. Ecchymosis of the mastoid area has long been recognized as indicating a fracture of the base of the skull, it is known

as *Battle's sign*. This discoloration may be more apparent two or three days after injury than immediately after the initial trauma. Lacerations of the scalp may be single or multiple. In present-day civilian injuries caused by repeated blows upon the head by a sharp instrument, multiple lacerations are common. Lacerations extending to the subgaleal region may be associated with varying degrees of avulsion of the scalp. Careful cleansing of the wound, which then should be meticulously sutured will frequently result in good healing.

In the depth of a scalp laceration, particles of bone and brain tissue may be seen on careful inspection, but in the final analysis a careful roentgen ray survey is much more accurate and dependable. Patients with extensive lacerations of the scalp may be conscious and may have had no period of unconsciousness following their trauma.

Hematomas of the scalp are frequent. At times, palpation of these may give the wrong impression of a depressed fracture. Hematomas occurring in the subgaleal region may extend to the limits bordering this space. This is particularly seen in young adults and children and may result in severe blood loss. In the infant, hemorrhage under the periosteal lining of the bones of the cranial vault may result in cephalhematoma. The characteristic feature of cephalhematoma is that it is within the confines of the periosteal attachment of the bone involved. Aspiration of the hematoma under aseptic conditions will enhance the healing and the absorption of blood. In cephalhematoma, aspiration or

in the middle fossa. At times there may not be an initial period of unconsciousness. The patient may reach the hospital conscious and eventually become unconscious or he may be brought into the hospital in an unconscious state with a history that initially there was no unconsciousness. A weakness of one half of the body may not develop with a clot more posteriorly or more anteriorly located. In most of the patients there is a linear fracture crossing the middle meningeal groove of the parietal and temporal bones. Particularly in those patients who have an initial period of unconsciousness there may be associated evidences of other involvement of intracranial structures such as pia arachnoid tears with bloody cerebrospinal fluid and bruises and lacerations of the temporal and frontal poles of the hemispheres. Massive intratemporal hematomas may occur. Oc-

asionally depressed fractures may be associated with an epidural hematoma.

The epidural hematoma is usually seen among adolescents and adults although it has also been described in children and in infants under the age of two years. In some patients, there may be third nerve paralysis on the side of the lesion and weakness or paralysis of the opposite half of the body. If the patient's condition is not recognized and immediate operation is not carried out, he may rapidly deteriorate with irreversible changes ensuing. The vital functions at first are characterized by a slow pulse, slow respirations, a temperature of about 100 to 101° F and a blood pressure which is usually within normal levels. In many instances the spinal fluid may be bloody and its pressure is moderately elevated. In the untreated patient, the pulse becomes faster and even-

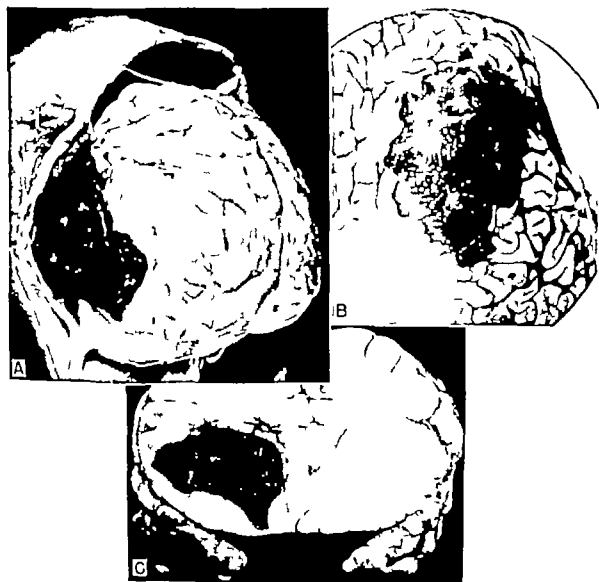


Figure 6 An epidural (A) acute subdural (B) and an intracerebral hematoma (C) seen in the autopsy room. Note the petechiae about the intracerebral hematoma in the right frontal lobe.

circumstances, the only basis for suspecting such a patent communication may be repeated attacks of meningitis. We have had a patient who has had twenty six attacks of meningitis due to a patent traumatic cranio-nasal communication.

Penetrating wounds of the head of the low velocity type, such as knife-blade and other sharp instrument wounds are commonly seen in civilian practice. Frequently there is no history of unconsciousness at the time of impact and it is possible for the blade of the knife to break off so that on inspection only a small laceration of the scalp may be seen with the end of the blade embedded deeply beyond vision, in the scalp, skull or cranium. The frequent error of misjudging such wounds must be emphasized. Careful inspection of all wounds and roentgen ray examination of the skull should prevent the occurrence of such a mistake. When such foreign bodies are left in the cranial cavity there may be an inflammatory reaction surrounding the foreign body and a brain abscess may form or meningitis may develop. When the patient has extensive involvement of the cranio-cerebral structures in open wounds, the diagnosis is usually made and prompt care of the wound results in a cure with little likelihood of infection.

High velocity wounds of the head present a different problem, particularly with bullets traveling over 1000 feet per second. The area of scalp penetration may be extensively de-vitalized so that incomplete débridement will result in breakdown of the wound. The passage of the missile through the cranial cavity results in forward as well as radial forces causing pulping of brain tissue and destruction of blood supply so that an adequate débridement requires complete removal of the brain tissue so destroyed. Improper débridement will result in late infection and cerebritis and brain abscess. Not seen in civilian life but commonly seen in war experience, are shell fragment wounds due to high velocity missiles with more extensive pulping of the brain because of the varying sizes and irregularity of the contour of the shell fragments. With wounds by shell fragments complete removal of the fragments and foreign particles is mandatory for a good result. Brain stem involvement may be associated with both bullet and shell fragment injuries of the brain so that these patients may be moribund soon after the injury. On the other hand some of the patients may show little or no evidence of brain stem involvement and consciousness may be retained until lost through factors other than

the initial trauma. Secondary involvement of the brain stem centers through brain edema or hemorrhage may occur. The possibility of intracranial hemorrhage associated with penetrating wounds of the brain is stressed by experiences in recent wars. Hematomas in the track of the bullet, particularly of small firearms, are present in fully half of the patients and removal may be lifesaving.

In open wounds, the rationale of operative management is to prevent the introduction of infection into the intracranial spaces and structures. The sooner the wound is debrided and closed the sooner protection from infection is secured. The possibility of associated hemorrhage causing compression should be considered.

Traumatic intracranial hemorrhage Intracranial hemorrhage due to trauma may be epidural, subdural, subarachnoid or intraparenchymatous. Combinations of hemorrhagic collections in various locations in the cranial cavity are quite common. Particularly is this true with the usual type of deceleration injuries such as in falls and automobile accidents.

Epidural hemorrhage (Fig 8) may be of middle meningeal, dural sinus or diploic origin. It is located between the dura and the skull. At times it forms a large mass and in some autopsy subjects as much as 350 to 400 cc. of blood may collect in such an epidural position. Occasionally the lesion is bilateral or it may cross the midline superiorly to become bilateral. The most common epidural hemorrhage is that of middle meningeal artery origin. Less common are epidural hematomas in the posterior fossa of lateral sinus tear origin and hematomas resulting from sagittal sinus tears in vertex injuries.

Middle meningeal hemorrhage is usually caused by tears of the middle meningeal artery and veins. A history of a fall or a low velocity impact, such as a bicycle-car accident falling on the sidewalk and falling down steps, is usually present. The patient may or may not have an initial brain stem syndrome with immediate post-traumatic unconsciousness. The injury may be followed by a lucid interval of a few hours to several days. This is followed by a primary or secondary attack of unconsciousness with weakness of one-half of the body and a dilated pupil on the same side as the lesion. The pupillary dilatation may not be marked in some instances and the more completely dilated the pupil, the greater the likelihood of the lesion being in a *post*

comatose with increase in the respiratory rate hyperpnea and pulmonary edema. Such patients when investigated may have a subdural hematoma on one or both sides. Prompt surgical intervention may be lifesaving. The mortality in this group is 30 to 40 per cent.

The chronic subdural hematoma (Fig. 7) as previously stated may either accompany a severe cranio-cerebral injury or it may occur without initial associated involvement of the brain stem centers. Under the latter circumstance, the patient may be well for three to seven weeks and then may complain of headache which becomes progressively worse. The headaches are usually lateralized to the side of the hematoma although in many instances subdural hematomas may be bilateral (20 per cent). In other patients there may be signs of increased intracranial pressure, choked disks and weakness of one-half of the body. Diagnostic studies may show evidence of a mass lesion. In some instances a brain tumor may be suspected when a history of trauma is not elicited from the patient or relatives, though usually such a history exists.

The chronic subdural hematoma is usually covered with a thick outer wall and a thin, almost single layer of cuboidal epithelium, inner wall. Within these walls is contained clotted and hemolyzed old blood. In other instances, the hematoma may be almost all liquid with a very thin wall surrounding the mass. Chronic subdural hematomas may be bilateral in 20 per cent of patients under any circumstance, both sides of the skull should be explored even though initial diagnostic studies may implicate only one side.



Figure 7 Chronic subdural hematoma seen in the operating room. The brain surface is exposed as the hematoma is removed through an osteoplastic flap.

The pathophysiology of the subdural hematoma may be summarized as follows: If the initial subdural bleeding is severe and if the host cannot tolerate its pressure, signs and symptoms of acute subdural hemorrhage supervene. If the hematoma is smaller the patient may tolerate its presence for three to ten days with gradually developing signs and symptoms of subacute subdural hemorrhage. If the clot is carried by the host without any symptoms for three or more weeks an attempt at absorption and organization results in formation of an outer thick wall on the dural side and a very thin wall on the arachnoid side. Its size may increase by transference of tissue fluids and cerebrospinal fluid because of its high osmotic pressure, with the arachnoid acting as a semipermeable membrane. The size may also increase from hemorrhages of blood vessels in its outer wall. With increase in size eventually the mass causes symptoms.

There are exceptions to these mechanisms. The hematoma may cause little or no symptoms for many months or years. In some patients it may calcify. In others a solid unexpanding mass of clot with a consistency of hepatic tissue may result. Some small hematomas may absorb and never cause symptoms.

The prognosis in the chronic type of subdural hematoma is gratifying. Mortality is usually 10 per cent or less and patients usually regain their faculties to return to their former occupations and live usefully.

Subdural collections of cerebrospinal fluid mimic subdural hematomas of the acute and subacute variety. They are almost never diagnosed until an operation is performed. At times such collections are found in infants and they may or may not be traumatic in origin. There may or may not be a membrane surrounding the fluid. Occasionally post-inflammatory collections (postmeningitic) in the infant and the young child are described as subdural effusions.

Traumatic subdural accumulations of cerebrospinal fluid may be due to a tear of the arachnoid membrane with escape of cerebrospinal fluid into the subdural area causing localized pressure against the brain. The presence of small amounts of bloody material in an extra-arachnoid position may attract cerebrospinal fluid and tissue fluids into the subdural area because of an increase in the osmotic pressure in this region. Contusions and tears of the brain over the convolitional peaks, with resultant increased osmotic pressure, may cause fluid accumulation in the subdural area in this neighbor-

tually theadry The respirations increase in rhythm and the patient may eventually become hyperpneic, with pulmonary edema, or there may be slow gasping respirations as a final phase before death. In the untreated patient late effects may be the result of brain stem herniation through the incisura with generalized rigidity bilateral pyramidal tract signs, dilation and fixation of pupils and loss of corneal reflexes.

The presence of an extradural hematoma of dural sinus origin in the posterior fossa should be suspected in association with an occipital fracture. The patient may have cerebellar hypotonia and weakness on the same side of the body as the hematoma. However because of the associated injury to other parts of the nervous system typical signs may not be found in the patient with a posterior fossa hematoma. Exploration along a fracture site in a patient whose condition is gradually deteriorating is justified and, under these circumstances, hematomas in the posterior fossa may be located.

Extradural hematoma from diploic and emissary veins is never of sufficient size to cause compression of the intracranial contents, although fairly large extradural hematomas in depressed fractures posterior and above the mastoid area have been seen.

Subdural hematoma is a collection of blood in the subdural area, a potential space between the dural lining and the arachnoid membrane. As previously stated, mass movements of the brain may result in tear of connecting veins between the sagittal sinus and the surface of the brain as well as between the dura and the surface of the brain. Such tears result in hemorrhage into the subdural area. Depending upon the size of the torn vessels the hemorrhage may be of proportions to result in signs of compression soon after its occurrence. On the other hand, a small amount of bleeding may occur into the subdural area and as the subdural pressure increases, the bleeding from the venous channel may be stopped. Such a hemorrhage may be silent for varying periods.

Such hemorrhages may occur over one or both hemispheres. Depending upon the severity of the head injury there may be clinical symptoms suggesting diffuse brain damage and masking a surface lesion. In the absence of brain stem involvement if the hematoma is small enough so that the patient can tolerate its presence, the condition may be asymptomatic for several days or weeks. If on the other hand, it is of larger size, there may be either immediate symptoms and signs or the symptoms and signs

may be manifested within a period of several days to a week or ten days. Thus, on the basis of the time of the appearance of clinical effects, a classification into *acute subacute* or a *chronic* type has been made. If the bleeding is severe and symptoms are manifested almost immediately one is dealing with an acute and dangerous process. If the bleeding is somewhat smaller in amount and/or if it is tolerated by the host for a week or ten days, one is dealing with a subacute form. Patients with the chronic variety may have associated severe craniocerebral injury and therefore may be ill following the trauma. These patients may improve for several weeks until evidence of a chronic subdural hematoma is noted. In other patients, the initial hemorrhage is silent for several weeks until signs of a chronic subdural hematoma supervene. The division of subdural hematomas into acute, subacute and chronic forms is of prognostic value. The earlier an acute subdural hematoma causes symptoms and signs necessitating intervention, the worse the prognosis the longer the time before intervention is indicated the better the prognosis.

The diagnostic features of acute subdural hematoma include a deteriorating conscious state with focal signs. The most common position is frontoparietal on one or both sides. However subdural hematomas may be found between the hemispheres in an interhemispheric position, in the anterior fossa and in the parieto-occipital area. They may also occur between the brain stem and the hemispheres and extend into the posterior fossa about the brain stem and medulla. The presence of a fracture of the skull is not of localizing value in most instances of acute subdural hemorrhage. In two-thirds of the patients the fracture is on the opposite side or there may be no fracture demonstrated by roentgen ray examination or by autopsy. In only one-third is the fracture on the same side as the subdural hematoma. The clot in the subdural space may be liquid and easily drainable or it may be solid with some liquid and hemolyzed portions. The type of clot is important in the management of the patient and determines the type of operative treatment to be instituted. The mortality in the acute form is 50 to 75 per cent.

Subacute subdural hematomas are tolerated by the patient for several days before signs of compression supervene. After three to ten days of a stabilizing clinical condition the patient may show increasing drowsiness, evidences of localization not previously noted and, if unt

become

OSTEOMYELITIS OF THE SKULL. Osteomyelitis of the skull is a rare complication of a craniocerebral injury at the present time but does occur and almost always it is the result of improper treatment of open fractures of the head. During the past four years we have had three patients with osteomyelitis of the skull due to this cause. As has been noted in Browder's classification osteomyelitis at the site of trauma may arise as a result of an open wound or it may occur in the area of trauma with the overlying scalp intact. The latter is the "puffy tumor" of Pott's description and at the present time it is rarely seen.

The more common form of osteomyelitis due to implantation of bacteria in an open wound is preventable by careful excision of the wound soon after injury. In instances where this has not been done an area of inflammation may supervene with purulent matter exuding from the compound wound. If improperly treated, the patient may enter the hospital with fever, leukocytosis, some rigidity of the neck and evidences of meningeal irritation. X-ray examination of the skull may show an area of bony destruction which is characteristic (Fig. 8). At operation, involvement may be seen with foreign particles, hair and other dirt embedded between the fragments of bone with purulent matter and sequestration of bone. Complete excision of the area and the use of local and systemic antibiotic therapy and chemotherapy usually result in a cure over a period of several weeks. The possibility that osteomyelitis of the skull may be associated with a subdural abscess should always be kept in mind and, if necessary, the dura should be

opened to inspect the subdural area. The skull defect may be repaired later with an inanimate transplant. However, it is important to wait at least nine months after complete healing before such a repair of the skull defect is undertaken.

CORTICAL ULCERATION, CEREBRITIS AND BRAIN ABSCESS. Cortical ulceration, cerebritis and brain abscess result from the implantation of organisms in open wounds involving the brain. Occasionally a blow to the head with concussive effects may be associated later with a brain abscess and may be unsuspected until operative removal. This type of abscess may be due to a contusional involvement with a blood borne infection of the area. Eventually encapsulation and evidences of compression may develop.

Cerebritis due to an infection of the brain which follows improper débridement of a penetrating wound is a common occurrence in war but is seldom seen in civilian practice. The management of such early brain abscesses or cerebritis is by complete excision of the area of inflammation with removal of all foreign matter.

The diagnosis of cerebritis is based upon the occurrence of fever and leukocytosis and demonstration of bone particles and foreign matter in the roentgen ray film in a patient with an improperly treated open brain injury.

Diagnostic Aids in the Management of Craniocerebral Trauma. In addition to the information obtained from the history and neurologic examination, various diagnostic aids are available in evaluating patients with craniocerebral trauma. These include



Figure 8. Trauma to the forehead in a patient with stomatitis resulted in an extensive osteomyelitis of the skull associated with a subdural abscess over the left hemisphere and an intracerebral abscess in the left temporoparietal junction. Note the area of bone removal (B).

hood Coughing and sneezing have been thought to cause tears of the arachnoid membrane in some patients with nontraumatic subdural accumulations of cerebrospinal fluid. Finally certain inflammatory diseases involving the pia arachnoid may cause collections in the subdural area. Subdural collections may also be found in the posterior fossa, under the tentorium and about the cerebellar lobes.

The symptoms of a subdural accumulation of cerebrospinal fluid include a lucid interval in some patients weakness or paralysis of one-half of the body generalized or jacksonian seizures and a general deterioration of the patient's status. Subdural collections of cerebrospinal fluid may be found bilaterally. They may coexist with subdural and epidural hematomas.

Intracerebral hematomas are less common than subdural hematomas. Deep contusions of the brain with necrosis and bleeding from vessels may result in a large intracerebral hematoma. Actual tears of vessels in the substance of the brain may be associated with a hematoma of this type or there may be areas of infarction with hemorrhage in the distribution of larger vessels. Frontal and temporal hematomas are most common. Parieto-occipital hematomas are occasionally seen. Parenchymatous hematomas also may occur in the posterior fossa in the cerebellar lobes.

The signs and symptoms of intracerebral hematoma depend upon the location of the lesion. In the temporal and frontal portions of the hemisphere there may be pyramidal tract signs in the opposite half of the body and speech disturbances if the lesion is on the left side. The intracerebellar hematoma is difficult to diagnose except when there is an open wound suggesting the presence of a lesion in this vicinity. If the patient can stand the initial effects of the intracerebellar hematoma, a diagnosis may be reached at the operating table by exploration of the posterior fossa as indicated by the ventriculographic studies. It is interesting to note that in several patients with intracerebellar hematomas a marked psychotic state was noted. This may possibly have been due to increased intracranial pressure brought about acutely by the compressing hematoma against the aqueductal area.

Infections. Infections complicating head injury include meningitis osteomyelitis and brain abscess. Before antibiotic therapy and chemotherapy otitis media and mastoiditis were fairly common following head injuries involving the middle fossa and temporal

bone. Otitis media usually was seen six to eight weeks following the injury but could also occur soon after the accident. Mastoiditis necessitating mastoidectomy was also fairly common. Such complications have been seen rarely during the past fifteen years in our services. At the present time meningitis is seen occasionally. Osteomyelitis may occur particularly in the improperly treated open injuries of the head. Cerebritis and brain abscess may develop in inadequately debrided open injuries of the brain.

MENTINGITIS. Traumatic meningitis may be noted six to eight days after an injury. In the seriously ill patient, its presence may be detected only through the study of the cerebrospinal fluid. When it occurs six to eight days after the injury it frequently is ushered in as a fulminating disease. In a patient who has improved from the initial effects of the accident, there may be severe headaches, disorientation and coma. Occasionally the disease may be initiated with a convulsive seizure. A high fever with a delirium state is common. The patient presents nuchal rigidity and there may be a Kernig sign. The spinal fluid examination reveals a turbid fluid with large numbers of polymorphonuclear cells and, in some instances, organisms which can be seen in the smear. Occasionally meningitic infection may follow upon the presence of a patent craniofacial or a cranioaural fistula. It may occur in conjunction with a brain abscess.

The prevention of meningitis is undoubtedly aided by the use of antibiotics and chemotherapy in patients who have open injuries of the head or who have communications between the outside and the cranial cavity through the nose or through the ear. Undoubtedly many meningitic infections are prevented with the use of these drugs. In such patients, the preventive use of 400,000 to 800,000 units of penicillin and 80 to 120 grains of sulfonamides per day may be effective.

When a diagnosis of meningitis is made, the drug dosage is increased. From 10 to 15 million units of aqueous penicillin are administered daily for four or five days until the meningitis has been controlled. Daily 200 to 400 grains of sulfonamides are given. In the meantime, the organisms are studied by smear and culture and their drug sensitivity is identified. As the results of these tests are available the medicinal treatment is altered as indicated. The spinal fluid obtained after the fever and the leukocytosis have subsided should contain few or no pus cells.

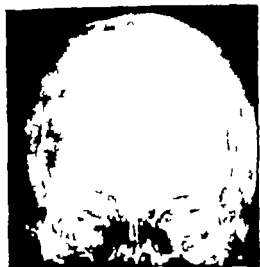


Figure 12. Pineal shift to the right showing the side of involvement. In bilateral lesions a pineal shift may be misleading, with the smaller lesion unsuspected. When a subdural hematoma is suspected, both sides should be explored in spite of lateralizing signs and diagnostic studies.

cranial conditions by means of four or more openings in the head, in the frontal temporal parietal and occipital regions on each side. The use of multiple trephinations without the additional use of diagnostic studies may lead to serious mistakes.

From studies carried out thus far it appears that electroencephalography in the patient with acute injury is of questionable diagnostic or prognostic value. In patients with the more chronic conditions such as a chronic subdural hematoma, electroencephalography is valuable and localizes the lesion in about 60 per cent of the cases.

Indications for Operative Intervention
About 25 per cent of patients with cranio-cerebral injuries need operative intervention, including diagnostic operative procedures. Among those with skull fractures, the incidence of required operation is higher (35 per cent). About 15 per cent require débridement for compound or open wounds of the



Figure 13. Anteroposterior (A) and lateral (B) views of angiogram in a patient with a left subacute subdural hematoma.



Figure 14. Angiograms in a patient with a right temporal intracerebral hematoma (anteroposterior [A] and lateral [B] views). Note the elevation and bowing of the middle cerebral artery and branches.

survey roentgen ray studies of the skull, lumbar or cisternal puncture, air studies including ventriculography and encephalography (Figs. 9 to 11) angiography and electroencephalography. Isotope studies have been infrequently used in craniocerebral trauma and, therefore, conclusive information pertaining to their diagnostic value is not available.

Survey roentgen ray studies are extremely valuable. The presence of a linear fracture, its position, the presence of comminution of fragments, depression, the number of fragments of bone in a depression and the presence of a foreign body in the cranial cavity may be detected by such studies. In those who have a calcified pineal gland, the presence of a pineal shaft may aid in the diagnosis of a mass lesion (Fig. 12).

Lumbar puncture is a valuable diagnostic aid. The concentration of blood in the cerebrospinal fluid may indicate the degree of subarachnoid hemorrhage and the severity of the intracranial involvement. The pressure of the cerebrospinal fluid in some instances may point to a mass lesion, although normal or low levels of cerebrospinal fluid pressure may be seen with mass lesions. The total protein content of the cerebrospinal fluid, if elevated, may indicate a poor prognosis. Glutamic oxalacetic transaminase determinations may show an increase in association with brain contusions and lacerations.

Air studies are valuable for localizing lesions or ruling out their presence. Ventriculography is preferable in the acute case. Encephalography may be used in the more chronic cases after the first two weeks. In the acute case, air studies may be dangerous. Such patients may have marked swelling of the brain and additional pressure caused by the injected air may be injurious. In order to have adequate air studies, sufficient air has to be introduced in the ventricular system of cavities. This is possible in patients who have survived the early effects of the injury for three or four days, but, in those who are injured ten to twenty hours earlier, ventriculography may be fatal (Figs. 10-12).

Angiography (Figs. 13 and 14) with the patient under local anesthesia is of value in craniocerebral trauma presenting a syndrome of increasing deterioration of the conscious state and focal manifestations. Hypaque is the contrast medium of choice.

Multiple exploratory trephinations are of ten unsatisfactory in many patients. Some surgeons feel that one may survey the intra-



Figure 9 Air encephalogram in a patient with a left parietal epidural hematoma.



Figure 10. Air encephalogram in a patient with a right subacute subdural hematoma. Note the relative absence of subarachnoid pathways on the right side.



Figure 11 Air encephalogram in a patient with a left chronic subdural hematoma. Note the ventricular shift to the right and the absence of subarachnoid pathways on the left.

help in patients with hypertonicity caused by hypothalamic or frontobasilar brain involvement. A kidney shutdown syndrome associated with extensive injury to other parts of the body should be carefully evaluated and the patient's fluid intake should be restricted during this period of kidney dysfunction. In patients with severe head injury the importance of proper blood matching when a transfusion is required cannot be overemphasized.

Specific measures. The use of certain drugs may be worth while to combat the effects of injury and prevent infection as well as for analgesia and sedation. Intravenous administration of hypertonic solutions and lumbar puncture for controlling cerebrospinal fluid pressure and brain swelling may be indicated.

Among the drugs used recently to help control the effects of a head injury are caffeine sodium benzoate, atropine, urea compounds and cortisone. Caffeine sodium benzoate was first suggested by Foster Kennedy and his associates as an agent to relieve increased intracranial pressure. The efficacy of this drug has not been widely accepted and its value as an agent to relieve brain swelling has not been definitely proved. Atropine in doses of 1/10 grain for the adult has been used by Ward who obtained excellent results in some severely injured patients with closed head injuries. The dosage is repeated every six hours for two or three days. Atropine—a parasympathetic paralyzing agent—may counteract acetylcholine liberated in the cerebrospinal fluid in the patient with a serious head injury. In our experience its use has not been of particular value.

Cortisone given in descending doses for a period of three to five days, has been employed by us with beneficial effects upon some of the patients. The stress of the injury may be in part responsible for the patient's serious condition and for such patients the use of cortisone has seemed valuable. In some instances, moribund patients have improved spectacularly. Patients unconscious for several days and proved not to have an intracranial mass lesion have seemed to become ambulatory sooner with the use of this drug than would otherwise be anticipated. However cortisone, or any drug, should be discriminately employed. One must not overlook the presence of a mass lesion in the cranial cavity when utilizing drug therapy.

The drugs used to prevent infection in the cranial cavity or to control infection include the sulfonamides and antibiotics. In patients with open wounds of the head, these agents

are given freely before and after operation. In patients who have cerebrospinal fluid otorrhea or rhinorrhea and bleeding from the ear these drugs should be employed routinely. Undoubtedly the occurrence of meningitis may be prevented by their use. When meningitis is manifest the cerebrospinal fluid should be carefully studied for the organisms responsible and adequate doses should be administered of the drugs to which these organisms are sensitive.

The restless patient may be made comfortable with provision for good hygiene—a clean dry bed, care of the distended bladder and attention to the bowels. By such measures restlessness may be reduced and the need for sedatives and narcotics minimized. A tracheotomy and clean respiratory passages relieve a patient's distress in breathing and promote comfort and rest. However patients who fail to obtain satisfactory relief through these measures may need sedatives. Sedatives in head injury patients should be used with care. Morphine should not be employed except in patients with long bone fractures and excessive pain and only a mild craniocerebral injury. Codeine and phenobarbital given by mouth or intramuscularly have been found adequate to allay restlessness in most patients with head injuries. These drugs do not influence the conscious state or localizing signs in a dynamic lesion as does morphine.

The use of hypertonic solutions was in vogue for many years but at the present time is seldom practiced. Weed and Mc Kibben were able to show a definite lowering of the cerebrospinal fluid pressure following intravenous administration of sodium chloride. Fifty per cent glucose solution was used by Peet as an agent to decrease intracranial pressure. Following the reports of these men, many others have recorded their results through the use of hypertonic solutions. Browder and his associates, and Webster and Freeman have shown that glucose administration results in a temporary decrease in the cerebrospinal fluid pressure followed by an increase which frequently surpasses the initial pressure. During our study of the effects of hypertonic solutions, it was noted that the seriously injured patient showed improvement as the pressure dropped but became restless and stuporous again as the cerebrospinal fluid pressure rose. In the majority of clinics today hypertonic solutions are used sparingly.

Recently experimental and clinical studies have indicated that the intravenous administration of 30 gm. of urea dissolved in 300

head. The rationale for the treatment is to prevent the introduction of infection into the cranial cavity. In the remaining 15 to 20 per cent, exploration is undertaken mainly for intracranial mass lesions.

Indications for operative intervention are clear cut in open wounds of the head. As soon as the patient's condition permits, the wound is debrided and repair is carefully made. In closed depressions, the degree and area of the depression must be considered and the advisability of surgical treatment determined. The majority of patients with simple depressions should be operated upon except for those having slight depressions in the frontal area and small simple depressions over the midline and the large venous sinuses.

Exploration for an intracranial hemorrhage is indicated when there is a deteriorating conscious state and when there are progressive focal localizing signs indicating a mass lesion.

Treatment. The conservative management of craniocerebral injuries is determined by the degree of the injury. Patients with minimal injuries are treated by observation in the hospital for two or three days. They are seen later in follow up from time to time since among this group a few will develop a subdural hematoma or subdural accumulation of cerebrospinal fluid. The patients with moderately severe injuries usually show improvement over a period of several hours to several days. These patients deserve careful neurologic observation during their stay in the hospital for the development of complications requiring surgical intervention. After the repair of small scalp lacerations the patient is made comfortable with mild sedation and is placed in semi Fowler's position in bed. The request is made for periodic observation of the pulse, blood pressure, respirations and temperature.

The patient with a serious injury deserves continuous attention. Many of these patients on entrance to the hospital have no need for surgical treatment. A few among them are moribund and a fatal outcome is evident. On the other hand, some patients in this group appearing near death may be resuscitated. Every effort should be made to accomplish this. Such patients may remain desperately sick for several days and their management involves meticulous nursing care. This includes keeping the air passages patent and clear, preventing the aspiration of vomitus, adequate fluid and food intake, temperature control, turning, bladder care and providing rest and comfort.

If there is continued respiratory distress, tracheotomy is imperative. This is usually done at the bedside and may be carried out with dispatch. Such a patient also should have a Levin tube passed into the stomach and the stomach contents removed to avoid aspiration during vomiting. The largest size tube should be used for tracheotomy intubation.

Frequent toilet of the upper respiratory passages by suction is essential. This is more important than the use of oxygen. The latter may be administered through a small catheter extending into the tracheotomy tube. Large tubes should not be placed in the tracheotomy tube because this reduces the airway and consequently makes breathing more difficult.

A small nasal tube passed into the stomach may be used for feeding the patient. The adult may be given 2000 calories of nutrient in 2500 cc of fluid per day and proportional amounts may be used for the younger patient.

A high temperature may quickly follow a cerebral injury without sufficient evidence of infection to account for it. In these patients, the use of an automatically regulated blanket to reduce the temperature is valuable. The production of a hypothermic state of from 31 to 35 C. in these patients with damage to the central temperature control mechanism in the brain stem has been helpful.

In the patient who remains unconscious for several days, determination should be made of the output of sodium and nitrogenous products in the urine and of the levels of urea nitrogen, sodium, potassium and chlorides in the blood plasma. These values should be checked frequently in order to detect the development of a metabolic disorder of central origin. Such an abnormality may be more common than is suspected. If depletion states are noted, these should be adjusted over a period of several days by intravenous administration of adequate amounts of solutions. If a state of hyposmolarity is noted, careful evaluation of the kidney function as well as the composition of the patient's feeding should be made. One may find that the patient is on a diet too high in protein which is resulting in derangement of kidney function not only for the protein but also for sodium, thus causing hyperosmolarity of the blood. Lowering of the protein intake and administration of adequate amounts of carbohydrates and fats may suffice to correct the condition. Mercurial diuretics and forced fluids may be of

thin cortex if the clot is mostly fluid. When the clot is mostly solid, an adequate bone flap, uncapping of the clot by suction and removal are indicated.

READING REFERENCES

- Braden, F. H. and Browder, J.: Evaluation of Sorbitol as a Dehydrating Agent. *Am. J. Surg.* 49:231, 1940.
- Browder, J.: Osteomyelitis of the Cranial Bone. Post-traumatic Epidural Abscess. Septic Thrombophlebitis of the Dural Venous Sinuses. In Brock, S.: *Injuries of the Skull*. Baltimore: William Wood & Company, 1910. Chap. 3, p. 41.
- Davis, L.: *Principles of Neurological Surgery*. 4th ed. Philadelphia, Lea & Febiger, 1933.
- Goodman, E. S.: Catatonic Motor Phenomena in Skull Fracture with Brain Injury. *J. Nerv. & Ment. Dis.* 53:493, 1931.
- Goodman, E. S. and Webster, J. L.: *Mechanism, Diagnosis and Management of Head Injury*. Boston, Little Brown & Company, 1939.
- Holmes, G. and Sargent, P.: Injuries of the Superior Longitudinal Sinus. *Brit. M. J.* 2:493, 1915.
- Hoover, C. R., and Mayfield, J. H.: Role of Upper Cervical Roots in Production of Pain in the Head. *Am. J. Surg.* 78:42, 1919.
- Javid, M. and Settlage, P.: Effect of urea on cerebrospinal fluid pressure in human subjects. *J. A.M.A.* 160:943, 1930.

- Kennedy, J. and Worth, S. B.: How to Treat Head Injuries and Appraise Them. *J.A.M.A.* 99:1352, 1932.
- Kleist, K.: Paralysis Agitans, Stammganglien und Mitteilungen. *Deutsche med. Wochenschr.* 51:1725, 1925.
- Munro, D.: *Treatment of Injuries to the Nervous System*. Philadelphia, W. B. Saunders Company, 1932.
- Pett, M. M.: Reduction of Increased Intracranial Pressure by Intravenous Administration of Glucose and Hypertonic Ringer's Solution. *J.A.M.A.* 84:1991, 1925.
- Raney, A. A., Raney, H. B., and Hunter, C. R.: Chronic Post-traumatic Headache and the Syndrome of Cervical Disc Lesions Following Head Trauma. *J. Neurosurg.* 6:458, 1919.
- Howarth, C. F., and Hammersley, D. P.: *Pictorial Introduction to Neurological Surgery*. Baltimore, Williams & Wilkins Company, 1933.
- Ryder, H. W. and others: Failure of Abnormal Cerebrospinal Fluid Pressure to Influence Cerebral Function. *Arch. Neurol. & Psychiat.* 70:563, 1933.
- Ward, A. Jr.: Atropine in the Treatment of Closed Head Injury. *J. Neurosurg.* 7:398, 1930.
- Weinert, J. E., and Freeman, V. E.: Studies on Cerebrospinal Fluid Pressure in Unanesthetized Dogs. *Ann. Surg.* 113:556, 1911.
- Weed, L. H. and McKibben, P. S.: Pressure Changes in Cerebrospinal Fluid Following Intravenous Injection of Solutions of Various Concentration. *Am. J. Physiol.* 48:512, 1919.

The Central and Peripheral Nervous Systems

By BRONSON S. RAY, M.D.

BRONSON SANDS RAY is an Indianan whose father is also a doctor. He graduated from Franklin College in Indiana and from Northwestern University Medical School. He received his surgical training in Chicago and at the Peter Bent Brigham Hospital in Boston. He has spent his professional life in neurologic surgery at Cornell University Medical School.

Cerebrum and Cerebellum

The surgical treatment of diseases of the brain has become an important specialty within this century. The contributing factors have been acceleration in the acquisition of knowledge of functions of the nervous system, classification of the pathologic characteristics of neoplasms, development of diagnostic methods, and the application of the

increasing improvements in surgical technique, in anesthesia and in supportive care of the patient.

INTRACRANIAL TUMORS

Although tumors constitute only one of the numerous intracranial diseases requiring surgery they have commanded the principal

cc. of invert sugar produces a significant and well sustained decrease in intracranial pressure which is elevated following injury or the presence of space-occupying lesions.

The therapeutic value of lumbar puncture is questionable. Amelioration both by lowering the cerebrospinal fluid pressure and removing the bloody fluid is doubtful. Lumbar puncture will lower the cerebrospinal fluid pressure in some patients with localized areas of swelling of the brain, this may be beneficial. We have seen patients with aphasia recover their ability to speak soon after a lumbar puncture was made and unconscious patients immediately improve, but such instances are rare. Sudden deaths have also been observed immediately following a lumbar puncture, mainly in children. In patients with dynamic lesions, such as epidural and subdural hematomas, the use of lumbar puncture is contraindicated. The hydrodynamics in the cranial cavity may be upset by lumbar puncture and hemorrhage will increase. False impressions as to conditions prevailing in the cranial cavity may be obtained. When a lumbar puncture is done it is best not to lower the pressure too rapidly. A good rule to follow is to bring the pressure down to three-fourths of its original level, even though this may involve removal of a variable quantity of cerebrospinal fluid and three-fourths of the original level may still be a high level of pressure. Incisural herniation in the presence of a mass lesion may be precipitated by a lumbar puncture.

The cautious use of a diagnostic lumbar puncture at any time in the course of the treatment of a patient with head injury is indicated. When infection is suspected, lumbar puncture should be done with dispatch to determine whether evidences of infection exist in the cerebrospinal fluid. In some instances, intrathecal medication may be advisable.

Operative management. The operative treatment of patients with head injury includes the management of simple and open depressions of penetrating wounds and of patent cranionasal and cranioaural communications manifested by cerebrospinal fluid discharge through these paths and/or pneumocephalus due to the introduction of air through the same pathways. Repeated meningitic infection after head injury may indicate the presence of a patent cranionasal fistula.

In patients with depressions, the possibility of involvement of large venous sinuses

should be considered. A torn sinus is repaired either by the use of mattress sutures or the application of Gelfoam, which is held in place over the tear in the sinus by bridging stitches. Occasionally repair by a vein graft may be indicated. Venous sinus injuries are more frequently seen in penetrating wounds of war than in civilian casualties. When torn the lateral sinus may be doubly ligated. The anterior third of the sagittal sinus, if torn may also be doubly ligated. The posterior two-thirds of the sagittal sinus and confluence of sinuses must be repaired to maintain the continuity of blood flow.

In the repair of a cranionasal fistula or a cranioaural fistula the use of a fascial transplant is desirable. A complete watertight closure of the area of fistulous communication must be accomplished. Patients having repeated meningitis following a head injury should have an adequate exposure of the area of fistulous communication for repair. Closure may be made with either mattress sutures or a fascial transplant. When extensively torn areas are exposed, a piece of fascia lata, obtained from the thigh, may be sutured as a graft inside the dural opening. The use of muscle stamps or Gelfoam should not be depended upon when closure by suture or a graft can be effected. The opening in the bone may be closed with a methylmethacrylate prosthesis.

In the management of the patient with an epidural hematoma, an adequate craniotomy opening should be made over the clot, the clot removed and the dead space between the dura and the bone should be obliterated. For a patient with a posterior fossa epidural hematoma, exploration in the vicinity of an occipital fracture may be followed by enlarging this opening to an adequate size for removal of the clot.

In a patient having a subdural hematoma (acute, subacute and chronic) the use of multiple trephine openings to drain and counterdrain a liquid clot is frequently successful. When the clotted material is solid, a small bone flap is indicated. Bilateral explorations should always be carried out. Subdural accumulation of cerebrospinal fluid is best treated by trephine openings on the affected side. Such collections are often bilateral.

Intracerebral hematomas may be identified through a trephine opening. They may be drained through a larger craniotomy opening after the area of hemorrhage is uncapped by applying suction to the overlying

intracranial tumors is indicated in Table 2 compiled from a survey for a twenty two year period at the New York Hospital. The figures are similar to those of compilations from numerous other clinics, the slight differences reflecting some particular circumstance which may have attracted more of one type of case to one clinic than another. For example, pituitary adenomas had a high incidence of 17.8 per cent in Cushing's series because of his interest in the pituitary gland, whereas in the series reported here there is a disproportionately high percentage of metastatic tumors because of the proximity of an affiliated neurosurgical service in a hospital for cancer. The variation in the relative frequency of types of glioma in different series, particularly between glioblastoma and astrocytoma and astroblastoma, reflects the different attitudes of pathologists in regard to these tumors.

CLINICAL SYNDROMES OF INTRACRANIAL TUMORS

There are certain general effects to be expected from the presence of a tumor in or on the brain without regard to the pathologic type, yet there are distinct clinical aspects and characteristics in behavior that individualize most types of brain tumor. In addition, there are different clinical syndromes which result from the presence of tumors in various areas of the brain. In children, the majority of tumors occur beneath the tentorium in the cerebellum and brain stem, and some types of these tumors do not occur in persons of older age. The converse is true in adults who more commonly have supratentorial tumors, many of which are meningiomas not seen in children. Symptoms indicative of pressure on the optic nerves and chiasm are likely in children to be due to craniopharyngioma, while in adults they are usually due to a pituitary adenoma.

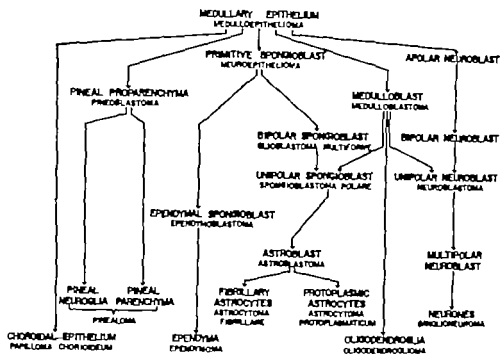
The manifestations of an intracranial tumor fall into two classes: those resulting from increased intracranial pressure and those caused by irritative or destructive effects on specific regions of the brain. While it is advantageous to recognize the presence of a tumor before papilledema and significant increase in intracranial pressure develop, tumors in so-called silent areas of the brain and those which block the cerebrospinal fluid pathways may cause only increased intracranial pressure. Supratentorial tumors, by displacing the cerebral hemispheres, may produce herniation of the temporal lobes through the tentorium, resulting in a variety

of effects including compression of the mesencephalon, narrowing of the aqueduct of Sylvius and occlusion of local blood vessels, any one of which effects may lead rapidly to serious and irreversible damage to the brain, particularly coma and the decerebrate state. Infratentorial tumors in addition to producing direct pressure on the medulla, may cause herniation of the cerebellum into the foramen magnum and additional medullary compression with signs of vascular and particularly of respiratory failure.

As intracranial pressure increases and before the more dire effects develop there are frequently symptoms of *headache*, *vomiting*, *falling visual acuity* and *diplopia*, though all of these may not occur. *Headache* rarely has value in localizing an intracranial tumor, exceptions being the occasional lateralization to one side of the head over a supratentorial tumor and localized suboccipital pain with a tumor of the posterior cranial fossa. In about 15 per cent of the patients having increased pressure there is no headache at all and when present, headache is rarely of severe degree; if it does become an excruciating pain there is imminent danger. *Vomiting* may at times be secondary to the pain but more often it is due to irritation of vagal centers in the medulla. Exertion, straining or sudden change in position by further altering the intracranial pressure may enhance the headache or lead to sudden and forceful vomiting. Matutinal vomiting on rising from bed is a common manifestation of posterior fossa tumors, particularly in children.

Some degree of *blurring in vision* is a very common accompaniment of papilledema which occurs with increased intracranial pressure, though significant loss of acuity is slow to develop. This characteristic is of value when a differential diagnosis must be made between papilledema and optic neuritis, since in the latter there is rapid loss of visual acuity. *Diplopia* which sometimes accompanies increased intracranial pressure, results from compression of an abducens nerve occasionally both abducens nerves and before a gross squint can be recognized the patient is aware of duplication of objects on the horizontal plane.

The features which make for precise localization of a brain tumor are those that have to do with motor and sensory functions. The development in a local area of the body of a change in muscle function, such as convulsive movements, weakness, rigidity, incoordination, adventitious movements and re-



SCHEME TO RELATE TYPES OF GLIOMAS ACCORDING TO THE PREDOMINANT CELLULAR CONSTITUTION OF EACH GROUP (BAILEY)

Figure 15

effort in the development of intracranial surgery and continue to present many unsolved problems. Intracranial tumors are relatively uncommon but are not to be regarded as rare. In one large clinic, a survey showed that 7 per cent of all neoplasms removed surgically were located intracranially. In the same survey the incidence of carcinoma of the stomach and of the rectum was about 10 per cent, of the colon about 9.5 per cent and of the female breast nearly 10 per cent. Although intracranial tumors occur less frequently in infancy and in youth than at other times, no age group is spared. The greatest incidence occurs in individuals between the ages of thirty and fifty years. The sexes are equally affected and, as far as is known, there is no racial variation.

PATHOLOGIC CLASSIFICATION OF INTRACRANIAL TUMORS

Intracranial tumors may be secondary, occurring by metastasis from malignant lesions elsewhere in the body or they may be primary in which case they are derived from the brain itself or some of the other intracranial structures such as blood vessels, cranial nerves, the meninges or the hypophysis. The commonest tumors, the gliomas, arise from the neuroglia, or supporting tissue of the brain, and make up nearly half of all primary brain tumors. The gliomas are subclassified according to the resemblance of

the predominating cells to the various embryonal or adult cells in the brain: the more anaplastic the cells, the more malignant the tumor. The classification of gliomas proposed by Bailey and Cushing and based on the histogenesis of the brain (Fig. 15) has been almost universally employed; other classifications have usually been modifications. A more recent classification proposed by Kernohan, Mahon, Siven and Adson is based on the idea that gliomas arise from adult cells still capable of proliferation by a process of dedifferentiation, or anaplasia. These authors suggest that the commonly used names polar spongioblastoma, astroblastoma and glioblastoma multiforme be replaced by a gradation of malignancies using the name astrocytoma, grade 1 to 4, grade 1 being the least malignant and grade 4 the most. This has the appeal of simplicity yet the histogenic classification has the advantage of long use and familiarity.

While the gliomas tend to infiltrate the brain, the next commonest intracranial tumors are the meningiomas which arise from the meninges and displace rather than invade the brain. The same obtains in most of the other tumors such as the neurinomas arising from nerve sheaths, the hypophyseal adenomas, the congenital tumors which arise from developmental defects and the blood vessel tumors.

The frequency of the different types of in-

intracranial tumors is indicated in Table 2 compiled from a survey for a twenty-two year period at the New York Hospital. The figures are similar to those of compilations from numerous other clinics, the slight differences reflecting some particular circumstance which may have attracted more of one type of case to one clinic than another. For example, pituitary adenomas had a high incidence of 17.8 per cent in Cushing's series because of his interest in the pituitary gland, whereas in the series reported here there is a disproportionately high percentage of metastatic tumors because of the proximity of an affiliated neurosurgical service in a hospital for cancer. The variation in the relative frequency of types of glioma in different series, particularly between glioblastoma and astrocytoma and astroblastoma, reflects the different attitudes of pathologists in regard to these tumors.

CLINICAL SYNDROMES OF INTRACRANIAL TUMORS

There are certain general effects to be expected from the presence of a tumor in or on the brain without regard to the pathologic type, yet there are distinct clinical aspects and characteristics in behavior that individualize most types of brain tumor. In addition, there are different clinical syndromes which result from the presence of tumors in various areas of the brain. In children, the majority of tumors occur beneath the tentorium in the cerebellum and brain stem and some types of these tumors do not occur in persons of older age. The converse is true in adults who more commonly have supratentorial tumors, many of which are meningiomas not seen in children. Symptoms indicative of pressure on the optic nerves and chiasm are likely in children to be due to craniopharyngioma, while in adults they are usually due to a pituitary adenoma.

The manifestations of an intracranial tumor fall into two classes: those resulting from increased intracranial pressure and those caused by irritative or destructive effects on specific regions of the brain. While it is advantageous to recognize the presence of a tumor before papilledema and significant increase in intracranial pressure develop, tumors in so-called silent areas of the brain and those which block the cerebrospinal fluid pathways may cause only increased intracranial pressure. Supratentorial tumors, by displacing the cerebral hemispheres, may produce herniation of the temporal lobes through the tentorium, resulting in a variety

of effects including compression of the mesencephalon, narrowing of the aqueduct of Sylvius and occlusion of local blood vessels, any one of which effects may lead rapidly to serious and irreversible damage to the brain, particularly coma and the decerebrate state. Infratentorial tumors in addition to producing direct pressure on the medulla, may cause herniation of the cerebellum into the foramen magnum and additional medullary compression with signs of vascular and particularly of respiratory failure.

As intracranial pressure increases and before the more dire effects develop, there are frequently symptoms of *headache, vomiting, falling visual acuity and diplopia*, though all of these may not occur. *Headache* rarely has value in localizing an intracranial tumor, exceptions being the occasional lateralization to one side of the head over a supratentorial tumor and localized suboccipital pain with a tumor of the posterior cranial fossa. In about 15 per cent of the patients having increased pressure there is no headache at all and when present, headache is rarely of severe degree. If it does become an excruciating pain there is imminent danger. *Vomiting* may at times be secondary to the pain, but more often it is due to irritation of vagal centers in the medulla. Exertion, straining or sudden change in position by further altering the intracranial pressure may enhance the headache or lead to sudden and forceful vomiting. *Matutinal vomiting on rising from bed* is a common manifestation of posterior fossa tumors, particularly in children.

Some degree of *blurring in vision* is a very common accompaniment of papilledema which occurs with increased intracranial pressure, though significant loss of acuity is slow to develop. This characteristic is of value when a differential diagnosis must be made between papilledema and optic neuritis, since in the latter there is rapid loss of visual acuity. *Diplopia* which sometimes accompanies increased intracranial pressure, results from compression of an abducens nerve, occasionally both abducens nerves and before a gross squint can be recognized the patient is aware of duplication of objects on the horizontal plane.

The features which make for precise localization of a brain tumor are those that have to do with motor and sensory functions. The development in a local area of the body of a change in muscle function, such as convulsive movements, weakness, rigidity, incoordination, adventitious movements and re-

Table 2. *Verified Intracranial Tumors (New York Hospital 22-Year Period)*

		NUMBER	PERCENTAGE
<i>Gliomas</i>		745	52.5
	Glioblastoma multiforme	392	
	Astrocytoma	202	
	Medulloblastoma	45	
	Oligodendroglioma	28	
	Spongioblastoma polare	13	
	Astroblastoma	6	
	Ependymoma	32	
	Pilocoma	12	
	Ganglioneuroma	3	
	Neuroepithelioma	1	
	Papilloma, choroid plexus	8	
<i>Meningiomas</i>		210	14.8
<i>Pituitary Adenoma</i>		110	7.7
	Chromophobe	85	
	Chromophil	11	
	Mixed	12	
	Adenocarcinoma	2	
<i>Acoustic Neuromas</i>		92	6.5
<i>Blood Vessel Tumors</i>		53	3.7
	Hemangioblastomas	18	
	Angiomas	35	
<i>Congenital Tumors</i>		35	2.4
	Craniopharyngiomas	20	
	Cholesteatomas	4	
	Dermoids	3	
	Teratomas	2	
	Chordomas	6	
<i>Metastatic</i>		156	11.0
<i>Miscellaneous and Unclassified</i>		19	1.3
	Cysts	6	
	Granulomas	5	
	Other	8	
<i>Total</i>		1420	

flex changes indicates involvement of motor areas of the brain, the cortex, the tracts or certain cranial nerves. If there are aberrations or defects in smell, vision, hearing or of the various tactile senses these too may point to the involvement of specific structures of the brain. Exceptions sometimes exist because of the remote effects of altered intracranial pressure as exemplified by hemiparesis, or even a lateralized fit which may occur with a tumor that occupies a region of the cerebrum removed from the motor cortex and tracts. But the localizing value of certain sensory disturbances is highly reliable. For example, an uncinate fit in which the patient experiences fleeting episodes of unpleasant odors occurs only when the uncus in one of the temporal lobes is directly affected by tumor. Loss of discriminatory and position sense in an extremity with preservation of other forms of sensation, indicates direct involvement of the postrolandic sensory cortex or its immediate subcortical connections, and a homonymous hemianopic visual field defect results only from direct involvement of the contralateral visual pathway.

Disturbances in speech are of two types: those which are related to expression and those related to reception, though rarely if at all, does one type exist without some degree of the other. Aphasia of any degree or type indicates that the tumor lies above the tentorium in the temporal lobe or in an immediately adjacent region. The early manifestations of aphasia are easily mistaken for psychotic changes, but aphasia is a defect in language and allied functions and is not related to mood or behavior.

Convulsions, when associated with intracranial tumors, indicate that the tumor lies above the tentorium: no fits result from tumors in the posterior fossa, with the possible exception of their rare occurrence in a patient with a tendency to convulsions who develops increased intracranial pressure from a posterior fossa tumor. Patients with posterior fossa tumors may develop episodes of rigid hyperextension, sometimes spoken of as cerebellar fits, but which are really manifestations of a decerebrate state and have a serious implication. Fits, usually with focal qualities and more often diurnal than nocturnal, are a fair accompaniment on accom-

partment of cerebral tumors. They occur in about 45 per cent of all patients with supratentorial tumors, but particularly in those with the more benign types of glioma (about 90 per cent with oligodendrogliomas) or with meningiomas with which there is an incidence of fits of about 65 per cent. Convulsions may be the only manifestation for months of a slowly growing tumor and an adult who develops them for the first time in life should be suspected of having a tumor.

Evaluation of visual symptoms and of fundoscopic, pupillary and extraocular signs is of very great importance. Reduction in visual acuity when accompanied by primary optic atrophy rather than by papilledema or its late effects, secondary atrophy is usually the result of involvement of the optic nerves or chiasm by tumor. Perimetry can be particularly helpful and although careful testing on a tangent screen is desirable, of ten much can be learned by confrontation testing with fingers in the initial examination. A defect in the visual field of one eye indicates involvement of the nerve on that side. Bitemporal field defects indicate a chiasmal lesion, chiasmal syndrome, while homonymous field defects, as mentioned heretofore, invariably indicate a lesion in the contralateral visual pathway somewhere between the chiasm and the calcarine area of the occipital lobe.

Diplopia with duplication of objects on a diagonal plane, results from palsy of the third or fourth cranial nerve or of a lesion in the tegmentum in the midbrain. This is in contradistinction to diplopia with horizontal duplication due to abducens palsy. Third cranial nerve palsy should be evidenced by an enlarged pupil and drooping eyelid on one side if not also by discernible defects in movements of the involved eye except in lateral deviation. An enlarged pupil due to incomplete oculomotor nerve palsy occurs particularly on the side of rapidly developing tumors of the temporal lobe, causing shift of the brain stem and compression of the nerve.

Impairment in upward gaze results from a lesion of the quadrigeminal plate or adjacent regions of the midbrain and is nearly pathognomonic of a tumor in this region. Defects in horizontal movements of the eyes, with preservation of vertical movements, result from a lesion slightly lower in the brain stem and are seen almost exclusively accompanying gliomas of the brain stem. Nyctagnus does not result from a supratento-

rial tumor but is primarily the result of a disturbance in the vestibular system and linked with tumors in the posterior cranial fossa.

TUMOR SYNDROMES

The foregoing discussions may be summarized in brief descriptions of some of the syndromes based on the pathologic characteristics of individual tumors and the symptoms and signs which they are likely to produce because of their predilection for certain locations in the brain.

Tumors of the Cerebral Hemispheres. The commonest tumors of the cerebral hemispheres are gliomas and unfortunately glioblastoma which thus far has been incurable outnumbers all others. Astrocytoma, which is a more benign growth and sometimes curable occurs about half as frequently while oligodendroglioma, the rarest of these three is the most slowly growing and most benign. All these tumors occur with about equal frequency in the several lobes of the hemispheres with the exception of the occipital, where there is a relatively low incidence of all types of tumor.

Meningiomas which affect the brain by compression and not by invasion occur principally above the tentorium and with about one-third the frequency of gliomas. They are regarded as usually benign, encapsulated and surgically removable, though their location, size and vascularity sometimes may defeat attempts at radical removal. They tend to originate from regions where arachnoid villi are clustered and thus are found along the sagittal sinus, "parasagittal" and "falcine" tumors, and along the course of meningeal vessels, where they compress the superior surface of the hemisphere and are sometimes referred to as convexity tumors. Other common locations are along the sphenoid ridges and the floor of the frontal fossae, particularly in the olfactory grooves, and on the tuberculum sellae. Those on the sphenoid ridge affect the temporal lobe and adjacent portions of the frontal and parietal lobes. Those in the olfactory groove encroach on the inferior surfaces of one or both frontal lobes and destroy the olfactory and optic nerves. The syndrome described by Kennedy of anosmia and visual loss on one side, with homolateral primary optic atrophy and contralateral papilledema, is pathognomonic of the olfactory groove meningioma. Tumors of the tuberculum sellae may cause compression of the optic nerves and chiasm in the early period of their

growth and can be detected by the pattern of visual changes, though sometimes they attain great size and displace the frontal lobes before vision is affected.

Frontal lobe tumors which invade or compress the brain cause subtle changes at first and later more pronounced alteration in personality, loss of interest, irritability, impaired judgment and memory and an attitude of facetiousness. Forced grasping can often be demonstrated and if the lesion encroaches on the prerolandic area, there may be convulsions or progressive corticospinal tract signs. Lesions on the left side may also cause aphasia, particularly of the anomia type.

Parietal lobe tumors cause striking motor and sensory changes on the contralateral side of the body. *Jacksonian convulsions* are a frequent manifestation, particularly in the early phase of the disease, while in later stages there is increasing hemiparesis. Involvement of the sensory cortex results in contralateral astereognosis and loss of discriminatory sense and lessens the degree of spasticity in hemiparesis. On the dominant side parietal lobe lesions also produce aphasic defects, especially of the receptive type.

Temporal lobe tumors may be relatively "silent" and attain considerable size before detection, particularly if they occur on the nondominant side, but when they are on the dominant side aphasia is to be expected. Deep lesions involve a part of the optic radiation, sometimes causing visual hallucinations of bizarre forms and usually causing contralateral homonymous upper quadrant anopsia of incongruous type. *Uncinate fits*, feelings of *déjà vu* and psychomotor seizures are inconsistent effects of temporal lobe lesions, but, when present, they are diagnostically important.

Occipital lobe tumors in particular cause visual hallucinations of light and homonymous hemianopsia of congruous type, often without other localizing signs. If the lesion involves the lateral convexity of the lobe on the dominant side, there may be difficulty in reading ability (alexia) and inability to recognize objects by sight (visual agnosia).

Tumors of the Cerebellum. The common brain tumor of childhood is the benign cerebellar astrocytoma which has its peak in age distribution at twelve years and is rarely seen in persons older than twenty years. The results of surgical treatment are excellent, which makes it all the more desirable to reach a diagnosis before irreversible changes occur. The symptoms are those of gradual

increase in intracranial pressure accompanied by ataxic movements, dysmetria and hypotonia. Nystagmus is an inconstant finding. Particularly in younger children, the significance of unexplained vomiting, the development of a squint from abducens palsy and the moderate enlargement of the head due to gradual growth of the tumor may go unrecognized for months.

The next most common tumor of childhood is the malignant cerebellar medulloblastoma. It produces much the same pressure effects and cerebellar disturbances as does astrocytoma, but the development of symptoms is more rapid and the peak age of occurrence is several years earlier. Even so, it is usually impossible to be sure of the differential diagnosis without operation. The medulloblastoma, as with few other primary intracranial tumors, tends to spread along the cerebrospinal fluid pathways and occasionally the tumor will give evidence of its presence in the lower spinal canal before cerebellar signs develop. The tumor is highly sensitive to x-ray treatment, sometimes for a number of years, but radiotherapy should not be employed without first identifying the tumor and performing surgical decompression.

Other tumors of the cerebellum of less frequency though not rare, are *ependymomas*, the *papillomas of the choroid plexus* and *hemangioblastomas*. These may occur at any age, but the first two are more common in early life while the last is the benign cerebellar tumor of adults. A differential diagnosis on the basis of neurologic signs is not usually possible, though there are a few special features which are helpful. Ependymoma and papilloma tend to become firmly attached to the floor of the fourth ventricle and at times produce facial palsy as a result of invasion of one or both facial nuclei. Because of this firm attachment, it is usually impossible to perform complete surgical excision. Ependymoma, like medulloblastoma, may seed itself along the fluid pathways and this tumor also has a tendency to bleed spontaneously causing the symptoms of acute subarachnoid hemorrhage. Hemangioblastoma which has a high rate of surgical cure, may produce any or all of the signs of a cerebellar tumor. But more especially it should be recognized that this tumor may be accompanied by true polycythemia, is sometimes familial and may be associated with hemangiomatosis elsewhere in the body most particularly in the retina, in which case the combination of

retinal and cerebellar tumor is referred to as von Hippel Lindau's disease.

Tumors of the Brain Stem Tumors of the brain stem are gliomas, usually relatively benign such as *spongioblastoma polare* or *astrocytoma* but because of their location are not amenable to surgery and can be controlled only for a while with radiotherapy. The pons is much more frequently involved than the medulla and the age of incidence is principally in childhood occasionally in young adulthood.

The characteristic of these tumors is that they produce cranial nerve palsies usually for some time before long tract signs or increased intracranial pressure appears. It is remarkable that in many cases the aqueduct and fourth ventricle though narrowed are not occluded, and papilledema never develops. Among the various cranial nerve palsies commonly seen are extraocular palsies, vomiting, dysphagia, dysphonia and facial monoplegia or diplegia but other cranial nerve palsies may occur. Nystagmus and ataxia in the extremities are also common.

Tumors of the Third Ventricle Neoplasms of the third ventricle for practical purposes, can be divided into anteriorly and posteriorly placed lesions. The *pinealoma* typifies the latter and characteristically produces increased intracranial pressure by blockade of the ventricle or aqueduct, impairment in conjugate movement of the eyes above the horizontal plane and a variety of pupillary reactions. These tumors are not suited to surgical removal, but some type of ventricular shunt is useful to overcome the ventricular block and some of the several types of tumor which occur in or about the pineal gland can be controlled by radiotherapy.

The *colloid cyst* a benign and curable tumor occurs in the anterior third ventricle and is so located that it blocks the foramina of Monro thus causing dilatation of the lateral ventricles and increased intracranial pressure. Except for symptoms and signs resulting from the altered pressure, there are no other manifestations. Other tumors, such as gliomas, cranio-pharyngioma and ectopic pinealoma, sometimes encroach on the anterior third ventricle and are likely to cause hypothalamic disturbances characterized by diabetes insipidus, diencephalic epilepsy, somnolence, obesity and precocious sexual development.

Tumors of the Cerebellopontile Angle. The large group produced by tumors in the

cerebellopontile angle deserves special mention because its recognition can be relied on to identify the presence of the *acoustic neuroma* which is surgically curable. Other tumors which may occur in this region are meningioma and cholesteatoma the latter being particularly favorable to treatment.

Symptoms of acoustic neurinoma develop insidiously beginning usually with unilateral tinnitus and progressive deafness. The vestibular response on the same side disappears early in the disease and there may be vertigo and instability in walking, but paroxysms of vertigo as occur in Meniere's disease are not encountered. After many months, involvement of the adjacent trigeminal and facial nerves becomes evident the former being particularly identified by loss of corneal sensation even before other sensory loss in the face develops. Additional growth of the tumor may cause palsy of additional cranial nerves in the posterior fossa and also ventricular block, though the presence of the tumor can usually be recognized before it has advanced to this stage.

Tumors of the Sellar Region Tumors of the sellar region can be separated into intrasellar tumors which are almost exclusively *pituitary adenomas* and suprasellar tumors, which include in order of frequency *cranio-pharyngioma*, *meningioma* of the tuberculum sellae and *gliomas* of the optic chiasm.

The *tumors of the pituitary* occur in early adulthood and arise from one of three types of cells which make up the anterior lobe of the gland. The commonest is the *chromophobe adenoma* derived from the indifferent cells. It produces hypopituitarism as a result of compression of the normal gland, enlarges ("balloons") the sella turcica and compresses the optic nerves and chiasm causing visual field defects, usually bitemporal hemianopsia, loss of visual acuity and primary optic atrophy. The growth of these tumors may be held in check by x-ray therapy but if there is any significant impairment of vision, surgical removal of the tumor should not be delayed for it has a high degree of success.

The *chromophil adenoma* is derived from the eosinophil cells which are known to control growth. The presence of the tumor can be easily recognized by the appearance of gigantism during puberty or of acromegaly after puberty. A relatively small percentage of these tumors enlarge enough to compress the optic nerves and chiasm.

Since the tumors are relatively sensitive to x-ray this has usually been the treatment of choice. But since x-ray therapy cannot be

relied on always to control the tumor and the subtle persistent bodily changes it produces surgical excision has come to be employed more often. The risk in the past of destroying normal pituitary functions by surgical removal of the tumor is no longer a hazard since the advent of ACTH and cortisone for substitution therapy.

The *basophil adenoma*, derived from basophil cells, is sometimes believed to be the cause of Cushing's syndrome. The tumor does not attain any significant size and thus far has been regarded as not suited to surgical removal attention centering more particularly on the target gland the adrenal.

In the suprasellar region, the *craniopharyngioma* is the commonest of the several tumors that may be found and may be difficult to differentiate from pituitary adenomas whose effects it mimics. However *craniopharyngioma* is a congenital tumor and often produces symptoms before the patient is fifteen years of age. It has a tendency to cause diabetes insipidus, sometimes for transient or intermittent periods, and to show calcification on roentgenograms findings which are unusual in pituitary adenomas. Meningiomas produce local hyperostosis of the tuberculum sellae and gliomas of the optic chiasm cause subclinoïd erosion, sometimes with enlargement of the optic foramina, which can be identified on roentgenograms.

Metastatic Tumors. The most common source of metastatic tumor of the brain is the lung but malignant tumors of the breast, kidney, gastrointestinal tract, nasopharynx and of many other tissues of the body may give rise to secondary growths in the brain. Metastases are more commonly found in the cerebral hemispheres and, in a surprising number of cases, there appears to be only one lesion in the brain. The progression of neurologic symptoms and signs is likely to be fairly rapid and may constitute the only complaints or discernible manifestation of the malignancy. The best that can be expected from surgical removal, which is often a simple operation, is prolongation of life and possibly some reversal of palsies and relief of headache. But to many patients and their families this much is very much worth while.

DIAGNOSIS

It would be a serious mistake to assume that the diagnosis of tumors and other diseases of the brain could do without diligent clinical study since the combination of an

accurate history and thorough physical examination remains our most reliable method. Yet without the aid of numerous present day adjuncts, the factor of error in identification and localization would be considerable.

Roentgenograms of the skull yield some type of positive information in about 30 per cent of brain tumors. The changes to be looked for are (1) erosion of the sella turcica, (2) shift in position of the calcified pineal gland (3) calcific deposits in the neoplasm (4) alteration in vascular markings of the skull (5) enlargement of certain foramina, (6) separation of cranial sutures in children, (7) thinning or destruction of the skull in adults and hyperostotic changes caused by meningiomas.

A roentgenogram of the chest should be obtained in every patient suspected of having an intracranial tumor because of the possibility of metastasis from lung cancer.

Lumbar puncture and withdrawal of spinal fluid cannot be performed without risk in the presence of increased intracranial pressure though occasionally the risk is justified. The possibility that symptoms of brain tumor may become rapidly worse following withdrawal of spinal fluid emphasizes the need for omitting lumbar puncture or restricting its use to a time when craniotomy can be performed promptly if need be. Compression of jugular veins sometimes erroneously performed in conjunction with the puncture adds materially to the risk.

In addition to determination of the resting pressure of the cerebrospinal fluid, the other important determinations are the protein content of the fluid and the cell count. The protein content in the fluid is elevated in the majority of but not in all, patients with brain tumor. The most consistent elevations occur with acoustic neuromas and meningiomas which exist near the floor of the skull. Occasionally glioblastomas which encroach on the ventricle or metastatic tumors will produce pleocytosis in the fluid. In some cases it is possible to identify the presence of neoplastic cells in centrifuged cerebrospinal fluid. In cases of extensive involvement of the meninges by tumor there may be significant decrease in the sugar content of the fluid. Recent studies of the transaminase content of cerebrospinal fluid suggest that some information is provided in differentiating vascular and neoplastic diseases of the brain, transaminase is usually increased in the former and less often altered by tumors.

Electroencephalography is found to be

useful in identifying the presence and location of a brain tumor in from 50 to 75 per cent of cases, depending on the experience and diligence of the examiner. In particular infratentorial and deep supratentorial tumors evade detection and often an abnormal

pattern due to vascular disease cannot be distinguished from that of a neoplasm. Even so, the electroencephalogram has value as an adjunct method of examination.

Radioactive isotopes have come to be employed in the identification of brain tumors

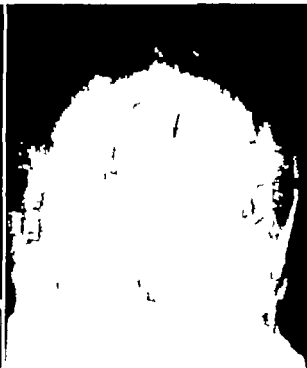


Figure 16 Angiogram showing "staining" in a large glioma of the frontal lobe and distortion of regional arteries.

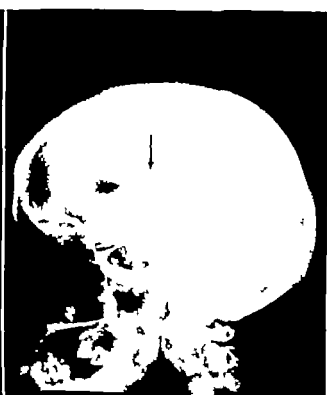


Figure 17 Ventriculogram showing depression of the body of the ventricle and a lateral shift of the ventricular system caused by a parietal tumor.

based on the premise that the presence of the isotope deposited temporarily in greater concentration in the tumor can be detected by a suitable counting device. The tests made with gamma-emitting radioactive I^{131} and a scintillation counter have shown an efficiency of about 70 per cent in the detection of brain tumors above the tentorium. The results in detection of tumors by this method are at least as good as those obtained by electroencephalography. Improved results in diagnosis have been reported by the use of a positron-emitting isotope and automatic coincidence scanning as compared to single or multiple channel scanning utilizing gamma radiation.

Angiography (Fig. 16) performed by the injection of a radiopaque dye (usually 50 per cent Hypaque) into the carotid or vertebral arteries has added significantly to diagnostic procedures and is attended by very little risk. The test is of less use in posterior fossa tumors than in supratentorial tumors where it can be of considerable advantage in identifying the presence, relative size and vascularity of the lesions and has about 80 per cent accuracy. With somewhat less accuracy, the type of tumor can sometimes be determined. For the diagnosis of vascular lesions such as aneurysms and arteriovenous anomalies, the method is ideal.

Ventriculography (Fig. 17) and **encephalography** by air injection into the ventricles or spinal subarachnoid space are still the most reliable methods of identifying the presence and the location of a tumor in any part of the brain. The efficiency of the tests is about 95 per cent. The latter method requires lumbar puncture and entails its attending risk in the presence of increased intracranial pressure. But if the air is injected without also withdrawing cerebrospinal fluid, the procedure may be used with relative safety particularly if preparations have been made to proceed immediately with removal of the tumor or at least for withdrawal of ventricular fluid through a trephine opening in the skull in case the patient's condition should be made worse.

DIFFERENTIAL DIAGNOSIS

In the diagnosis of a tumor there are many other diseases of the brain which require differentiation. Some of these are characterized by increased intracranial pressure alone, such as atresia of the aqueduct of Sylvius, or pseudotumor cerebri. Some present convulsions or various palsies or both, such as may occur with a variety of degenerative and cerebral vascular diseases.

Still others demonstrate increased pressure as well as various other neurologic signs. Included among these conditions are abscess, parasitic and granulomatous infections and several kinds of intracranial hemorrhage.

Most of the diseases which require differentiation can be identified by relatively simple diagnostic measures, whereas a few require time and repeated observations. Some of the diseases though not neoplastic are amenable to surgical treatment.

TREATMENT OF INTRACRANIAL TUMORS

Surgical Treatment. If it were possible, all brain tumors should be surgically removed, but many are confined to vital parts or have extended to these parts by the time of operation and defeat any hope of cure by excision. However, even partial removal of most tumors of this kind is repaying in terms of improvement in symptoms and extension of life. Certain tumors which constitute about 35 per cent of the total number can be totally removed, or nearly so, and the patients restored to useful life. The tumors included in this last group are meningiomas, pituitary adenomas, acoustic neuromas, hemangiomas, astrocytomas (particularly cerebellar), cranio-pharyngiomas, cholesterolomas and a miscellaneous group of rarer neoplasms.

The two great adjuncts to neurosurgical technique, strong suction and an electro-surgical unit for control of bleeding, have been used to advantage for over twenty-five years, but many refinements have been added. Considerable thought has been given to the cosmetic result of cranial operations and an unsightly wound is an exception.

The Torkildsen operation of ventricular shunt by means of a tube passing from a lateral ventricle to the cisterna magna has been highly useful in certain inremovable tumors such as the pineuloma.

In many clinics local anesthetics, the use of which was tedious for both the surgeon and the patient, have been replaced by Pentothal administered intravenously or ether given endotracheally. There is a distinct advantage in the protection against respiratory difficulty during operation provided by the endotracheal tube and operations can be continued for hours without fear of difficulty from this source.

The ready availability of blood for transfusion has simplified the correction of blood loss and has virtually done away with the need for multistage operations that formerly were used in the treatment of some tumors.

Though muscle is

best ma-

terial for use in the control of more brisk bleeding from torn blood vessels in the brain or dura, Gelfoam and absorbable cellulose provide ready means of dealing with the general ooze of blood that can be such a nuisance to control.

The advent of antimicrobial agents has been a godsend in neurosurgery for the risk of infection and death from meningitis is now virtually a thing of the past. In this regard it can be added that a tuberculoma can now be removed without the almost inevitable sequence of meningitis and death thanks to the advent of streptomycin and other antituberculous drugs.

Three recent developments give promise of adding significantly to the facility and safety of operations. One of these is the production and maintenance of a hypotensive state during operation by means of a sympathetic blocking agent (Arfonad) which minimizes blood loss, reduces the bulk of the brain, has a complementary effect on anesthesia and yet does not expose the brain to anoxic effects which often occur when blood pressure falls because of blood loss.

A more recent development is the lowering of body temperature to a degree that reduces the metabolic requirements of the brain, thus making it possible to occlude the main blood supply to the brain safely for minutes at a time while dealing with a vascular lesion.

Another recent development has been the use of urea, usually administered intravenously for reducing intracranial pressure through "shrinking" of the brain. It appears that this agent has advantages which will lead to its replacing all other chemical agents used heretofore for this purpose.

Radiation Therapy There is little place for radiation therapy as a substitute for surgery in tumors of the brain. Gliomas of the brain stem cannot be excised and sometimes are dramatically improved for limited periods by radiation therapy. Pituitary adenomas which have not grown to a size to compress the optic nerves may benefit by x-radiation. Pinealomas and tumors not well suited to direct surgical attack will often show favorable effects from treatment.

As a supplement to partial removal of certain tumors, x-ray treatment is beneficial, but the results are difficult to evaluate and there is a wide diversity of opinion regarding its use. Some would limit its use postoperatively to a few tumors, such as medulloblastoma and pituitary adenomas but, at one time or another claim of benefit from x-ray

treatment has been made for nearly every type of intracranial neoplasm. Some of these claims are unjustified but others undoubtedly represent the occasional observation of benefit and thereby emphasize the vagaries in the response of tumors to irradiation. It is sufficient to say that no brain tumor has ever been cured by x-radiation and the decision to employ treatment is usually dependent on a number of factors pertinent to the individual case.

Recently there has been renewed interest in the use of radiation therapy since the development of the high voltage machine (betatron rays) radioactive cobalt and the activation of boron injected intravenously by exposing the patient in the atomic pile. None of these methods of radiation therapy have shown results thus far which justify enthusiasm.

PSEUDOTUMOR AND ALLIED CONDITIONS

Pseudotumor is a term applied to a condition in which increased intracranial pressure develops without discoverable cause. The condition has also been labeled serous meningitis, otitic hydrocephalus, toxic hydrocephalus and arachnoiditis.

Clinically most of the subjects have in common headache, papilledema, increased intracranial pressure by spinal measurement and occasionally diplopia, transient palsies and convulsions. The patient's feeling of well being is out of keeping with the potentialities implied by the increased pressure. But for elevation of pressure the spinal fluid is normal, the electroencephalogram at times shows an abnormal pattern of nonspecific nature and the pneumoventriculogram, most important of all tests, shows a normal or perhaps small-sized ventricular system.

Some of the patients have been found by sagittal sinus venography to have unsuspected thrombosis of the sagittal sinus or of a dominant lateral sinus. The condition is believed to have been present in many of the disorders designated as otitic hydrocephalus associated with mastoiditis and middle ear infections.

In a study of twenty-eight consecutive cases followed for one to five years, it was found that in six of the patients the nature of the illness was not evident until some months had passed but within ten months it was determined that three patients had neoplasms, one had multiple sclerosis another had periarteritis and one had an aneurysm of the basilar artery.

In ten of the twenty-eight patients venous

obstruction was conclusively demonstrated as the cause of the increased intracranial pressure. In the remaining twelve, no cause could be discovered. The fortunate thing is that most patients with venous obstruction or without any demonstrable disease tend to recover in a period of several months without visual loss or other sequelae. A few begin to lose vision and then it is desirable to resort to decompressive procedures, such as unilateral or bilateral subtemporal decompression. Repeated lumbar puncture is safe and may serve a useful purpose in diminishing the risk of visual loss.

In *arachnoiditis* there is unexplained thickening of the arachnoid membrane resulting in obstruction to the cerebrospinal fluid pathways. The effect is at times localized about the chiasmatic cistern causing visual loss and there are reports of dramatic return of vision after resection of the arachnoid and release of trapped fluid. When *arachnoiditis* occurs in the cisterna magna and lateral recesses of the posterior fossa there is ventricular obstruction and increased intracranial pressure. The condition is distinguished from pseudotumor by the fact that the ventricles tend to become dilated in *arachnoiditis*. Exposure of the cisterna magna by craniotomy and disruption of the arachnoid, together with suboccipital decompression will usually terminate the increased intracranial pressure.

INTRACRANIAL ABSCESES (BRAIN ABSCESES)

Localized suppurative infection of the brain, once a relatively common and fear some disease, has nearly become a rarity principally because of the advent of anti microbial therapy. Brain abscesses result from extension of infection in adjacent regions of the head or by metastasis from remote foci in the body. Adjacent sources include middle ear and mastoid, the nose and accessory nasal sinuses, the cranium and scalp and also compound fractures and penetrating wounds of the skull through which there is implantation of infected material. Of this group middle ear infection is by far the commonest source in civilian practice. Metastatic abscesses originate principally from lesions in the chest and less frequently from endocarditis, osteomyelitis, abscessed teeth, carbuncles, septic endometritis and suppuration in any part of the body. Occasionally the source of a brain abscess cannot be determined, in which case it is as

sumed that the infection started in a period of unrecognized bacteremia.

The abscesses which result from direct extension of an adjacent infection are usually solitary while metastatic abscesses are often multiple. The infecting organisms most frequently found are *Staphylococcus pyogenes* var. *albus* and *aureus*, the hemolytic streptococci and the pneumococci. Other organisms as well as mixtures have also been found, their nature depending on the source of the contamination.

Pathology. Extension of infection to the brain from adjacent foci in the head may occur by one of two pathways or possibly at times by a combination of the two. The commonest is the successive infection of intervening structures in which osteomyelitis or localized osteitis first develops, followed by the formation of an epidural abscess which penetrates the dura and invades the underlying brain, passing through the highly vascular cortex to lodge and take growth in the white matter. In the course of progression of the infection, the subdural or subarachnoid spaces may become contaminated, giving rise to a subdural abscess and leptomeningitis respectively. A part of this process of extension undoubtedly entails thrombosis of local veins and arteries in the bone, meninges and brain and this serves to extend the infection as well as to make the tissues more susceptible by devitalization. Brain abscesses arising from frontal sinusitis thus come to occupy chiefly the frontal lobes, while those arising from the middle ear and mastoid occupy the temporal lobe or the cerebellum.

The other common mode of extension is by way of the veins draining the structures about the upper portion of the face and head toward the dural venous sinuses. By this means retrograde thrombosis or septic emboli may carry the infection through tributary veins to any part of the brain, thus accounting for what have been called "distant abscesses." Occasionally abscesses have resulted from implantation by an exploring brain needle which has passed through an infected area. The location of a metastatic abscess from a distant source of infection is purely fortuitous and in about two-thirds of the cases more than one abscess develops. When an abscess results from a penetrating wound, the inciting factors are pulped brain and blood clots, which provide a splendid medium for the growth of bacteria, plus retained bone fragments and foreign material.

which have carried in bacteria that are largely skin inhabitants of relatively low virulence. Shell fragments are not usually infective even when they are retained in the brain.

Irrespective however of what routes the infection takes to the brain the local reaction which follows in the brain is always essentially the same. As the infection grows in the devitalized tissues the clinical symptoms are those of encephalitis. After some days an equilibrium is reached between the infection and the reaction of the surrounding brain. Regional blood vessels throw off a heavy exudation of leukocytes constituting the first line of defense. Behind this wall the blood vessels increase in size and number and connective tissue cells proliferate producing a wall, or capsule for the abscess. From this time on, it is a struggle between the wall and the leukocytes on the one hand and the invading bacteria on the other. If a break through occurs there may result a secondary daughter abscess, spreading encephalitis, or rupture into the ventricles, the last two conditions being rapidly fatal.

While the wall of an abscess begins to form in the first week, it is not firm enough to offer appreciable resistance to an exploratory needle until at least two or three weeks have passed. In some types of infection of strong virulence particularly in metastatic abscess of pulmonic origin a wall may not develop and the clinical course is one of fatal, spreading encephalitis from the start.

If surgical evacuation of the abscess is instituted, the wall of the abscess will contract and form a scar. Rarely an otogenic abscess has been known to discharge spontaneously into the external ear or mastoid wound. Occasionally spontaneous cures take place and some abscesses when drained are found to contain sterile pus. But in spite of apparent quiescence reactivation of even a drained or sterile abscess may take place long after the process has been thought to have subsided.

Symptoms and Signs. The symptoms of a brain abscess vary with the stages of the disease, the virulence of the infection, the degree of increased intracranial pressure and the location of the abscess. Early in the clinical course it may be difficult to make an accurate diagnosis, even though it may be obvious that infection has reached the brain. Usually the temperature remains high for the first few days and then takes on a low spiking contour. The blood leukocyte count may rise to 20,000 or more, but it too may

decline and rarely the count is normal from the start or soon afterward. Examination of the spinal fluid discloses an increase of pressure, protein content and leukocytes chiefly of lymphocytes unless frank suppurative leptomeningitis occurs.

Headache and vomiting develop early and persist. Irritability or apathy and lethargy reflect the development of encephalitis and increasing intracranial pressure. The neck becomes stiff as a result of the associated meningeal reaction and in time, as the pressure increases, the pulse rate at first fast, becomes slow and papilledema appears. These are the general symptoms and signs of the initial stage of the disease representing the struggle between destructive and reparative forces. If the virulence of the infecting organism is great and the patient's resistance is poor surgical drainage of the abscess may be useless and profound coma followed by respiratory failure may ensue.

In more favorable cases the infection becomes localized and a wall develops around the abscess. Signs of sepsis subside and fever and leukocytosis recede. Edema of the brain abates and all symptoms improve, with the possible exception of localizing symptoms, which will usually have made their appearance some time during the first week of the illness. The patient still appears apathetic and complains of headache so that the clinical picture at this stage may be much like that of an intracranial neoplasm.

The symptoms may continue for months throughout the quiescent stage until the abscess is evacuated or the patient dies from rupture of the abscess into the ventricle or as may rarely happen the abscess resolves spontaneously.

Localizing signs depend upon the size and position of the abscess. The commonest sites for abscesses in the brain are in the order of frequency, the frontal lobes, the temporal lobes and the cerebellar hemispheres. Less commonly the parietal and occipital lobes and the brain stem are involved. Abscesses in the *frontal lobe* may attain considerable size and still remain relatively silent, yet it is unusual if there are not at least a few suggestive signs, such as alteration in mood, contralateral convulsions, muscle weakness or pathologic reflexes and possibly motor aphasia if the lesion is on the left. *Temporal lobe* abscesses give more striking signs of their presence, namely homonymous hemianopic defects, paralysis, sensory impairment and pathologic reflexes on the contralateral side and palsy of the third and sixth

cranial nerves on the homolateral side. If the left temporal lobe is involved, aphasia is the earliest and most striking sign. Occasionally the acute displacement of the brain stem caused by a temporal lobe abscess may cause homolateral corticospinal tract signs due to pressure on the crus cerebri by the margin of the tentorium on the side opposite the abscess. Cerebellar abscesses produce the usual signs of cerebellar dysfunction, namely dysmetria in the homolateral extremities, nystagmus and frequently pathologic reflexes on the opposite side of the body. It has occasionally been observed that the patient prefers to lie on the side of the lesion and that turning the head to the opposite side induces vertigo, vomiting and vigorous nystagmus. Another sign of apparently reliable localizing value which has been observed in the occasional case of cerebellar abscess, is strong conjugate deviation of the eyes toward the opposite side from the lesion. This is in contradistinction to the occasional occurrence of deviation of the head and eyes toward the side of the lesion in abscesses of the frontal or parietal areas.

Diagnosis. In the initial stage, it may be difficult to differentiate a brain abscess from purulent pachymeningitis (subdural abscess) or leptomeningitis. The former is a much more fulminating infection than a brain abscess, though obviously difficulties arise if both exist, as may sometimes be the case. Leptomeningitis produces relatively more cellular changes in the spinal fluid and less striking localizing neurologic changes. Differentiation from a brain tumor when the signs of infection are minimal or are overlooked, may be difficult and most neurosurgeons have had the experience of accidentally coming upon an abscess when exploring for a tumor. Thrombosis of the lateral venous sinus in association with otitic infections may produce general symptoms like those of the early stages of an abscess, but not localizing neurologic signs. The Tobey-Ayer test of jugular compression to determine lateral sinus thrombosis is potentially dangerous, because jugular compression during the performance of a lumbar puncture in the presence of a brain abscess may lead to medullary compression and rapid respiratory failure.

Electroencephalography, angiography and ventriculography each has value in diagnosis, though often there is enough evidence of the presence and location of an abscess so that these adjuncts are not required.

Treatment. The treatment of a brain ab-

scess except possibly when the brain stem is involved, is always surgical drainage. The history of the surgical treatment of brain abscesses is replete with a variety of methods and even now there is lack of unanimity of opinion regarding the preferable method of evacuation of certain types of abscess. In the early stages of formation of a brain abscess, hasty operations are usually futile. At this time, reliance must be placed on supportive measures and antibacterial therapy. The opportune time for operation is after the abscess has become walled off at least two weeks after the onset and before the patient's condition becomes critical or before irreparable damage to important brain centers has occurred.

Although complete extirpation of the intact abscess wall or evacuation of the abscess plus removal of its wall is the ideal treatment, it is not well suited to abscesses of the cerebellum, those deeply situated in the cerebrum or those occupying the Rolandic and speech areas. For abscesses located in these latter areas, simple drainage or repeated tapping, accompanied by local and systemic antimicrobial therapy, often proves to be effective treatment.

EXTRADURAL ABSCESS (PACHYMENINGITIS EXTERNA)

Localized suppurative infections between the dura and the cranium are usually the result of otitis media and mastoiditis, frontal sinusitis or osteomyelitis of the skull. The infection may travel by continuity or by continuity via blood vessels, chiefly veins which drain toward the dural venous sinuses.

The degree of development of the inflammatory process varies between the formation of circumscribed granulation tissue and a thin deposit of fibrin, and the formation of a local collection of thick pus. Extensive lateral spread of the abscess is prevented by adhesion of the dura to the skull, and it is rare for an uncomplicated extradural abscess to assume proportions sufficient to cause intracranial pressure. Its importance lies wholly in the necessity for early recognition and drainage before the infection spreads to deeper structures. Occasionally infection in the petrous tip may result in an extradural abscess and Gradenigo's syndrome, which is characterized by symptoms of irritation of the trigeminal and abducent nerves, namely diplopia and pain in the eye on the side of the lesion.

Early surgical drainage and sequestrectomy are usually followed by prompt re-

covery. Complications arise only when there is inadequate debridement and drainage.

SUBDURAL ABSCESS OR EMPYEMA (PACHYMENINGITIS INTERNA)

Etiology. A subdural abscess, or empyema, is the result of extension of infection from an adjacent focus principally the middle ear, mastoid or the frontal sinus. It may rarely follow maxillary sinusitis, septic thrombosis of the cavernous sinus, compound wounds of the skull and evacuation of a brain abscess.

Two possible pathways of infection exist. In one type the infection advances from osteitis to an extradural abscess and thence through the necrotizing dura directly to the subdural space. In the other there is retrograde extension along venous channels leading from the infected mucosa. By this means, infection arising in the frontal sinus may invade the superior longitudinal venous sinus, while in cases of otitis media and mastoiditis the infection spreads to the lateral venous sinus. From the venous sinuses, the retrograde septic thrombosis involves the cerebral veins and the infection thus comes to lodge in the subdural space which these veins traverse.

The purulent exudate which collects in the subdural space has little tendency to localize and often the entire space over one or both cerebral hemispheres will contain 100 cc. or more of pus. Unfortunately after surgical drainage there is a tendency for isolated collections to become walled off and surrounded by a neomembrane. The remarkable property of the arachnoid to impede the spread of infection to the subarachnoid space accounts for the existence of little more than a mild leptomeningeal reaction and low-grade pleocytosis of the spinal fluid until the last stage of the disease. Thrombosis of the cerebral veins leads to edema of the brain and not infrequently to the development of large and small abscesses in the brain.

Symptoms. The symptoms of a subdural abscess at first are not readily distinguishable from the symptoms of acute infection of the frontal sinus or ear. But increasing fever, malaise and headache usually frontal and on the side of the lesion, should give early warning of the possible existence of a subdural abscess. After a variable time, not exceeding a few days, drowsiness increasing to stupor and coma supervenes. At about the same time, focal neurologic signs develop, the most significant being jacksonian con-

vulsions, hemiplegia and if the lesion is left sided aphasia. The profound illness of the patient and neurologic signs distinguish the condition from an extradural abscess. A cerebral abscess can usually be distinguished by its milder initial stages, the more insidious onset of neurologic signs and less febrile reaction.

Treatment. In the treatment of subdural abscess, it must be kept in mind that usually this is a fulminating disease which rapidly terminates in death if the patient is not treated. With the earliest sign or suspicion of the disease exploratory trephines should be performed and if pus is encountered it is drained through this and additional small openings. The space should be treated with an initial instillation of penicillin. Drains are usually useless since the space tends to close rapidly and reliance must be placed thereafter on obliteration of the source of infection and systemic treatment with antimicrobial therapy.

VASCULAR LESIONS OF THE BRAIN

The vascular lesions of the brain include aneurysms of the major cerebral arteries, congenital arteriovenous anomalies and carotid artery-cavernous sinus fistulae.

Aneurysms. Aneurysms of the circle of Willis and its main branches are believed to be principally congenital, the exceptions being occasional mycotic and arteriosclerotic dilatations. The congenital aneurysms represent remnants of embryonal vessels or weakness in the angle of juncture of two arteries resulting in a saccular outpouching. They rarely produce symptoms before adulthood at which time, however, they may rupture or attain sufficient size to cause pressure on adjacent structures. Although these lesions are common, interest in them has been greatly increased since the development of angiography and improved methods of surgical treatment. Experience has shown that the majority of cases of subarachnoid hemorrhage result from rupture of aneurysms and their importance is reflected in the 50 per cent mortality rate which occurs in untreated patients.

Rupture of an aneurysm is spontaneous and characterized as a rule by sudden severe headache, most intense in the frontal or occipital regions, and by vomiting, collapse and coma in many instances. Stiff neck nearly always ensues. There may be no localizing signs, but the commonest local effects resulting from pressure and irritation of adjacent structures are pain in the eye



Figure 18 Angiograms by carotid injection showing a saccular aneurysm of the internal carotid artery at its junction with the posterior communicating artery

diplopia, unilateral dilatation of the pupil ptosis of the lid and sometimes sensory impairment of the cornea and face. These are the result of involvement of the third and fifth cranial nerves. There may be other signs resulting from compression of the brain by collections of blood in the sylvian fissure or from subcortical hematomas which occur particularly in the temporal and frontal lobes. From the effects of blood in the cerebrospinal fluid spaces, patients may remain restless and confused for many days. There is often fever which sometimes confuses the diagnosis with meningitis, and usually there is increased intracranial pressure. Patients surviving the initial hemorrhage are subject to one or more recurrent hemorrhages in the first month and any one of these may be fatal.

The diagnosis is confirmed by lumbar puncture which reveals grossly bloody fluid. Xanthochromia develops in the spinal fluid within several hours after hemorrhage and persists for ten days or longer.

The precise location of the aneurysm cannot always be determined from the clinical features alone, but angiography (Fig 18) can be expected to identify the presence and location of an aneurysm in 60 per cent of patients having spontaneous subarachnoid hemorrhage. There are other possible sources of hemorrhage, such as from neoplasms and arterial rupture in vascular hypertension. But it is believed that often when an aneurysm is not visualized by angiography it is

too small to be recognized or has become thrombosed. Fortunately there seems to be less tendency to recurrent hemorrhages when aneurysms are not demonstrated by angiography.

The main problem pertaining to intracranial aneurysms is not so much their recognition, but the preferred manner of treatment. The most appropriate plan of procedure is to verify the subarachnoid hemorrhage immediately by lumbar puncture and follow this as the patient's general condition permits by angiography any theoretical risks attending these procedures are minimal compared to the advantages.

The demonstration of an aneurysm of the vertebral or basilar arteries, or of multiple aneurysms, usually precludes surgical treatment, but all aneurysms deserve consideration. If the angiogram discloses the presence of a hematoma, it should be evacuated promptly but it is preferable to delay direct surgical attack on an aneurysm for ten days or so, until cerebral edema has subsided. In the waiting period, there may be advantage to ligation of the carotids in the neck, particularly for aneurysms of the internal carotid artery and many times this comparatively safe procedure is all that is employed. Undoubtedly the most logical surgical approach is, as in aneurysms of vessels elsewhere to occlude the vessel on each side of the sac. This is not as easily accomplished, however in intracranial aneurysms.

The commonest place for intracranial

aneurysms to be found is on the internal carotid, more often in its supraclinoid than in its infraclinoid portion. The next commonest place is on the anterior cerebral arteries at their anterior communication.

The serious risks* entailed are related to unmanageable hemorrhage which may occur at the time of operation and the cerebral damage that may follow occlusion, or damage to one of the main arteries.

The employment of controlled hypotension and hypothermia has added significantly to the facility and safety of the direct intracranial attack on aneurysms but the final evaluation of the surgical treatment remains to be made.

Arteriovenous Anomalies. These lesions variously referred to as angiomas (Fig. 19) cirrroids, varices or aneurysms and malformations, are believed to be congenital and result from failure of development of the capillaries. In a small number of patients there are manifestations of the lesions in childhood, but more often they come to attention in adulthood particularly in the third and fourth decades of life.

The lesions are found most often to involve the middle cerebral artery but they have been observed in all parts of the brain and in sizes ranging from a small marble-sized nest of racemose vessels to the extensive involvement of half the cerebral hemisphere.

Convulsions are a common manifestation of the presence of the lesions in the cere-

brum and doubtless some patients believed to have idiopathic epilepsy have arteriovenous anomalies. In others, the lesion has been overlooked as a cause of retarded development on one side of the body which, instead is presumed to be due to birth palsy. Headache is sometimes a symptom but usually the lesion is brought to attention when a subarachnoid hemorrhage takes place and an angiogram demonstrates the unsuspected anomaly. A cephalic bruit can be detected in over half the patients but is not diagnostic since there are other causes for bruit. Recurrent hemorrhage into the subarachnoid space of the brain is common, but fatal hemorrhage is infrequent.

Treatment of the arteriovenous anomalies is a recent surgical accomplishment aided by exact delineation of the abnormal vessels by angiography the use of controlled hypotension during operation and more recently by hypothermia, which permits temporary occlusion of the carotids while the vessels are being isolated and ligated.

The principal indication for surgical removal of the lesions is spontaneous hemorrhage, but occasionally progressive palsies or uncontrolled convulsions will be also benefited.

Carotid Cavernous Fistula. The development of a fistula between the intracranial portion of the internal carotid artery and the cavernous sinus is usually the result of a basilar skull fracture, though occasionally it is seen following penetrating wounds or following spontaneous rupture of the carotid.

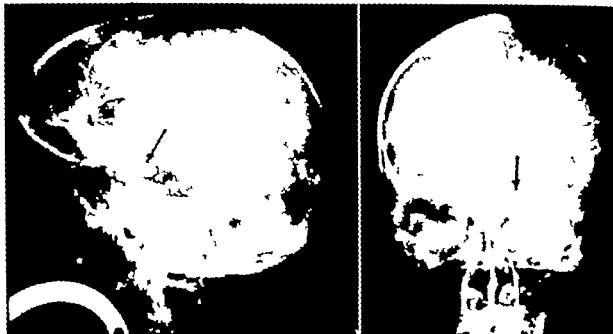
The classical symptoms and signs include unilateral pulsating exophthalmos, chemosis, edema of the lids and fullness of orbital and retinal vessels. The patient is aware of a bruit, may suffer from considerable pain about the eye and may lose motion and sight of the eye occasionally the other eye will become involved too.

Treatment initially is conservative since the fistula may close spontaneously. Thereafter if closure does not take place, the carotid artery in the neck should be ligated, usually in a two-stage procedure, or it should be occluded gradually over a period of days by the use of a specially devised clamp on the artery*. If this does not serve to result in closure of the fistula, the intracranial portion of the carotid just above the cavernous sinus should be ligated. It is important to know that double ligation of the carotid in

Editor's note: It has been shown that ligation of the common carotid artery is less hazardous when performed under local anesthesia.



Figure 19 Angiogram showing small arteriovenous anomaly ("angioma"). Note the veins (arrows) connecting the lesion to the sagittal and straight sinuses.



Figures 18 Angiograms by carotid injection showing a saccular aneurysm of the internal carotid artery at its junction with the posterior communicating artery

diplopia, unilateral dilatation of the pupil, ptosis of the lid and sometimes sensory impairment of the cornea and face. These are the result of involvement of the third and fifth cranial nerves. There may be other signs resulting from compression of the brain by collections of blood in the sylvian fissure or from subcortical hematomas which occur particularly in the temporal and frontal lobes. From the effects of blood in the cerebrospinal fluid spaces, patients may remain restless and confused for many days. There is often fever which sometimes confuses the diagnosis with meningitis, and usually there is increased intracranial pressure. Patients surviving the initial hemorrhage are subject to one or more recurrent hemorrhages in the first month and any one of these may be fatal.

The diagnosis is confirmed by lumbar puncture which reveals grossly bloody fluid. Xanthochromia develops in the spinal fluid within several hours after hemorrhage and persists for ten days or longer.

The precise location of the aneurysm can not always be determined from the clinical features alone, but angiography (Fig. 18) can be expected to identify the presence and location of an aneurysm in 60 per cent of patients having spontaneous subarachnoid hemorrhage. There are other possible sources of hemorrhage such as from neoplasms and arterial rupture in vascular hypertension. But it is believed that often when an aneurysm is not visualized by angiography it is

too small to be recognized or has become thrombosed. Fortunately there seems to be less tendency to recurrent hemorrhages when aneurysms are not demonstrated by angiography.

The main problem pertaining to intracranial aneurysms is not so much their recognition, but the preferred manner of treatment. The most appropriate plan of procedure is to verify the subarachnoid hemorrhage immediately by lumbar puncture and follow this as the patient's general condition permits by angiography; any theoretical risks attending these procedures are minimal compared to the advantages.

The demonstration of an aneurysm of the vertebral or basilar arteries or of multiple aneurysms usually precludes surgical treatment, but all aneurysms deserve consideration. If the angiogram discloses the presence of a hematoma, it should be evacuated promptly, but it is preferable to delay direct surgical attack on an aneurysm for ten days or so until cerebral edema has subsided. In the waiting period, there may be advantage to ligation of the carotids in the neck, particularly for aneurysms of the internal carotid artery and many times this comparatively safe procedure is all that is employed. Undoubtedly the most logical surgical approach is as in aneurysms of vessels elsewhere to occlude the vessel on each side of the sac. This is not as easily accomplished, however in intracranial aneurysms.

The commonest place for intracranial

greater number of patients with chronic mental disease who would otherwise have remained in institutions. In addition many who remain in institutions are more tractable. While most patients show evidence of frontal lobe deficit this is acceptable in exchange for their previous emotional illness. Convulsions and urinary incontinence which develop in some patients immediately after operation do not usually persist. The use of tranquilizing drugs in the last few years has greatly diminished but not wholly replaced the need for surgery in selected patients.

Psychosurgery in patients with persistent pain has been found not to interfere with the perception of pain but it does reduce the emotional factors which may be more distressing than the pain particularly in patients who recognize that they have incurable disease. It has not been of much use in patients who have severely painful lesions who would benefit more by some other pain relieving neurosurgical procedure. Also psychosurgery in a patient of this type produces much more inertia and more profound intellectual and emotional deficits than it does in a mental patient.

CONVULSIVE DISORDERS

Convulsive disorder more commonly referred to as epilepsy is a symptom of disease and, strictly speaking, should not be used as a name for the disease. It is evidence of dysfunction in the brain resulting from the effects of a noxious stimulus which causes a sudden and intense discharge of cells within gray matter of the cerebrum. It is believed that the discharge then spreads into adjacent regions of the cortex or into projection tracts which reach the brain stem, from whence there is a discharge in turn to other parts of the brain. If the discharge remains localized, the nature of the convulsive effects give a clue to the area of the brain from which the impulses arise. If the discharge spreads to adjacent regions there will be a march of symptoms reflecting in some degree the successive areas that are excited, though often events happen so rapidly that there is a limit to how accurately the spread of excitation can be correlated. If the discharge spreads to subcortical centers, a generalized convulsive seizure usually follows.

In patients said to have idiopathic epilepsy (essential epilepsy cryptogenic epilepsy) there are none of the elements of localized cortical discharge and the attack is charac-

terized by an immediate generalized response which varies from minor forms in which there is only momentary suspension of consciousness petit mal to immediate loss of consciousness and violent generalized convulsion (grand mal). No anatomic substratum or etiologic factor has been discovered for this type of epilepsy though some congenital abnormality in the brain has been invoked and treatment is dependent on medical management, which fortunately is adequate to control the attacks in many patients.

Surgical interest in convulsive disorders obtains only in patients who are known or believed to have some localized disease of the cerebral cortex from which attacks are easily incited (focal epilepsy). Such diseases include intracranial tumors, granulomas, arteriovenous anomalies, abscess, cortical scars with meningeal adhesions which follow inflammation or trauma, localized atrophic lesions of the cortex having numerous causes and a variety of other lesions including porencephalic cysts, calcific lesions, gliosis, birth trauma, embolic and thrombotic lesions and tuberous sclerosis. Since every patient with a brain tumor or cortical scar does not have convulsions it must be assumed that some predilection exists in those who do have convulsions.

In the patients with tumors, arteriovenous anomalies, granulomas and abscesses, the surgical problem is to eradicate the disease and once this is accomplished the convulsions may subside. But often the seizures do not subside and it can only be assumed that damaged cortex or disease left behind is the source.

Focal epilepsy a term employed when a demonstrable localized cortical lesion exists, is recognized by the occurrence of initial events in the attack which reflect the site of the lesion. These phenomena include initial unconsciousness, motor phenomena, sensory phenomena, autonomic motor and sensory phenomena, psychic phenomena and miscellaneous phenomena.

Initial unconsciousness has been the most difficult to ascribe to a specific localization, but localization appears in many to reside in the frontal lobes anterior to the motor area. Initial motor phenomena, exemplified particularly by jacksonian seizures, are characterized by turning of the head and eyes usually though not always away from the side of the lesion, vocalization and jerking of the muscles of some part or all of the opposite side of the body. Initial sensory phenomena

this manner will not cause blindness because of the rich arterial anastomosis with the external carotid artery in the orbit.

PSYCHOSURGERY

Psychosurgery which was in the beginning a term synonymous with prefrontal lobotomy now includes a variety of operations for interruption of frontal, temporal and thalamic connections by numerous technical methods designed to relieve mental disease. In 1935 Moniz and Lima performed frontal lobotomy as a therapeutic procedure in psychotic patients and, soon thereafter Freeman and Watts introduced a variation on the procedure in the United States. A huge literature has grown up concerning the implications, methods and results in psychosurgery and to date many thousands of persons have undergone operation for mental disease.

Although the operations at first were based in large part on empiricism and rather less functional anatomy has been determined than might be expected after the large experience with the operation, it can be said that the area of the frontal lobes designated as prefrontal represents the highest association area for thought processes. The prefrontal area has connections with the dorsal medial nuclei of the thalamus, the temporal lobe, particularly the hippocampus and the hypothalamus so that some operations have been directed to locations other than those of the fibers in the white matter of the frontal lobe.

Originally the operations were performed with some form of leukotome and the placement was determined by directional measurements. Lyster devised a technique however which permitted direct visibility and a modification of this operation was employed by Poppen in about 500 patients.

Since some patients having complete interruption of the frontothalamic fibers show considerable mental blunting and personality changes, search has been made for an operation that by partial division of the fibers would accomplish the desired benefit but avoid the undesirable effects.

Topectomy was introduced by Pool and the Columbia Greystone Associates as a possible means of selecting certain areas of the frontal cortex as a means of benefiting abnormal mental states. There is reason to doubt that ablation of any one circumscribed region of the cortex is of special importance in psychosurgery and Pool concluded that in topectomy there was no essential differ-

ence in end results obtained whether cortex was removed from the top or bottom of the frontal lobe. But the quantitative factor proved to be important and it was determined that to obtain a therapeutic result, it was necessary to remove 30 to 40 gm. from each side. Scoville has devised a simpler operation in keeping with these findings, whereby the cortex is undercut on the orbital surface of the frontal lobes.

A still simpler technique of interrupting fibers in the inferior portion of the frontal lobes has been devised by Freeman in the use of the transorbital leukotome. There is a risk of uncontrolled hemorrhage in this procedure though Freeman reports a mortality of but 1.7 per cent. Also some patients with more severe psychosis subsequently have required a more extensive lobotomy to obtain benefit.

Thalamotomy by a stereotaxic method has been introduced as another method of treatment by Spiegel and Wycis. They report benefits but from the standpoint of results compared to other methods, it is difficult to draw conclusions.

Grantham has devised a technique whereby an electrode is introduced into the inferior medial aspect of each frontal lobe and by application of endothermy a core of white matter is destroyed. Still another method of recent date has employed ultrasonic waves to destroy the white matter.

It seems obvious that the numerous techniques have a single purpose namely to interrupt frontothalamic connection by some safe means and in such a way that the ultimate benefit will be accomplished with the minimal alteration of the patient's intellectual and psychologic equipment. I found that interruption under direct vision of the medial and inferior connections of the frontal lobe at the level of the tip of the ventricle has produced benefits comparable to those claimed by other methods and with a minimum of complications and risks.

Results of Psychosurgery. What can now be said about the indications for and the results of psychosurgery after years of collective experience is in large part what has been learned by trial and error. Psychosurgery has proved effective in relieving destructive drives, chronic depressions and obsessions, hypochondriasis, impulsiveness and overactivity. In general, results have been good in one third, fair in one-third and of no benefit or disadvantageous in another third of the subjects. The operation has resulted in return to the community of a

dence in support of their belief that many epileptics suffer from temporal lobe seizures resulting from birth trauma. The pathologic findings suggest that compression of the lobe by temporary herniation through the ten-

torial incisure at birth is the mechanism whereby injury takes place. They believe this process was evident in 63 per cent of patients with temporal lobe seizures treated by surgical resection of the temporal lobe.

Spinal Cord

INJURIES

The majority of injuries to the spinal cord are the result of fractures or dislocation of vertebrae which follow violent flexion, extension or torsion. Less often the injuries are from direct blows or penetrating wounds. Fractures and dislocations are most common in the cervical (C5 and 6) and lumbar (L1 and 5) vertebrae and least in the thoracic vertebrae where the rib cage lends stability and protection. The damage to the cord varies from small discrete hemorrhages to complete division and, in addition there are the factors of edema and compression which may contribute to the deleterious effects on the cord.

Cervical cord injuries usually result from falls that may also produce a head injury, and if there is unconsciousness attention may be directed to the head while an important accompanying cervical injury is overlooked. The midcervical cord injury results in a classical group of neurologic changes: the forearms are held in flexion and the biceps reflex is present, while the triceps action and reflex are absent; the sensory level is at the fifth cervical segment; there is a bilateral Horner's syndrome, and respiration exists only by diaphragmatic movements. A special and infrequent type of fracture or dislocation occurs at the atlantoaxial joint where the odontoid process may fracture at its base or the ligament supporting the odontoid posteriorly is disrupted. This type of injury is the one which takes place in hanging and, if the cord is seriously damaged, death occurs shortly. With less pronounced dislocation at this joint, the cord may be partly spared enough to support respiration and some degree of neurologic function in the trunk and limbs.

Injury to the *thoracic cord* is unusual except by direct blow or penetrating wounds common in warfare: the resulting neurologic

changes correspond to the level of the injury. It must be remembered that in the upper and middle portions of the thoracic cord the segmental levels lie one to two segments above the corresponding vertebrae. All of the lumbar and sacral segments of the cord are between the levels of the eleventh thoracic and first lumbar vertebrae.

Injury to the upper *lumbar cord* produces motor and sensory paralysis of both lower limbs though sparing of the first lumbar may preserve some sensation and flexion of the thighs. Injury to the cord of significant degree at any level produces loss of sphincter control as well as spasticity of muscles, exaggerated tendon reflexes, pathologic reflexes and loss of sensation below the lesion. If the injury is in the cervical or thoracic cord there is loss of sweating and the skin becomes dry and warm below the lesion.

Damage to the *cauda equina* from spinal injury usually is patchy and incomplete since the nerve roots are less vulnerable than the cord. The resulting *paraparesis* or *paraplegia* is of the flaccid rather than spastic type and reflexes are absent. Loss of sphincter control occurs readily and at times with incomplete injury to the cauda equina there may be little other deficit.

Care of patients with cord injuries is of extreme importance from the initial handling through the prolonged period of rehabilitation. Improper transportation from the place of injury has too often added to the damage of the cord and deprived the patient of the recovery that might otherwise have taken place. Attention must be given particularly to avoiding flexion positions; extension position of the neck and back is preferred, but this can be overdone. A flat supporting appliance to which the patient is firmly fixed is safest while he is being transported to a hospital and while being moved after reach-

and their corresponding cortical areas of representation are as follows: somatic—contralateral, central or postcentral; visual—contralateral occipital lobe; auditory—either temporal lobe; vestibular—either temporal lobe; olfactory—either temporal lobe. Autonomic motor and sensory phenomena include a variety of visceral sensations and have been found to be related to areas in the region of the fissure of Sylvius and Isle of Reil. Psychic phenomena have much evidence to support their localization in either temporal lobe.

The clinical study of a patient with focal epilepsy and, in particular, a scrutiny of the pattern of the seizure is all important in arriving at a localization of the lesion, but additional evidence is sought from roentgenograms, pneumograms, angiograms and electrograms. Great emphasis has been put on the use of the electrogram in determining the exact epileptogenic focus or firing point, since present-day surgical treatment is dependent on the assumption that seizures can only be alleviated if the irritable focus is removed. Because of the limitations of the extracranial electroencephalogram, the corticogram taken from the exposed brain has sometimes been employed in the hope of attaining more precise localization.

Those engaged in the surgical treatment of convulsions have usually insisted on the need for the assistance of electrograms, but unfortunately the number of patients who have been relieved of epileptic seizures following excision of suspected cortical foci has not met expectations. The disparity has been accounted for on the basis of practical difficulties in identifying the firing point, incomplete excision of the focus, the probable existence of other unrecognized firing points and the need for limiting the excision to avoid unwanted neurologic deficits. It is only fair to say that some have raised a doubt of the justification of this manner of rationalizing the failures of operation and they have raised, in particular, the question of whether even with the acceptance of the epileptogenic focus as a primary factor the site of the focus is revealed by the spike potentials that happen to be recorded by the electrogram.

To add to the difficulties of interpreting results of cortical excision in the treatment of convulsions is the older literature which records many cures and beneficial effects in epileptics following an amazing variety of surgical procedures, many of which seem without the slightest scientific justification.

But even with the recognized uncertainties of surgical treatment of convulsive disorders it seems important to continue efforts in this direction on selected patients. The indications for surgery are generally agreed to include a clinical pattern of seizures that have focal aspects, a relatively long duration of the ailment, relatively frequent, severe seizures, and failure of control of attacks by adequate anticonvulsant measures. Under the present possibilities for benefit from operation, it appears that about 10 per cent of the populace afflicted with convulsions would conform to the indications set forth.

Penfield and his associates, who have been leaders in the effort to treat convulsive disorders in recent years, have reported their results from time to time. The operative mortality has been between 1 and 2 per cent. The evaluation of cortical excision for all types of focal convulsion has been judged to demonstrate that about 25 per cent of the patients have been cured and another 30 per cent significantly benefited in terms of lessening of the frequency and severity of seizures. The results reported by Meyers and associates are less salutary; they estimated about 11 per cent were cured and 23 per cent were improved with an operative mortality of 6 per cent.

Walker summarized the results of cortical excision in thirty-six patients who had developed convulsions following head injuries sustained in World War II. One year after operation, approximately one-third had had no attacks and another fifth had had only one attack or the isolated aura of their former attacks. The criticism of drawing final conclusions from any report is that the beneficial effects bear an inverse relation to the time of follow-up and at least ten years should elapse before the evaluation is reliable.

There have been impressive reports in the last several years of beneficial effects resulting from cerebral hemispherectomy or extensive resection of the abnormal cortex in children who have had birth palsies resulting in hemiplegia, convulsive seizures and mental retardation. Some of the reports have given convincing evidence not only of cessation of convulsions, but also improvement in mental status which can be assumed to have been secondary to termination of the seizures. Also a surprising observation has been that limbs previously spastic on the hemiplegic side have lost much of their rigidity and some useful motion has remained.

Penfield and associates have presented ev-

cial adjustment to his suddenly altered way of living and this may be the more difficult task. Passive and active exercises together with massage preserve motion at joints and prevent contractures. Braces and various supportive devices will help the patient to stand and walk, though efforts in this direction often require great effort by the patient as well as perseverance and the sympathetic direction of one skilled in management. The development of paraplegic training centers since World War II has done much to improve and advance rehabilitation methods.

TUMORS

Tumors of the spinal cord, as a category are usually meant to include intraspinal tumors of great variety which implicate the cord, its roots and the cauda equina. Such tumors may arise from the cord itself or from nerve roots, the meninges the vasculature of or about the cord the vertebrae or occasionally from tissues adjacent to the spinal column. In addition, there may be metastases from tumors elsewhere in the body which spread to the vertebrae, the epidural tissues or rarely to the cord.

The tumors of the spinal canal are classified anatomically as *intramedullary* or *extramedullary* and the latter as *intradural* or *extradural* (Fig. 20). Also some tumors are both intraspinal and extraspinal connected by a narrow tube of tumor tissue lying in the root foramen or the interlaminar space—such tumors are referred to as hour glass or dumb-bell tumors.

Incidence. The incidence of intraspinal tumors is about the same in the two sexes, with a few exceptions. For example, meningiomas which are one of the two common primary tumors, have a higher incidence in women, while the commonest metastatic tumors occur nearly exclusively in one sex, namely bronchogenic metastases in males and mammary carcinoma in females. Though tumors are occasionally encountered in children or in the aged, the third to the sixth decades, and most of all the fifth decade of life, are the periods when tumors most often make their appearance. If all types of tumors are included, a survey shows that they are found with about equal frequency in the cervical, thoracic and lumbosacral levels, although their occurrence at the extremes

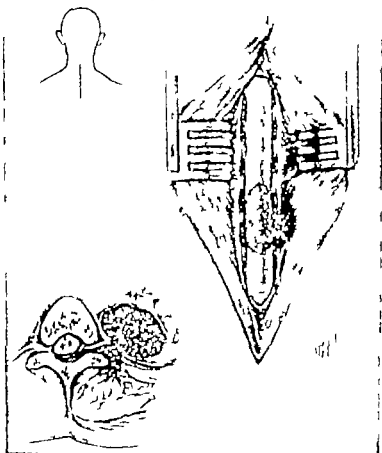


Figure 20 Extradural tumor of the spinal canal extending from the lung and mediastinum (after Ray B. S.: Surg. Gynec. & Obst., vol. 67)

ing the hospital. Since so much depends on subsequent care, it is worth while to select a hospital equipped to care for cord injuries.

In the hospital all examinations should be performed with the least manipulation of the patient. Early evaluation of neurologic changes provides a basis for comparing any subsequent changes. Early lateral and anteroposterior roentgenography is imperative but should be acquired cautiously reserving special views until a more appropriate time.

In cases of fracture or dislocation of the cervical spine, traction applied to the head is the treatment of choice. There is no justification for attempting blind manipulative procedures for reduction of malalignment. While halter traction may be used as a temporary measure skeletal traction applied to the skull is far better. Traction is begun with 5- to 10-pound weights and, if necessary for reduction of the deformity increased to 15 or 20 pounds within twenty-four hours. But greater weights of 35 or 50 pounds, sometimes recommended, are unsafe and usually not necessary since with rare exceptions even severe dislocations are realigned with lesser weights.

Compression fractures of the thoracolumbar vertebrae occur principally as a result of hyperflexion of the spine. The anterior surface of the body of the fractured vertebra becomes compressed ("wedged") causing an acute angulation of the spinal canal. Such injuries are best treated in hyperextension by either bed rest or a body cast.

Surgical treatment of spinal injuries with accompanying trauma to cord or cauda equina in many instances is controversial. In treatment of unreduced dislocations crush injuries of the laminae and most penetrating wounds, there is unanimity of opinion in favor of exploratory operation, decompressive laminectomy and fusion if necessary. There is less agreement about closed cord injuries in which there has been no displacement of vertebrae or realignment has soon been accomplished by head traction or immobilization of the trunk in hyperextension. The decision becomes important if the degree of paralysis is severe.

The following are the criteria usually accepted in deciding on the need for decompression laminectomy: a progression of neurologic signs; a complete spinal block as demonstrated by lumbar puncture and manometric studies of the spinal fluid pressures upon jugular compression (Queckenstedt test) and occasionally when there is doubt

as to the effectiveness of reduction of the vertebral deformity by traction or positioning of the trunk.

While early operation in selected cases may be desirable, a high mortality accompanies hasty surgery performed while the patient is in shock or having respiratory difficulty from high cervical trauma or when facilities are inadequate for maintaining traction, proper anesthesia, adequate suction and blood replacement if needed.

Nursing and medical care of the patient with cord injury can be one of the most exacting yet repaying efforts in medicine. The prevention of skin abrasions and decubitus ulcers requires almost fastidious attention. Such lesions develop quickly but may require weeks and months for healing and if infected seem actually to retard recovery of neurologic function. The use of a Stryker frame makes it relatively easy to change the patient's position frequently and a great boon to the prevention of decubitus ulcers has come with the development of the air mattress with chambers of alternating pressure.

For care of the paralyzed bladder an indwelling urethral catheter is introduced immediately and a closed system of intermittent bladder irrigation maintained. It is imperative that all means be taken to prevent or minimize infection of the urinary tract. In some patients, urinary control returns at least in part while some develop a degree of automaticity in evacuation of the bladder. In others the permanent loss of bladder control becomes a major problem in management and a threat to life. These cases constitute special urologic problems about which there is no general agreement.

Bowel habits including use of periodic enemas, low residue diets, digital evacuation and self-care, can be taught to the intelligent patient so that loss of anal sphincter control need not be an important problem.

Spasms of muscles of the lower limbs, particularly flexion spasms at times constitute a serious problem and are aggravated by infections of the bladder and by decubitus ulcers. Sometimes correction of these inciting lesions will control or minimize the spasms, but if not it is appropriate to resort to extensive rhizotomy or intrathecal injection of alcohol to abolish reflex action.

A rehabilitation program should be organized as soon as the patient's condition permits. As a part of the program goes a recognition of the need for the patient's so-

compression of the cord or invasion of its fibers, is evidenced by symptoms related to changes in the long tracts. The symptoms may make their appearance simultaneously with radicular pain, although occasionally tumors cause progressive myelitis without accompanying pain; this is more likely to be true of intramedullary growths.

Unilateral compression of the cord results in the Brown-Sequard syndrome in which there are homolateral alteration of reflexes, muscle weakness, impaired touch, position and vibratory senses, and contralateral loss of pain and temperature senses below the level of the lesion. Compression of the anterior part of the cord may result in loss of pain and temperature senses with preservation of other functions, while compression of the posterior cord may produce only decreased positional and vibratory senses. Intramedullary tumors, in addition to their relative painlessness, can sometimes be discerned by a handlike area of loss of pain and temperature senses corresponding to the level of the tumor and by preservation of sensation in the saddle region in the presence of sensory loss in the trunk and lower limbs. In cases of incomplete myelitis, the upper level of sensory loss does not correspond to the segmental level of the lesion, but can be from several to many segments lower, thus making it impractical to localize the level of the lesion accurately, while in complete myelitis the sensory level corresponds more exactly.

In the *third phase* with the advent of complete myelitis, there is complete loss of all but reflex functions below the segmental level of the lesion. If the myelitis has developed rapidly even the segmental reflexes may be absent for a while and the muscles become flaccid. But in slowly progressing myelitis and in some days or weeks after more acute myelitis, the tendon reflexes become exaggerated, pathologic reflexes develop and muscles become spastic. There is also loss of sweating and of sphincter control. The sensory loss and the presence of muscle inaction predispose to the development of bed sores, ulcerations from pressure, edema and wasting of soft tissues and demineralization of bones, erroneously referred to as trophic changes.

Diagnosis. A comprehensive history of the nature and chronology of the symptoms is of particular importance in detecting intraspinal tumors. The neurologic examination must be complete from head to foot and not just limited to the region under suspicion

since other common diseases of the central nervous system and even brain tumors may simulate for a while, a cord tumor. A practical knowledge of the segmental innervation of reflexes, muscle groups and sensory areas is invaluable in determining the location of a tumor. A useful accessory test is application of heavy pressure or percussion over the spinous process in the region of a tumor thereby eliciting local pain and occasionally peripheral shocklike sensations (Lhermitte's sign).

Some of the common diseases that require differentiation from intraspinal tumors are abnormalities of the intervertebral disks, multiple sclerosis, syringomyelia, amyotrophic lateral sclerosis, subacute combined cord degeneration, myelodysplasia (Guillain-Barré syndrome), diabetic myeloneuropathy, idiopathic lateral sclerosis, arachnoiditis, and syphilis. All of these diseases produce some symptoms similar to those of tumor, but each has also some distinguishing features that set it apart.

Useful adjuncts in diagnosis include roentgenography of the spine, spinal fluid studies and myelography. Roentgenograms may reveal the presence of a metastatic or other invasive tumor or disease of the vertebrae. Primary intraspinal tumors present for long periods often produce erosion of bone by pressure which may be evident particularly in the pedicles of the vertebrae or by enlargement of interlaminar or foraminal spaces.

Lumbar puncture as a diagnostic aid is particularly valuable. The Queckenstedt test will provide positive identification of a complete or nearly complete block of the spinal canal. In the majority of cases the presence of a tumor causes elevation of the protein content in the fluid while a total block of the canal will produce not only an increased protein, at times sufficient to clot the fluid, but also a degree of xanthochromia in the fluid. But it must be remembered that, while the information to be gained by lumbar puncture is important, there is distinct risk of thereby increasing the neurologic deficit through altering the intraspinal dynamics. It is preferable, therefore, that lumbar puncture be performed as one of the last tests and only when surgical facilities are made ready or available.

Myelography performed with Pantopaque or a comparable iodized radiopaque agent is the last test to be used since it is attended, similarly to lumbar puncture, by the possibility of rapidly worsening the condition.

namely the uppermost cervical and the sacral regions, is exceptional.

Some conception of the great variety of intraspinal tumors is evident in the following classified list encountered on the neurosurgical service at the New York Hospital over a twenty year period. The list includes certain non neoplastic lesions which have qualities of true neoplasms in their effects on the cord and its roots.

Intramedullary

- Gliomas (varia)
- Ependymoma
- Hemangioma (blastoma)
- Angioma (vascular anomaly)
- Metastatic tumors (rare)
- Dermoid, epidermoid and teratoma (congenital)
- Benign cysts (syrix)
- Abscess
- Tuberculoma

Extramedullary-Intradural

- Meningioma
- Nerve sheath tumor
- Hemangioma (blastoma)
- Angioma ("varicosities")
- Metastatic tumors
- Gumma

Extramedullary-Extradural

- Meningioma
- Nerve sheath tumor
- Lymphoma
- Hemangioma (blastoma)
- Lipoma
- Chordoma
- Chondroma
- Cholesteatoma
- Osteogenic tumors (benign and malignant)
- Metastatic tumors
- Dural cyst

Of the primary intradural neoplasms nearly 75 per cent were either meningiomas or nerve sheath tumors, both are benign and usually cured by surgical removal. Of all intraspinal tumors encountered at operation, nearly 50 per cent were benign and removable. *Meningiomas* are variously known as meningotheioma, dural endothelioma, meningoendothelioma, pachymeningioma, leptomeningioma, meningeal fibroblastoma, dural sarcoma, arachnoidal fibroblastoma, psammoma and by various qualifying terms, as pigmented, angiomatous and others. *Nerve sheath tumors* are also referred to as neurilemmoma, neurinoma, schwannoma, neurofibroma and perineural fibroblastoma.

Gliomas of the cord arise from the interstitial elements of the parenchyma and comprise 15 per cent of the primary intradural tumors. The commonest type is the ependymoma arising, as the name implies, from

the ependyma of the central canal or from the vestigial flum terminale. The tumors of the flum terminale may reach large size, virtually filling the lumbosacral canal, but can be removed safely providing they have not invaded the conus medullaris. The ependymomas arising in the cord sometimes extend for many segments and are visible through the widened posterior sulcus of the cord, a few have been successfully removed. Any of the types of glioma occurring in the brain may also be encountered in the cord, but whereas the commonest glioma of the brain is the highly malignant glioblastoma, the commonest in the cord next to the ependymoma is the less malignant astrocytoma. These tumors are incurable, but occasionally they can be removed in large part. They often grow very slowly over a period of years and may be slowed still more by roentgenotherapy.

The term malignancy when applied to the primary intradural tumors, refers only to the tendency for the tumors to enlarge locally and spread to adjacent parts of the cord, distant metastases do not occur.

Symptoms. The symptoms of intraspinal tumors can be conveniently divided into three phases: nerve root symptoms, beginning compression of the cord and advanced myelitis.

In the first phase initial involvement of nerve roots causes pain, which may be severe and lancinating, distributed in the corresponding dermatome and often aggravated by various forms of straining which suddenly increase the intraspinal pressure. A feature of the pain is that in many it is worst when the patient reclines, compelling him to seek relief in sitting upright or walking about. In addition to pain, irritation of a sensory root may cause a sense of numbness and paresthesias in the same area while at times the involved area of the skin may become hypersensitive particularly to the light touch of clothing. Though the patient may experience abnormal sensation corresponding to the nerve root involved by the tumor there is usually little or no demonstrable sensory loss in the area because there is considerable sensory overlap from adjacent dermatomes. With involvement of the motor fibers of the root, the corresponding muscles develop atrophy, weakness and fibrillations. In the trunk, localized paresthesias may go unnoticed, but in the extremities, particularly in the hand, the muscle wasting and weakness can be more easily recognized.

The second phase caused by beginning

Repeated recurrences of sciatic pain which in the beginning is relieved by conservative measures commonly result in a chronic persistence of symptoms which demand surgical treatment. If pain and disability persist beyond a reasonable time or if paralysis of muscles controlling the foot or bladder develops at any time surgical removal of the nucleus pulposus is desirable. Some employ myelography for the corroboration of diagnosis and localization but this is not routinely necessary. Diagnosis on clinical findings is usually reliable the only difficulty arising if the symptoms and motor and sensory signs are atypical and differential diagnosis uncertain. If operation is to be performed it is perhaps as simple to explore the disks without previous myelography. The decision to operate on a disk should largely be made on the clinical findings and not alone on the myelogram.

The surgical treatment consists of unilateral exposure, excision of the ligamentum flavum, widening of the interlaminar space, retraction of the nerve root and excision of all of the nucleus pulposus. The operation thus performed does not alter the structure of the back and can be regarded as a means of accomplishing what nature has not in removing the offending nucleus pulposus. Some favor fusion of the joint by bone grafting as a part of the operation and at times there may be a need for it but fusion is not necessary particularly in patients whose complaints and physical findings point to root compression as the source of their complaints.

Following surgery a regimen of rehabilitation is desirable before full activity is resumed and this in turn depends on a number of factors, not the least of which is the patient's emotional make-up and the factor of legal compensation. Some patients who minimize or accept minor discomforts and have a need for return to work do so without delay.

In the cervical region a ruptured disk, by compression of a corresponding nerve root, causes changes comparable to those of a ruptured lumbar disk. The neck is stiff and painful, particularly so on hyperextension. Pain extends from the neck across the shoulder into the arm and forearm, usually not farther than the wrist, while paresthesias and numbness are often present in the fingers. There are accompanying alterations of reflexes and at times weakness of muscle groups. As in the case with ruptured lumbar disks, it is usually possible with clinical

findings to localize the source of trouble. Rupture of the fifth cervical disk (C5 to 6) compresses the sixth cervical roots characteristically causing numbness and paresthesias in the first two or three fingers. Impairment of the biceps reflex and weakness in flexion of the forearm. Rupture of the sixth cervical disk (C6 to 7) compresses the seventh cervical nerve roots causing numbness and paresthesias of the middle fingers, impairment of the triceps reflex and weakness in extension of the forearm.

Lateral protrusions of the nucleus pulposus which compress the nerve roots may also be large enough to compress the spinal cord, particularly in its anterolateral quadrant. The first signs of such compression may be miotic and lid ptosis of the homolateral eye, which constitute a warning of the potential seriousness of the condition. Further compression of the cord may produce a Brown Sequard type of hemimyelitis, though there is usually preservation of the posterior columns of the cord which subserve positional and vibratory senses. Less laterally placed or centrally placed protrusion of a cervical disk is not so likely to cause nerve root compression and much more prone to affect the cord.

When the symptoms and signs point to irritation of the nerve roots only it is appropriate to employ expectant treatment just as in the case of sciatica. Often, light traction on the head will give relief by lending support to the neck and overcoming painful spasm in neck muscles. An immobilizing neck collar is also helpful. Added comfort may follow the use of heat applications, light massage of muscles and analgesic drugs. Spontaneous recovery occurs in the majority as the reaction in the involved nerve root subsides.

Surgical removal of the herniated nucleus pulposus and decompression of the involved nerve roots are indicated in the severe and persistent cases of radiculitis. The operation is performed by means of a unilateral approach between the widened interlaminar space, much the same as in the operation for lumbar disk herniation. Through a similar approach the larger herniations affecting the cord may also be removed from the extradural space, but, in some, wider exposure through bilateral laminectomy is required.

Cervical disk "ridging" is a condition which stimulates herniation of the nucleus pulposus and often the two cannot be distinguished except at operation. The ridging consists of unyielding calcified fibrous tissue

But the test may be invaluable in detecting the presence and exact location of a tumor.

Treatment. Tumors of the spinal canal, with few exceptions, should be treated surgically without needless delay. Postponement of surgery for a few hours in rapidly advancing myelitis may make the difference between permanent paralysis and recovery of function. Meningiomas and nerve sheath tumors can usually be removed completely with negligible risk and the same applies to a number of other less common extramedullary tumors. Intramedullary tumors are less favorable, but occasionally an ependymoma can be totally removed and in many of the other gliomas benefit results from partial excision, evacuation of cystic formations and by decompression through laminectomy and the opened dura. In metastatic tumors partial excision of the compressing extradural mass and decompression laminectomy followed by roentgenotherapy will often restore function and avoid disabling paralysis during the patient's remaining life. An exception to the need for urgent operation for intraspinal tumors obtains in the lymphomas which fairly commonly invade the spinal canal, particularly in the thoracic region by extension from mediastinal nodes. These tumors respond readily to roentgenotherapy, especially if it is administered in conjunction with nitrogen mustard. But such patients must be closely observed and, if myelitis progresses under treatment, surgical decompression is necessary.

INTERVERTEBRAL DISK

Rupture of an intervertebral disk and protrusion of the nucleus pulposus can occur at any spinal level but the commonest sites are at the last two lumbar and the fifth and sixth cervical interspaces. Protrusions in the thoracic region are seldom encountered.

The intervertebral disk is made up of several structures: a tough fibrous membrane which corresponds to a joint capsule and is known as the annulus fibrosus; cartilaginous plates which face the surfaces of the opposing vertebral bodies, and the nucleus pulposus, the remnant of the embryonic notochord, which occupies the closed space. Repeated stress may lead to thinning or rupture of the annulus fibrosus with bulging or actual extrusion of the nucleus pulposus into the spinal canal. The nature of the resulting symptoms depends on the degree and location of the protrusion.

In the lumbar region the initial symptoms, which often begin simultaneously or soon

after some stress on the lower back, are most often pain and limited movement in the lower back. It is believed that this initial pain arises from the torn annulus fibrosus, but the pain is indistinguishable from that which may come from any number of other structures and pathologic processes in the region. Later the pain extends into a lower limb along the course of the sciatic nerve as the nucleus pulposus compresses adjacent nerve roots. Because of the strong support of the longitudinal posterior spinal ligament the protrusion of the nucleus is to one side or the other accounting for the laterality of the symptoms.

The pain characteristically extends from the lower back on one side to the buttock, posterior thigh and calf as far as the ankle. It is aggravated by movements of the back and various forms of straining. In some patients numbness and paresthesias also occur particularly in the leg and toes. Tilting of the spine and limping with the painful limb are common. In more severe cases there is weakness of muscles which control the foot and toes and, rarely, there may be loss of bladder control.

Since it is rare for rupture to occur in lumbar disks other than the last two, the symptoms and signs are attributable for the most part to compression of fifth lumbar roots by the fourth disk or the first sacral roots by the fifth. Painful limitation of low back movements and of straight leg raising are common to both and may be the only demonstrable abnormalities. But differences in location of the symptoms and of elicited neurologic abnormalities usually permit localization of the level of the rupture. For example, rupture of the fourth disk (L4 to 5) and compression of the fifth lumbar root are more likely to cause paresthesias in the great toe, weakness in dorsiflexion of the foot and toes and diminution of the knee reflex with preservation of the ankle reflex. Conversely, rupture of the fifth disk (L5 to S1) and compression of the first sacral roots more often cause sensory changes in the last toes, weakness in plantar flexion of the foot and toes and absence of the ankle reflex.

The degree of disruption of the disk and of the symptoms varies widely. The majority of patients recover spontaneously or improve greatly in a period of six to eight weeks aided by relative inactivity and comforted by various forms of symptomatic treatment. Natural recovery is dependent on shrinkage of the nucleus pulposus and subsidence of reaction in the nerve root.

ing on the location of the pain are at the points of emergence of the nerves from the skull, as the maxillary and mandibular divisions or the several peripheral branches, as the supraorbital, infraorbital and mental nerves. It must be remembered that it is one thing to infiltrate procaine in the general region of a nerve for anesthesia and another to instill a small amount of absolute alcohol directly into the nerve. The procedure of alcohol injections is sometimes an unpleasant experience for the patient and not without occasional complications. Even so many physicians feel that relief of the neuralgia, though temporary, is worth while and that the facial numbness experienced by the patient is valuable in acquainting him with what he may expect if eventually surgical division of the nerve root is required.

Injection of alcohol or other destructive agents, such as boiling water directly into the gasserian ganglion via a needle introduced through the foramen ovale has been recommended by a few. The procedure has the advantage of avoiding an operation and, if successful, in preventing regeneration of the nerve, but it has the disadvantage of being a blind procedure not without possibility of complications and it does not permit the selectivity which can be accom-

plished by partial surgical division of the nerve root.

Auclation of the supraorbital or infraorbital nerves at their foramina is a more certain and often more effective procedure than attempts at alcohol injection of the nerves but is useful only when pain is limited strictly to areas supplied by these nerves. The surgical scars are unnoticed since one is within the eyebrow and the other within the mouth.

Rhizotomy surgical interruption of the sensory root of the trigeminal nerve, is the means by which the pain of trigeminal neuralgia can with certainty be terminated permanently. The root is that portion of the nerve lying between the gasserian ganglion and the brain stem and is exposed by an opening either in the temporal bone or the occipital bone. In the former operation the root is exposed extradurally while in the latter the root is intradural. The choice of surgical exposure is more or less arbitrary each possessing advantages and disadvantages. But important complications of either operation are relatively few and mortality from operation does not exceed 1 per cent. In about 5 per cent of surgical cases there is paresis or paralysis of facial muscles on the side of the operation. The most likely cause of this complication in the temporal opera-

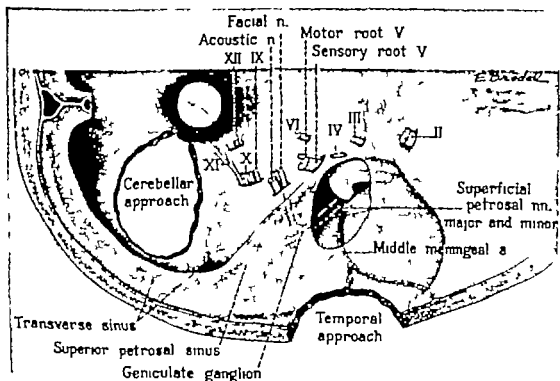


Figure 21 Drawing to show the relative positions of the cranial nerves and the two operative approaches (from Ray B. S. *Operative Technique in Specialty Surgery*, 2nd Ed., edited by W. H. Cole, Appleton-Century-Crofts, Inc.)

which constitutes the changes visible on roentgenograms and is referred to as hypertrophic osteoarthritis. The condition develops as a part of the aging process and of degeneration of the intervertebral disk. An exuberance of this tissue in the region of a cervical nerve root foramen may narrow the opening to the degree that the nerve is easily irritated. In other cases a prominent ridge may extend across the anterior surface of the cord, causing a chronic state of dis-

tortion, increased vulnerability of the cord to frequent neck movements and possibly changes in the intrinsic blood supply. The result is the subtle and insidious development of myelitis at this level, which at times has been confused in diagnosis with cord tumor, syringomyelia, amyotrophic lateral sclerosis and other diseases of the cord. The treatment consists of appropriate surgical decompression to alleviate the pressure on cord and nerve roots.

Cranial Nerves

While any one of the cranial nerves (Fig. 21) may become involved by the many diseases of the central nervous system, only certain nerves may require surgical treatment for either intrinsic disease of the nerves themselves or for relief of disease in areas supplied by them. The trigeminal nerve is by far the commonest nerve requiring some form of surgical treatment because of the frequency of neuralgia and of painful diseases within the area supplied by the nerve.

Trigeminal Nerve. Trigeminal neuralgia (synonyms: trifacial neuralgia, tic douloureux, Fothergill's disease) is one of the few true neuralgias. It is a disease of unknown origin and is characterized by paroxysms of sudden excruciating pain in the area of the face supplied by the trigeminal nerve. Since there are no physical changes to be demonstrated in the disease except some transient flushing of the face and the patient's convincing evidence of agony during a paroxysm, the diagnosis is made on the basis of the history. Because of the need for differentiating the disease from the numerous other causes of facial pain, it is imperative that the characteristic properties of the pain be recognized. The salient properties of the pain constitute a triad: (1) it is strictly limited to some part, or occasionally all, of the region supplied by the sensory root of the trigeminal nerve; (2) it is paroxysmal and of short duration, a single paroxysm rarely lasting longer than 60 seconds and (3) it is induced by stimulation of the nerve endings (trigger zones) such as light touch of the face, talking and eating.

The disease usually makes its appearance between the ages of forty and sixty years

and rarely develops at earlier or later ages. Men and women are affected equally. Though the etiology is unknown, the appearance of the affliction principally in older ages suggests a relationship to vascular deficiency or some aging process of the nerve. There is a higher incidence of trigeminal neuralgia in patients with multiple sclerosis than occurs in the general populace. Also, occasionally a tumor of the gasserian ganglion or a tumor lying adjacent to the nerve such as acoustic neuroma, cholesteatoma or meningioma occupying the cerebello-pontile angle will produce pain difficult to distinguish from trigeminal neuralgia. These accompanying diseases unlike trigeminal neuralgia, should be evident by the presence of abnormal neurologic signs.

The pain of trigeminal neuralgia never crosses the midline and bilateral disease is a rarity. Although spontaneous remissions of pain may last for months the natural history of the disease is one of unrelenting recurrence of pain with shorter and shorter intervals of relief. Rarely if ever does the untreated disease fail to recur.

Over the years many nonsurgical treatments have been proposed, but none of them provides more than temporary or partial relief at best. The pain can be relieved with certainty only by interruption of the trigeminal nerve somewhere along its pathway.

Injection with alcohol of one or more of the branches of the nerve supplying the painful region of the face is a common means of providing relief but the effect is always temporary until the nerve regenerates as it always does. The sites of injection, depend-

is uncertain but the facial nerve has been found to be swollen in the narrow facial canal without demonstrable inflammatory lesions or proof of infection. The paralysis develops abruptly usually within a few hours, and may be accompanied by some premonitory pain in the ear or face by hyperaesthesia and by blunting of taste if the nerve damage is proximal to the point of juncture of the chorda tympani. Spontaneous subsidence of the paralysis in about 85 per cent of cases commonly begins in two to four weeks or may be prolonged for several months. The more severe the degree of paralysis and delayed the return of function the more likely will be some motor aberrations of the face such as mass movements, twitching and overcontraction. Because of these undesired effects in the cases of delayed recovery surgical decompression of the nerve in the facial canal is recommended, but the merits of this procedure are uncertain.

Trauma to the facial nerve in the facial canal has most often been a complication of mastoid operations now a rarity since the advent of antimicrobial treatment has minimized the need for mastoidectomy. However injury to the nerve is greater today from other sources such as automobile accidents and more radical operations both inside and outside the cranium. Whenever possible, anastomosis of the divided nerve is preferable. Nerve grafting within the facial canal has sometimes yielded surprisingly good results and is one of the few instances in which nerve grafting to bridge a defect has been successful. When the ends of the divided nerve cannot be approximated, the method usually resorted to for restoration of facial mobility is anastomosis of the distal end of the facial nerve to the proximal end of a divided adjacent nerve such as the spinal accessory or hypoglossal nerve. The functions of either of these latter nerves may reasonably be sacrificed in exchange for restoration of even partial facial motility.

In *facial hemispasm* the aim of surgical treatment is to alter the facial nerve by partial selective sectioning of the motor fibers, thereby lessening the degree of facial movements and at the same time preserving some degree of voluntary and involuntary motion. An ideal technique for accomplishing these ends has not developed, but sometimes the results of the operation have been an improvement over the preoperative condition.

The acoustic nerve The importance of the acoustic nerve is brought to attention particularly in the surgical relief of symptoms designated as Meniere's disease. The disease is characterized by a syndrome which includes paroxysms of vertigo and unilateral tinnitus and loss of hearing. Without the existence of all of these symptoms, the diagnosis cannot be made with certainty and though paroxysms of vertigo may for a time exist alone its origin cannot be lateralized until unilateral tinnitus or deafness appears. Various causes of Meniere's disease have been proposed, but all lack convincing proof and it is not established whether the inciting lesion lies in the peripheral endings of the nerve or in the nerve itself.

The vertigo occurs characteristically in explosive attacks so severe at times that the patient may be thrown off balance. There are usually associated vomiting and at times prostration by the more severe attacks which may not subside for many hours. Between the paroxysms of vertigo there exist only the tinnitus and hearing impairment. In some, the vertigo is of mild degree insufficient to cause concern, and in some there is spontaneous subsidence of the vertigo. A variety of medical treatments have been given credit for benefit, but the occasional patient is so distressed by the severity and frequency of the paroxysms of vertigo that relief is sought in surgical measures.

Relief of the vertigo can be accomplished by either surgical destruction of the labyrinth or division of the acoustic nerve in the intracranially on the side of the tinnitus and hearing loss. The results of nerve section are good in terms of relief of the vertigo, but tinnitus is not always relieved and hearing is made worse unless a differential section of the nerve is performed by which the cochlear neurons are preserved. Unless some useful hearing exists at the time of nerve section, it is usually preferable to divide the entire nerve since a partial section may preserve some of the vestibular fibers and perpetuate the vertigo and, too, there is greater likelihood of terminating the tinnitus if a total nerve section is performed.

The glossopharyngeal nerve and sensory fibers of the vagus nerve The principal interest in these nerves pertains to glossopharyngeal neuralgia which in all respects is akin to trigeminal neuralgia except in its location. While the condition is not common, neither is it rare and sometimes neuralgia of both nerves exists in the same patient. The pain characteristically occurs in sharp,

tion is traction during the operation on the superficial petrosal nerve a branch of the geniculate ganglion of the facial nerve. Recovery of facial motion is much the same as occurs in Bell's palsy. If the operation results in corneal anesthesia, precautions must be taken to avoid trauma to the insensitive cornea and resulting ulceration. With reasonable care of the eye such ulcerations can be avoided. All patients experience some degree of paresthesias in the anesthetic area and, for most, accommodation is no problem. But for a few particularly the over-anxious, there may be considerable unhappy preoccupation over the sensations for which, unfortunately there is no satisfactory remedy.

Partial or selective section of the sensory root is employed particularly in patients whose pain is restricted to the lower part of the face, in which case nerve fibers supplying the cornea can be preserved, thus obviating the risk of corneal ulceration. The motor root of the trigeminal nerve is preserved if possible since its loss results in paralysis of masticator muscles on that side. It may not always be possible to preserve the root, but fortunately patients readily adjust to the paralysis.

Section of the descending tract pain fibers only of the trigeminal nerve in the medulla oblongata was proposed by Sjöqvist in the hope thereby of avoiding total anesthesia of the face and possibly diminishing the degree of paresthesias that follow rhizotomy. The operation is attended by greater risks, but these might be acceptable if the operation had accomplished what was hoped for. Unfortunately in about 35 per cent of cases there was recurrence of pain and paresthesias in the same degree as following rhizotomy. For these reasons tractotomy is now rarely performed for relief of trigeminal neuralgia.

"Decompression" of the root of the trigeminal nerve was proposed by Taarnhøj. It was his concept that the neuralgia might result from some degree of compression and irritation of the root along its course and that relief might result from dividing the meningeal coverings of the root. A certain degree of success attended the operation, but the uncertainty of the means by which the operation accomplished success has led to a variety of other technical procedures in which the nerve root or ganglion is exposed and lightly traumatized. No doubt, in such operations some of the sensory fibers are interrupted, but the principal claim of ad-

vantage made for the operations is preservation of most of the facial sensation. The rate of recurrence of neuralgia following the operations is not established, although it is probably at least 50 per cent. But if even half the patients were relieved of neuralgia by an operation which preserved most of the facial sensation this would be one of the useful methods at our disposal for treatment of this disease.

Other Cranial Nerves. The other cranial nerves which sometimes may be advantageously divided for therapeutic effects are the seventh (nervus intermedius) eighth, ninth tenth (sensory portion) eleventh and twelfth. These nerves all lie in an area, intracranially readily exposed by a suboccipital craniotomy of the type used to expose the trigeminal nerve in the cerebellopontile angle. The seventh, ninth, eleventh and twelfth nerves in their extracranial course lie in general proximity in the superior triangle of the neck.

The facial nerve. The sensory division of the facial nerve is the nervus intermedius of Wrisberg. A rare type of neuralgia comparable in quality to trigeminal neuralgia, but principally localized deep in the ear is believed to implicate the nervus intermedius and relief is obtained by division of the nerve intracranially. The principal risk of the operation is damage to the motor division of the facial nerve with resulting facial paralysis. There is also chance of damage to the auditory nerve which lies in proximity to the facial nerve.

Disturbances of the extracranial portion of the facial nerve are evidenced by either paralysis or paroxysmal spasm of the face. Paralysis is most commonly of the Bell's palsy type but may result from operations on the parotid gland or mastoid bone and from lacerations of the face skull fracture through the petrous bone, facial nerve tumors, geniculate herpes zoster birth injury and congenital maldevelopment. Hemifacial spasm is often the sequel of incomplete recovery from Bell's palsy but the etiologic factors are usually not evident. The condition is to be distinguished from facial tic, or habit spasm, which is a manifestation of neurosis.

Bell's palsy a unilateral paralysis of the face involves all branches of the nerve and is sometimes spoken of as "peripheral" in contradistinction to "central," a supranuclear type of paralysis seen for example in hemiplegias in which the distortion of the face is not so profound and motion in the forehead is unaffected. The cause of Bell's palsy

Peripheral Nerves

INJURIES

Peripheral nerve injuries in peacetime even in busy civilian hospitals are not common and teaching clinics find a paucity of material suitable for sustaining interest and for demonstrating to students the principles of this important subject. But the two world wars produced thousands of these injuries and it is to the credit of many physicians working under the hardships of war that important advances were made in diagnosis and treatment. Not only was this concerted effort invaluable to the wounded soldier but it has provided us with far more understanding of civilian nerve injuries than would otherwise have been possible.

The increased number of injuries from automobile accidents has made most physicians aware of the problems of acute trauma but the point cannot be overstressed that lacerations, fractures and deep tissue bruises of the extremities are likely also to injure important nerves. Failure to recognize the nerve injuries and institute early care can be a serious oversight. It is especially important to identify the presence of a nerve injury before attempting to reduce fractures or encase an extremity in dressings or plaster.

Besides lacerations and fractured long bones there are other common causes for peripheral nerve injuries, such as dislocations, retained hematoma, accidental injury by the surgeon's scalpel, compression by tourniquet, splint or cast, aneurysms, sudden stretching, malposition during sleep or while under anesthesia, birth palsies and recent callus formation, repeated local trauma, and injury from injection needles.

From United States Army records, Pollock reported the distribution of peripheral nerve injuries in 7050 cases. There is prob-

ably little variation from this in civilian cases.

Pathology. A divided or seriously traumatized nerve undergoes wallerian degeneration characterized by fragmentation and disappearance of myelin and axons of the segment distal to the injury. Regenerative changes soon begin but in the distal segment are abortive. In the proximal segment, within the first week, protoplasmic bands form providing pathways along which regenerating neurofibrillae of the segment find their way distally. If the nerve has not been divided, the downward growth of neurofibrillae continues to complete the regeneration. Regeneration occurs only from the proximal segment.

If the nerve has been divided, the ends draw apart and scar tissue fills the interval. As regenerating axons of the proximal segment meet the scar tissues in their downward growth, they are turned back to form a coiled bulbous neuroma. A bulbous enlargement also develops at the end of the distal segment but is made up of connective tissue and known as a false neuroma. A neuroma may also form on the proximal end of interrupted fibers of an incompletely divided or "notched" nerve trunk, while in transected injury to an intact nerve may result in a fusiform neuroma within the nerve sheath also referred to as neuroma in continuity. Scar tissue resulting from injury or infection adjacent to a normal nerve may occasionally compress the nerve and result in a degree of degeneration, but more often the scar in perineural tissue is a part of a traumatic scar in the nerve.

Symptoms. All of the peripheral nerves in the extremities are made up of afferent, efferent and sympathetic fibers. Interruption of a nerve therefore results in loss of motions subserved by muscles supplied by that nerve and the muscles become atrophied. Sensation of all forms disappears within an area, the pattern of which with slight variations is fairly constant for each nerve. Loss of sympathetic innervation is evidenced by loss of sweating and a degree of warming and reddening in the corresponding area due to loss of sudomotor and vasomotor control. Not only do muscles atrophy in the area supplied by the divided nerve, but many so-

Table 3. Distribution of Peripheral Nerve Injuries

Brachial plexus	139
Median	1376
Ulnar	2261
Radial	997
Musculocutaneous	87
Sciatic	1161
Tibial	252
Peroneal	558
Femoral	21
(and small groups of others)	

short lived paroxysms in the region at the base of the tongue and tonsillar fossa on one side. It is usually induced by swallowing or by internal or external pressure in the tonsillar region. Intracranial division of the glossopharyngeal nerve is a satisfactory form of treatment since it can be counted on to relieve the pain and does not result in any demonstrable neurologic deficit. Experience has shown that in a few instances full relief of pain is not obtained unless several rostral sensory fibers of the vagus nerve are divided as well as the glossopharyngeal nerve. For this reason both nerves are usually divided to insure the desired relief of pain. Rarely the paroxysms of glossopharyngeal neuralgia are accompanied by syncope resulting from cardiac arrest. The phenomenon is explained by the role which the glossopharyngeal nerve serves in the transmission of afferent impulses arising in the carotid sinus. In the investigation of this striking combination of glossopharyngeal neuralgia and syncope from cardiac arrest, the latter effects can be obliterated by atropinizing the patient while the paroxysms of pain persist. Both can be temporarily abolished by cocaineization of the region of the tonsillar fossa and permanently abolished by section of the glossopharyngeal nerve.

Occasionally and particularly in older patients with arteriosclerosis, hypersensitivity of the carotid sinus and syncopal attacks exist in a degree sufficient to constitute a serious threat to the patient's safety. While the condition can be corrected by local resection of the nerves about the involved carotid sinus, intracranial section of the glossopharyngeal nerve can be performed with as much safety and with more assurance of benefit.

Because intracranial section of the glossopharyngeal nerve interrupts afferent fibers from the carotid sinus which influence the cardiovascular reflexes, there is frequently a significant but temporary rise in blood pressure following rhizotomy. The elevation of pressure, if present, rarely lasts longer than a few hours.

Another instance in which the glossopharyngeal nerve may be involved is in the condition known as the auriculotemporal syndrome and characterized by excessive sweating in the temporal region induced by gustatory stimuli. The condition results from

an abnormal union of the chorda tympani and the auriculotemporal branch of the trigeminal nerve following penetrating wounds or surgical procedures in the region of the parotid. Section of the glossopharyngeal nerve intracranially interrupts the reflex and causes no untoward effects.

The spinal accessory nerve. This nerve has origin from both cranial and spinal levels and is a motor nerve supplying the sternocleidomastoid and trapezius muscles. It has surgical significance in several circumstances. Injury to the nerve from penetrating wounds or surgical procedures in the neck is relatively common. While the loss of muscle function resulting from the division of the nerve is not seriously disabling and is accompanied by certain compensatory muscular actions repair of the divided nerve should be performed if feasible. Damage of the nerve should be avoided in operations on the neck.

In surgical treatment of spasmodic torticollis the spinal accessory is sometimes divided along with several upper cervical motor roots to accomplish deliberate paralysis of the muscles responsible for moving the head and neck. The operation has limited use but has occasionally proved helpful.

A more common practice in which the spinal accessory nerve is divided is in repair of facial paralysis, particularly that resulting from interruption of the nerve in the removal of acoustic neurinomas in the cerebello-pontile angle. In the reparative operation, the main trunk of the nerve is divided and its proximal end anastomosed to the distal end of the facial nerve at its site of emergence at the stylomastoid foramen. An additional refinement of the operation is to divide the descending hypoglossal nerve and anastomose its proximal end to the distal end of the divided spinal accessory nerve. This operation provides a degree of innervation for the muscles supplied by the spinal accessory nerve and, in particular has value for its cosmetic effects.

The hypoglossal nerve. As an alternate procedure to the use of the spinal accessory nerve for anastomosis to the facial in the alleviation of facial paralysis, the hypoglossal nerve may be used to equal advantage. The choice in use of either nerve for the anastomosis is arbitrary—any advantages or disadvantages being about equal.

arm, the most notable deformity is wristdrop in which there is inability to extend the hand at the wrist or the fingers at the metacarpal joints (Fig 22). In addition there is loss of extension and abduction of the thumb and failure of the brachioradialis to contract with forceful flexion at the elbow. It is not possible for the patient to make a firm fist or hold objects in the hand with the wrist in full flexion. With support to the wrist such as a splint holding the wrist in mid position or slight cock-up full strength in grasping is possible. The thumb deprived of extensor and abduction motions interferes with flexion of the index finger and patients may hold the thumb aside with the other hand when making a fist.

If the nerve is injured at a high level in the arm there is loss of triceps action and inability to extend the forearm in addition to the other paralyses. If the branch of the nerve in the forearm is injured the typical palsies in the hand occur but without loss of extension at the wrist. Injuries of this kind are not uncommon because of the frequency of lacerations of the forearm and of accidents in surgical operations near the elbow such as removal of the head of a fractured radius.

Sensation is lost in a small area in radial nerve lesions (Fig 23). It is unimportant to the patient and easily overlooked by the

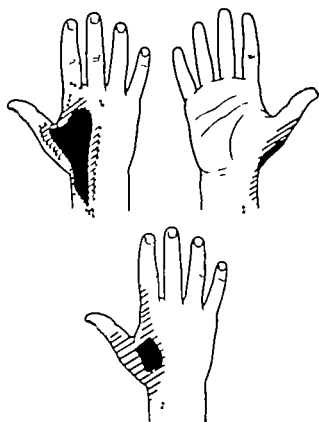


Figure 23 The sensory loss of radial nerve paralysis is small because of the overlap from adjacent uninjured median and ulnar nerves. The solid black represents loss to all modalities; the dots represent the loss to pinprick, and the straight lines, the loss to touch.



Figure 22. The characteristic wristdrop deformity of radial nerve paralysis.

examiner but if present along with motor palsies it is a fairly reliable sign of the completeness of the nerve injury. The isolated area of anesthesia lies in the region of the dorsal aspect of the thenar web adjacent areas of sensory overlap being supplied by the median ulnar and musculocutaneous nerves.

Median Nerve. Although the commonest site of injury to the median nerve is at the wrist, particularly from cuts by glass, the nerve may be injured at higher levels by stab wounds and lacerations of the arm and forearm or by extensive fractures about the elbow. The most striking changes are in the small muscles in the hand but with the higher lesions of the nerve, the characteristic loss of flexion of the distal phalanx of the index finger from loss of contraction of the flexor profundus is a highly reliable sign (Fig. 24). This defect is easily demonstrated by having the patient clasp fingers of the two hands in the attitude of prayer a position which will emphasize the absence of flexion of the index fingers or the patient

called trophic changes take place the result principally of disuse and minor trauma to anesthetic tissues. These changes include demineralization of bones stiffening of joints, atrophy of skin, keratosis, diminution of subcutaneous tissue, ulcerations overgrowth and ridging of nails and overgrowth of hair. Incomplete severance of a peripheral nerve results in partial loss of the normal functions and tissue changes are less profound. If regeneration of a divided nerve takes place and in the meanwhile proper care of the denervated tissues is provided, these changes can be kept at a minimum and are in large part reversible with reinnervation.

A variety of unpleasant sensations may accompany interruption of peripheral nerve function. They include principally paresthesias, hyperalgesia, stiff and wooden feelings, coldness and pain. The pain is of two varieties, so-called spontaneous pain and causalgia. The former pain is ill defined and in constant. Its nature is not fully understood, but it probably represents overresponse to unrecognized stimuli from marginal areas.

Causalgia is a term coined by S. Weir Mitchell at the time of the Civil War. It is characterized by intense, persistent, burning pain radiating diffusely up the injured limb and is adversely affected by extremes of temperature, light touch and psychologic stimuli to the degree that an afflicted patient will hold the limb immobile and devote his entire attention to avoiding the stimuli which he soon learns add to his misery. Causalgia occurs only with partial division of nerves and is more likely to result from injuries to the median nerve in the upper limb and the tibial nerve in the lower limb. In spite of many investigations, the cause of causalgia remains an enigma, for none of the proposed theories accounts for all the facts. Unfortunately the diagnosis is too often used loosely to include many or all discomforts associated with nerve injury. This error becomes important since sympathectomy will usually alleviate causalgia, whereas sympathectomy is wholly ineffective in other complaints associated with the injury.

Diagnosis. The recognition of common nerve injuries is not difficult if a few simple tests for motor and sensory functions are employed, particularly in the distal parts of the extremities. One need be acquainted only with the major functions of the principal nerves, though once the existence of a nerve injury is recognized more detailed studies are important.

In examining muscle function to determine the possible interruption of nerve supply there are several factors to consider. One of these is to have in mind the levels at which branches to various muscles leave the main trunk of the nerve. For example in testing for possible injury to the median nerve from a wrist laceration one would not conclude that the nerve was intact because the patient could flex the last joint of the index finger. This action is produced by a flexor muscle which receives its innervation from a branch of the median nerve above the wrist.

Other factors to be considered are anomalous or dual innervation of some muscles, such as the flexor and opponens muscles of the thumb and anomalous muscle action. The latter term is used to describe unusual action of a muscle without reference to its nerve supply. Two types are recognized—supplementary and trick movements. These movements result from (1) tension on paralyzed muscles by their antagonists as occurs when formation of a fist causes extension at the wrist in the presence of radial paralysis (2) rebound movements which occur when normal muscles are suddenly relaxed and (3) movements resulting from gravity. Failure to recognize these accessory movements leads to error in evaluating the presence and degree of a nerve injury.

In sensory tests for determining nerve injury there is also variability. For example, the autonomous sensory zone of the ulnar nerve in the hand usually includes the fifth finger and the ulnar half of the ring finger but occasionally it is limited to the fifth finger and median nerve sensory fibers overlap the ring finger.

Numerous electrodiagnostic tests have proved to be useful in the study of peripheral nerve injuries both in determining the degree of the injury and in assessing regeneration. These electrical tests refer particularly to the results of stimulation of various strengths to a motor nerve, the determination of chronaxia as an index of excitability and electromyography which measures action potentials of muscles. These refinements in examination of nerve and muscle functions are important and at times of great practical use but not always simple to perform or interpret.

Radial Nerve. The intimate relationship of the radial nerve to the humerus makes the nerve particularly vulnerable to injury and, as a result, it is the most frequently injured nerve in the arm. With a lesion in the mid

tude which is due to unopposed action of the extensor digitorum communis (Fig. 27)

When the lesion is at the elbow or higher there is loss of ability to flex the proximal or distal phalanges of the last two fingers to abduct or adduct the fingers when they are put in extension by placing the hand on a flat surface to adduct the thumb to abduct or oppose the little finger and to contract the flexor carpi ulnaris. In lesions at the wrist or distal part of the forearm the flexor muscles of the last two fingers and the flexor carpi ulnaris are spared.

There are a few simple tests well suited to demonstrating the loss of function in ability to bring the fingers together when

held in extension loss of strength in grip in the ulnar side of the fist inability to hold an object by adduction of the thumb without flexing it and inability to approximate the tips of the last three fingers in forming a cone (Fig. 28)

Sensation is lost over the little finger ulnar side of the ring finger and ulnar side of the hand on both the palmar and dorsal aspects (Fig. 29) Occasionally sensory overlap will reduce the area of absolute sensory loss to the fifth finger. The sympathetic paralysis accompanying ulnar nerve lesions causes loss of sweating, dryness of the skin and a violaceous discoloration on the ulnar side of the hand.

Combined Median and Ulnar Nerve Lesions. It is not uncommon that both the median and ulnar nerves are divided in lacerating injuries at the wrist. Such injuries also divide the flexor tendons to the hand. Serious disability can be avoided only by the most painstaking care in surgical repair of tendons and nerves followed by diligent after-care. The deformity resulting from the combined nerve injuries represents a composite of the changes seen with each. The simian hand is even more striking than that occurring in median nerve paralysis the

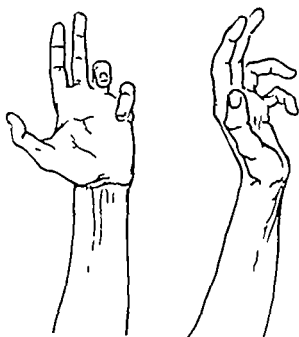


Figure 27 The clawhand deformity of an ulnar nerve lesion.

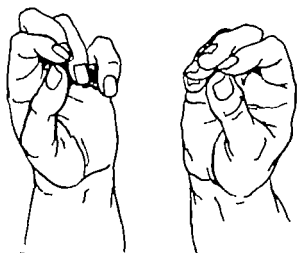


Figure 28 The patient with an ulnar nerve lesion is unable to make a cone with the tips of the finger and thumb.

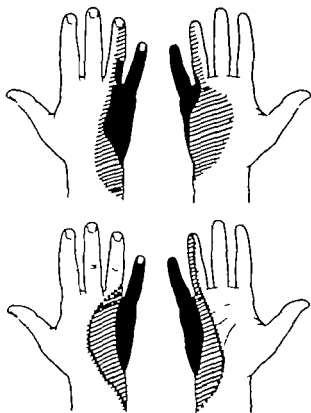


Figure 29 A drawing which illustrates the loss of all modalities and pinprick and touch sensation in an ulnar nerve lesion.

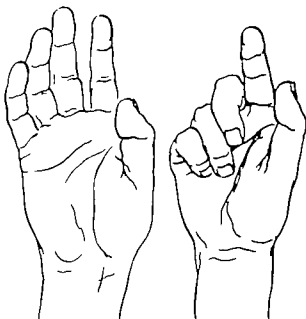


Figure 24 The deformity of median nerve paralysis is characterized by the position of the thumb in the same plane as the palm of the hand, and inability of the patient to flex the index finger as in making a fist.

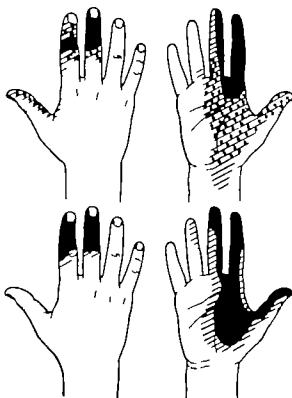


Figure 25 A drawing which represents a typical loss of sensation to all modalities, pinprick and touch as a result of a median nerve lesion.

when asked to scratch with the nail of the index finger while holding the hand on a flat surface will be unable to do so

The striking changes in the hand from atrophy of the small hand muscles due to median nerve paralysis produce a characteristic deformity referred to as ape hand, in which the thenar eminence becomes flattened and the thumb takes a position in the plane of the palm. The single examination most often used in testing for median nerve paralysis is to have the patient oppose the tip of his thumb to the tip of the little finger to form the letter "O". Care must be taken, however in the testing since supplementary movements develop readily in median nerve palsy with the exception of flexion of the distal phalanx of the index fingers

The area of sensory loss is over the radial side of the palm and the palmar aspect of the thumb, index, middle and radial side of the ring finger (Fig. 25). On the dorsum of the hand the loss is limited to the distal half of the index and middle fingers. Because of extensive overlap from the ulnar and radial nerves there is considerable variability in the extent and degree of sensory loss except for the distal ends of the index and middle fingers. Even in patients with extensive overlap of sensation, the region of lost sensation imposes a considerable degree of impairment in use of the hand. Because of sympathetic paralysis, moisture of much of the palmar

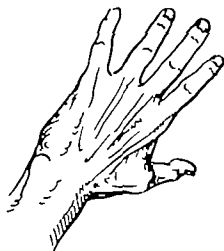


Figure 26 The atrophy of the dorsal interosseous muscles is marked in an ulnar nerve injury particularly between the metacarpal of the thumb and the index finger

skin is lost, resulting in dryness and chapping.

Ulnar Nerve. The most striking changes in ulnar nerve paralysis are in the hand.

The marked degree of wasting in the dorsal aspect of the thenar web and of the interosseous spaces gives the hand a skeleton-like appearance (Fig. 26) and the last two or three fingers assume a clawing atti-

determine at the time of injury whether a nerve has been anatomically or physiologically interrupted and in nonpenetrating injuries there is seldom need for immediate surgery. Recovery will often occur spontaneously but it is imperative that frequent periodic examinations be made and if within three to five months recovery is uncertain or not proceeding satisfactorily there should then be no hesitation about exploring the nerve surgically. Even earlier exploration is sometimes justified since the attending risks of the operation are negligible. If the nerve is found intact no harm has occurred. For example when the radial nerve is caught between the fragments of a fractured humerus, valuable time is saved in healing of both bone and nerve if early exploration is performed. In traction and pressure paralysis of the brachial plexus or some of its branches such as the axillary nerve there is so little to be expected from operation that exploration is rarely required.

In the presence of puncture or lacerated wounds the ideal time for repair of injury to a peripheral nerve is immediately. It is even reasonably safe with the use of antimicrobial therapy to repair a wound as long as forty-eight hours after its infliction and carefully repair a divided nerve. Even if infection develops in the wound the nerve may regenerate or if a secondary operation and reanastomosis of the nerve are required later the ends will be approximated and retraction will have been prevented.

In the repair of a divided nerve the approximation of the freshened ends without tension and by the use of fine silk stitches in the epineurium is sought. When tension on the nerve is necessary in order to approximate the ends usually sufficient relaxation can be had by flexion or extension of the extremity and immobilization in that position for six weeks. Occasionally transposition of the nerve will afford the needed gain in length for anastomosis. For example, the radial nerve divided in the midarm has a shorter route in the arm if its position is transposed to the opposite side of the humerus. Larger defects in a nerve have not been successfully treated by interposing grafts but occasionally a defect can be overcome by procedures designed to stretch the proximal segment of the nerve gradually to bring it in approximation with the distal segment.

When important peripheral nerves cannot be repaired one has recourse at times to

muscle and tendon transplantation or arthrodesis of joints with good functional result. Also occasional good results follow direct implantation of a nerve end into a muscle.

After-care. It is not possible to overemphasize the place of carefully supervised and persistent physiotherapy to the limb following peripheral nerve injury, both before and after surgical repair. The treatments are directed toward preservation of the intact skin, prevention of fibrosis and atrophy of muscles and of contraction of joint capsules. The methods should include application of wet and dry heat, massage, passive movement of joints, electrical stimulation of paralyzed muscles and re-education exercises as soon as possible. Mechanical splints help not only in early use of the limb but provide support in the normal position thereby preventing contraction at a joint or overstretching of paralyzed muscles.

As a part of the after-care it is important to detect the early evidences of regeneration of the nerve and to follow the progress. One of the oldest and most easily applied tests is Tinel's sign. The test consists of eliciting a tingling or shocklike sensation in the area of sensory distribution of a nerve by tapping gently over the nerve distal to the site of injury. The response is presumed to result from stimulation of regenerating unmyelinated sensory axons. Its accuracy as a test is sometimes questioned but, in practice, it usually proves to be a simple and satisfactory way of following the downward progress in a regenerating nerve.

A highly reliable test which can be utilized when it is important to detect regeneration of motor fibers in a nerve is intraneural stimulation by means of fine wire electrodes introduced percutaneously directly into the nerve.

Electromyography and chronaxy tests, useful means of detecting function in the initial evaluation of the nerve injury are of equal value in assessing regeneration. The tests can show evidence of reinnervation of a muscle some time before it can be recognized by clinical methods and thus give early assurance that a nerve suture is intact.

Tardy Nerve Palsy. Occasionally paresthesias of peripheral nerves in the upper extremities develop insidiously years after a fracture which has healed either with excessive callus that compresses the nerve or with a deformity that displaces the nerve and exposes it to easy trauma. Such lesions of the radial or median nerves are not often seen,

thumb is held in abduction in a plane with the palm and the last two phalanges are held in moderate flexion. Sensation is absent in the palmar side of the hand, both sides of the fingers and the ulnar side of the dorsum of the hand, the thumb is partly supplied by overlap from the radial and musculocutaneous nerves. Discoloration from loss of vasomotor control, dryness of the skin and trophic changes develop in the entire hand.

Brachial Plexus. Lesions of the brachial plexus are usually designated on the basis of the anatomic site or the mode of injury. The plexus arises from the fifth cervical to the first thoracic nerve roots, the roots combine to form trunks and the trunks to form cords. The modes of injury are traction, pressure and laceration.

A common type of traction injury is that which occurs to the child at birth. If by some means the head and shoulder are forced apart, the upper part of the plexus is stretched and the resulting paralysis of the deltoid, biceps and brachioradialis muscles is referred to as Erb's palsy. The same type of injury is encountered postnatally from a great variety of accidents which cause stretching of the head and neck away from the shoulder, sudden forceful depression of the shoulder or violent downward pull on the arm.

If hyperabduction and traction are put on the arm the injury occurs to the lower part of the plexus and is referred to as Klumpke's palsy. These injuries also occur during childbirth as well as in later years and result in palsies of ulnar or ulnar and median nerve types, often accompanied by Horner's syndrome.

Pressure injuries to the plexus occur in a variety of circumstances, some of the more common being prolonged dislocation of the shoulder, pressure in the axilla from a crutch, pressure from shoulder braces in prolonged Trendelenburg position, aneurysm of the subclavian artery and displaced fragments of a fractured clavicle.

Injury to the axillary nerve occurs almost exclusively as a complication of dislocation of the humerus at the shoulder. Usually the continuity of the nerve remains intact though it may be disrupted. The paralysis is recognized by loss of function of the deltoid muscle and by a small patch of sensory loss over the muscle. The motor deficit requires differentiation from disruption of the "cuff" of rotator muscles attached to the humerus.

Lesions of the cords of the plexus more often result from penetrating wounds. In

their effects they simulate the changes which follow nerve injuries more peripherally. Involvement of the medial cord affects areas innervated by the ulnar lower portion of the median and the medial cutaneous nerves. Lateral cord injuries cause paralysis of the areas supplied by the musculocutaneous and upper portions of the median nerves. Depending on the location of injury in the posterior cord, the thoracodorsal, subscapular axillary or radial nerves are affected. But it is rare that injury to the plexus involves a single component and recognition of the exact degree of injury is often further complicated by local edema, hemorrhage, traumatic aneurysm or the later development of scar tissue.

Sciatic Nerve. Severance of the main trunk of the sciatic nerve from penetrating wounds is a seriously crippling injury since it results in paralysis of all muscles below the knee and loss of sensation over the outer side of the leg and the entire foot with the exception of the inner border of the arch. Partial injuries of the sciatic nerve, particularly in the lower part of the thigh, may involve one or the other of its two main divisions, the peroneal and tibial nerves.

Peroneal Nerve. The peroneal nerve is the most frequently injured nerve in the lower extremities as a result of common fractures of the upper end of the fibula and from pressure by casts, splints, traction devices and under knee rolls. To this list of causes should be added pressure from the crossed-leg position that some people assume for hours while in sitting posture, and resting pressure in emaciated bedridden patients. The deformity is easily recognized by foot drop and alteration in gait characterized by lifting the leg to avoid tripping with the toe. Partial paralysis can be identified by weakness in dorsiflexion of the foot and great toe when an attempt is made to walk on the heel. The sensory loss is over the lateral aspect of the leg and dorsum of the foot to the base of the toes.

Tibial Nerve. Isolated lesions of the tibial nerve are uncommon, particularly from injuries below the knee. The principal motor supply is to the muscles that plantar flex the foot and toes and the paralysis is identified simply by inability to walk or support weight on the toes. Sensation is lost over the sole of the foot except the inner border of the arch. Traumatic ulceration of the sole is a complication which readily occurs if the patient walks on the foot.

Surgical Treatment. It is not possible to

Recklinghausen's disease In this disease there are multiple tumors, sometimes hundreds, varying in size and in degrees of malignancy though they are usually benign. Less often the same type of tumor occurs as an isolated single lesion. Whether these tumors take origin from the neurilemma, perineurium or epineurium is often difficult to determine and has led to confusion in classification as well as many synonyms (perineural fibroma, neurinoma, neurilemmoma, schwannoma, neurofibroblastoma, neurofibrosarcoma and others).

Since the longest course of peripheral nerves is in the extremities, it is understandable that these neoplasms are most numerous there. In the upper limbs they are somewhat more common on median and ulnar nerves in the forearms but may arise from small nerve branches even in the fingers. In the lower limbs they are usually found on the posterior aspect of the thighs and legs. The neck, with its multiplicity of nerves, is a favorite location and nerve tumors must always be considered in the differential diagnosis of tumors in this region.

Von Recklinghausen's disease is of congenital origin though the tumors do not usually grow to recognizable or disturbing size till young adulthood. Not only are peripheral nerves involved but in many cases the cranial nerves, spinal nerve roots and sympathetic nerves as well. Associated conditions include scattered areas of pigmentation of the skin producing café au lait spots, abnormalities of bone, cutaneous tumors that are sometimes plexiform or pedunculated, evidence of defective development of the nervous system and other malformations. Any of the tumors may undergo sarcomatous degeneration and become actively invasive in adjacent tissues.

Neurofibromas that involve major nerves and threaten paralysis should be removed and this can usually be accomplished without sacrifice of the nerve if malignant changes have not developed. Tumors known to be or suspected of being malignant should be excised with a wide margin and residual tumor can be expected at times to respond well to radiation therapy. Rarely such tumors develop distant metastases.

Neuromas are usually understood to be traumatic enlargements at the ends of divided nerves or along the course of injured nerves though true neuromas of benign nature are occasionally found on peripheral and sympathetic nerves. Traumatic neuromas in scars and in amputation stumps as

sume importance sometimes because of pain associated with them. Surgical revision of painful scars and stumps may be repaying in relief of pain but more often fails due in part to psychologic factors.

Glomus tumors are made up of the end organs of the nerve together with elements of small blood vessels and smooth muscle. They are rare but their presence should be recognized by the intense pain caused by pressure over them. They are usually small, rarely more than a few millimeters in size, and though they have been encountered in the subcutaneous tissues in many parts of the body the majority occur in the subungual region of the fingers. The tumors are benign and curable by excision.

Incastation by malignant tumors adjacent to peripheral nerves is often met with, particularly in the brachial plexus and in the lumbosacral plexus in the pelvis. The malignant tumors of the breast and lung which invade the brachial plexus produce neurologic symptoms and signs referred to as the superior sulcus syndrome or Pancoast syndrome. Initial symptoms are pain in areas supplied by the lower part of the plexus, namely the inner side of the arm and fore arm, and also sympathetic paralysis in the limb from involvement of the region of the stellate ganglion. With advance of the disease there are progressive motor and sensory palsies in the limb. Treatment is restricted to appropriate methods of controlling pain.

READING REFERENCES

- Adson, A. W.: Surgical Treatment of Glossopharyngeal Neuralgia. *Arch. Neurol. & Psychiat.* 12:487 1924.
- Adson, A. W.: Intraspinal Tumors; Collective Review. *Internat. Abstr. Surg.* 67:225 1938.
- Basley P.: Intracranial Tumors, 2nd ed. Springfield, Ill., Charles C Thomas, Publisher 1918.
- Basley P., and Cushing, H.: A Classification of Tumors of the Glioma Group on a Histogenic Basis with a Correlated Study of Prognosis. Philadelphia, J. B. Lippincott Company 1926.
- Bassett, R.: Surgical Experiences with Arteriovenous Anomalies of the Brain. *J. Neurosurg.* 8:59 1951.
- Botterell, E. H., and Drake, C. G.: Localized Encephalitis, Brain Abscess and Subdural Empyema. *J. Neurosurg.* 9:348, 1952.
- Covall, D. A., Cooper, I. S., Hoen, T. L., and Rusk, H. A.: Early Management of Patients with Spinal Cord Injury. *J.A.M.A.* 151:89 1953.
- Crutchfield, W. G.: Skeletal Traction in the Treatment of Injuries to the Cervical Spine. *J.A.M.A.* 155:29 1954.
- Dandy W. E.: Meniere's Disease; Its Diagnosis and Method of Treatment. *Arch. Surg.* 16:1127 1928.
- Dandy W. E.: Surgery of the Brain. Hagerstown, Md., W. F. Prior Co., 1945 pp 167-202.

but tardy ulnar palsy is a well recognized syndrome that follows injuries about the elbow which alter the carrying angle of the extremity. There is a gradual loss of sensation, sometimes accompanied by paresthesias in the ulnar distribution of the forearm and hand, more particularly in the fifth finger at the outset. Palsy of muscles supplied by the ulnar nerve also occurs and is usually evidenced first by wasting of the dorsum of the thenar web and the dorsal interosseous spaces. Later the little finger assumes an abducted position and cannot be approximated to the ring finger. If the process is permitted to go on untreated complete ulnar paralysis may occur.

Surgical treatment should be employed just as early as the condition is recognized since often the best that can be hoped for from surgery in chronic palsy is cessation of the progress with relatively little reversal of the neurologic changes. Treatment consists in moving the ulnar nerve from its position in the olecranon groove to a position in the cubital fossa where it is no longer exposed to repeated stretch and easy trauma.

Median Nerve Compression in the Carpal Tunnel. The insidious development of median nerve palsy in the hand has sometimes been found to occur with osseous deformity of the wrist following fractures and has been appropriately regarded as tardy median palsy. However the carpal tunnel syndrome of median nerve palsy has come to attention in recent years. The pathogenesis of the syndrome is not entirely understood but it is believed the nerve somehow becomes irritated and swollen within its tunnel through the volar carpal ligament. The condition is more often seen in patients who have overindulged in some kind of unaccustomed manual work.

The clinical picture is characteristic. Sensory symptoms in the distribution of the median nerve in the hand and fingers constitute the earliest and major disability. Tingling, burning and sometimes painful dysesthesias are usual and, as numbness increases the patient finds increasing limitation in facile use of the fingers. Motor symptoms and signs are less prominent and may be absent. Moderate wasting and weakness occur in the opponens pollicis and abductor pollicis brevis muscles. A helpful sign in diagnosis is the elicitation of Tinel's sign on tapping over the median nerve at the wrist.

Rest and immobilization of the wrist are sometimes helpful in mild early cases, but in more advanced cases surgical decompression of the nerve by division of the carpal liga-

ment over the course of the nerve has proved to be highly successful.

CERVICAL RIB AND ANTERIOR SCALENE SYNDROME

A cervical rib is a supernumerary rib arising from the seventh cervical vertebra. It is accompanied by a number of variations in anatomic arrangement of the regional structures, including muscles, nerves and blood vessels. A relatively small percentage of those born with cervical ribs develop symptoms and, then, usually not till middle life.

The symptoms and signs resulting from the presence of a cervical rib are due to stretch and compression of the subclavian artery and components of the brachial plexus in the acute angle between the rib and the scalenus anticus muscle. The muscle is a very important part of the arrangement and this is emphasized by the fact that somewhat the same though less marked, symptoms occur at times in the absence of an extra rib the anterior scalene syndrome.

The characteristic symptoms include pain, disturbances in sensation, wasting of muscles and peripheral vascular changes. Paresthesias and sensory loss occur along the ulnar side of the forearm and hand. Pain of variable degree is experienced along the inner aspect of the arm and forearm and occasionally in the suprascapular region. Aggravation of pain occurs on rotation of the neck, depression of the shoulder or overhead stretching movements. The same motions may obliterate the radial pulse.

Motor signs include gradual muscle wasting and weakness in the hand in a pattern comparable to that of ulnar palsy. But in more severe cases the muscle changes simulate combined ulnar and median palsy. The peripheral vascular changes due to compression of the subclavian artery are not always present but can be of such severe degree that gangrene develops in one or more fingers.

The treatment consists of dividing the anterior scalenus muscle at its lowest attachment for alleviation of the anterior scalene syndrome. The same procedure may also be adequate for relief in the presence of a cervical rib but if the nerves of the plexus and the subclavian artery do not appear to have been fully released by division of the muscle the cervical rib is resected.

TUMORS

The commonest primary tumor of peripheral nerves is the neurofibroma. In you

The Autonomic Nervous System, The Neurosurgical Relief of Pain

By JAMES C WHITE M D

JAMES CLARKE WHITE was born in Vienna Austria where his father was studying dermatology His father's family originated in Maine dating back to their departure from Ireland after the siege of Londonderry His mother's forebears, too were New Englanders. Educated at Harvard University and its medical school he is Chief of the Neurosurgical Service of Massachusetts General Hospital His interest in the surgery of the sympathetic nervous system and treatment of chronic painful conditions has been long recognized, as has the scholarly presentation of his experiences

THE AUTONOMIC NERVOUS SYSTEM

Surgery of the autonomic nervous system did not develop with the earlier knowledge of its anatomy but with an understanding of the function of the craniosacral and thoracolumbar systems of nerves which has been built up by physiologists since the time of Claude Bernard

The modern neurosurgeon stands particularly in debt to W H Gaskell and J N Langley of Cambridge University to François-Franck of Paris and to Walter B Cannon of Harvard. He is especially indebted to the Cambridge physiologists for the knowledge of the finer arrangement of the autonomic neurons and also for the classification of these nerves into two functionally antagonistic systems the *craniosacral* or *para sympathetic* system on the one hand and the *thoracolumbar* or *sympathetic* system on the other. The latter leaves the spinal cord only between its first thoracic and second lumbar segments, while the former is given off from the brain stem and the sacral segments (Fig. 30)

Cannon is chiefly responsible for the investigation of the activity of the adrenal medulla. He also discovered that the sympathetic hormone liberated at the nerve endings on smooth muscle, which he called *sympathin* could not be epinephrine alone. His studies foreshadowed the recognition of the role of norepinephrine as a chemical mediator of nerve impulses in smooth mus-

cle, similar to the role of acetylcholine in striated muscle and the synaptic junctions in sympathetic ganglia.

While Gaskell and Langley were investigating the motor activity of the visceral nerves François-Franck first conceived of their importance in pain and urged surgeons to remove the cervical sympathetic chains for the relief of angina pectoris.

Surgeons of the present generation have applied these fundamental principles most successfully to cases of peripheral vascular disease, to the reduction of hypertension and to the relief of many types of visceral pain. By regarding each sympathectomy as an experiment in human physiology certain surgeons have contributed a practical understanding of the subject far beyond the limits of laboratory investigation.

Tests for Evaluating the Response to Sympathectomy* In addition to determining the degree of arterial occlusion by observation of pulsations in the major vessels of the extremities, the competence of collateral circulation by noting the degree of blanching on elevation, the rate of venous filling, the amount of cutaneous congestion on dependency and the demonstration of arterial obliteration by arteriography it is necessary to estimate the degree of superimposed vascular tone of neurogenic origin.

Editor's note: Dr Robert S Shaw Director of the Peripheral Vascular Research Laboratory at Massachusetts General Hospital, assisted in revising the text concerned with vasospastic disorders.

- Davidoff L. M. Pseudotumor cerebri Benign Intracranial Hypertension. *Neurology* 6:605, 1956.
- Davis, L. *Neurological Surgery* 4th ed. Philadelphia, Lea & Febiger, 1953.
- Davis, R. A., Wetzel, N., and Davis, L. An Analysis of the Results of Treatment of Intracranial Vascular Lesions by Carotid Artery Ligation. *Ann. Surg.* 143:641, 1955.
- Earl, K. M., Baldwin, M. and Penfield, W. Ictal Sclerosis and Temporal Lobe Seizures Produced by Hippocampal Heretation at Birth. *Arch. Neurol. & Psychiat.* 69:27, 1953.
- Elshberg, C. A. *Surgical Diseases of the Spinal Cord, Membranes and Nerve Roots*. New York, Paul B. Hoeber, 1941.
- Furrow, L. T. The Douleurcure of the Nervus Intermedius. *J.A.M.A.* 110:253, 1941.
- Grant, F. C. Results in the Treatment of Major Trigeminal Neuralgia. *Ann. Surg.* 107:14, 1938.
- Guklett, B. Tractotomy for the Relief of Trigeminal Neuralgia. Observations in 124 Cases. *J. Neurosurg.* 7:499, 1950.
- Hamby, W. B. *Intracranial Aneurysms*. Springfield, Ill., Charles C. Thomas, Publisher, 1932.
- Kahn, E. A., Bassett, R. C., Schneider, R. C. and Crosby, E. C. *Correlative Neurosurgery*. Springfield, Ill., Charles C. Thomas, Publisher, 1955.
- Kernohan, J. W., and Sayre, G. P. *Tumors of the Central Nervous System*. Washington, D. C., Armed Forces Institute of Pathology, 1953.
- Kristoff, F. V. and Odum, G. I. Ruptured Intervertebral Disk in the Cervical Region. A Report of 20 Cases. *Arch. Surg.* 54:287, 1947.
- Krynauw, R. A. Infantile Hemiplegia Treated by Removal of One Cerebral Hemisphere. *South African M. J.* 24:539, 1950.
- McKenzie, K. G., and Alexander, E. Restoration of Facial Function by Nerve Anastomosis. *Ann. Surg.* 132:411, 1950.
- Metzowsky, A. M. and Harsh, G. R. The Surgical Management of Cerebritis Complicating Penetrating Wounds of the Brain. *J. Neurosurg.* 10:373, 1953.
- Mettler, F. A., ed. *Selective Partial Ablation of Frontal Cortex: A Correlative Study of Its Effects on Human Psychotic Subjects by the Columbia Greyhound Associates*. New York, Paul B. Hoeber, 1949.
- Meyers, R. The Surgical Treatment of "Focal" Epilepsy: An Inquiry Into Current Premises, Their Implementation and the Criteria Employed in Reporting Results. *Epilepsia* 3:1, 1934.
- Oliverson, H., and Rives, J. Arteriovenous Aneurysms of the Brain. *Arch. Neurol. & Psychiat.* 39:867, 1948.
- Penfield, W., and Kristiansen, K. *Epileptic Seizure Patterns*. Springfield, Ill., Charles C. Thomas, Publisher, 1951.
- Peripheral Nerve Regeneration. A follow-up study of 3,656 World War II Injuries. Veterans Administration Medical Monograph*, 1956. Editors: B. Woodhall and G. W. Beebe, U. S. Govt. Printing Office, Washington, D. C.
- Pollock, L. J. *Extracranial Injuries of Multiple Cranial Nerves*. *Arch. Neurol. & Psychiat.* 31:523, 1920.
- Pollock, L. J., and Davis, L. *Peripheral Nerve Injuries*. New York, Paul B. Hoeber, 1933.
- Pollock, L. J., and others. *Electrodiagnosis of Lesions of Peripheral Nerves in Man*. *Arch. Neurol. & Psychiat.* 60:1, 1948.
- Poppert, J. L., and Martinez Niochet, A. Spasmodic Torticollis. *S. Clin. North America* 31:835, 1951.
- Ray, B. S. The Management of Intractable Pals by Posterior Rhizotomy. *A. Res. Nerv. & Ment. Dis. Proc.* 23:361, 1943.
- Ray, B. S. Differential Diagnosis between Ruptured Lumbar Intervertebral Disk and Certain Diseases of the Spinal and Peripheral Nervous Systems. *S. Clin. North America* 26:272, 1946.
- Ray, B. S. Lesions of the Cranial Nerves. In *Operative Techniques*, edited by W. H. Cole. New York, Appleton-Century-Crofts, 1948.
- Ray, B. S., and Dunbar, H. S. Thrombosis of the Dural Venous Sinuses as a Cause of Pseudo Tumor Cerebri. *Ann. Surg.* 134:378, 1951.
- Ray, B. S., and Parsons, H. Subdural Abscess Complicating Frontal Sinusitis. *Arch. Otolaryng.* 37:538, 1943.
- Ruge, D., Broecker, R., and Davis, L. A Study of the Treatment of 637 Patients with Trigeminal Neuralgia. *J. Neurosurg.* 15:523, 1958.
- Schneider, R. C. *Trauma to the Spine and Spinal Cord, Correlative Neurosurgery*. Springfield, Ill., Charles C. Thomas, Publisher, 1955.
- Seddon, H. J., ed. *Peripheral Nerve Injuries. Medical Research Council Special Report Series No. 262*. London, Her Majesty's Stationery Office, 1954.
- Sjogvist, O. Ten Years Experience with Trigeminal Tractotomy. *Brain-med.* 10:259, 1948.
- Spurling, R. C. *Lesions of the Lumbar Intervertebral Disc*. Springfield, Ill., Charles C. Thomas, 1953.
- Taamheij, P. Decompression of the Trigeminal Root and the Posterior Part of the Ganglion as Treatment in Trigeminal Neuralgia. Preliminary Communication. *J. Neurosurg.* 9:288, 1952.
- White, J. C., Sweet, W. H., Hawkins, R., and Niles, R. C. Anterolateral Cordotomy: Results, Complications and Causes of Failure. *Brain* 73:346, 1950.
- Wilson, A. A. Geniculate Neuralgia, Report of a Case Relieved by Intracranial Section of the Nerve of Wrisberg. *J. Neurosurg.* 7:473, 1950.
- Woltman, H., Kernohan, J., Adson, A., and Craig, W. *Intramedullary Tumors of the Spinal Cord and Gliomas of Intracanal Portion of Filum Terminale*. *Arch. Neurol. & Psychiat.* 65:378, 1951.
- Woodhall, B., and Beebe, G. W., eds. *Peripheral Nerve Regeneration, A Follow up Study of 3,656 World War II Injuries. Veterans Administration Medical Monograph*. Washington, U. S. Government Printing Office, 1956.

This initial steady state may be secured by one hour's exposure in an environment of 68° F and 50 per cent relative humidity. Peripheral autonomic activity is determined by observing the effect of body warming or nerve block on skin temperature and digital plethysmographic pulse in the exposed extremity. Sudomotor activity is determined by measuring electrical skin resistance.

Measurement of circulatory impairment produced by organic vascular occlusive disease requires the elimination as far as possible of circulatory decreases from vasoconstriction. The patient studied is warm and his peripheral vessels are maximally dilated. Study of limb circulation during the reactive hyperemia, after release of a tourniquet by a method such as venous occlusion plethysmography, allows the best determination of circulation without vasoconstriction.

Prediction of the degree of warming which can be expected after sympathetic denervation is accurate in Raynaud's disease when there is only a slight degree of endarteritis, although the initial increase in temperature of the hands will not be fully maintained. In the advanced case complicated by sclerodactylia, any increase in circulation is limited by the constricting fibrosis of the skin and subcutaneous tissue. In occlusive vascular disease when lumbar sympathectomy is so often helpful for relief from cold feet with burning discomfort, the predicted degree of improvement follows lumbar sympathetic ganglionectomy in seven out of ten individuals. In the others the permanent clinical response is likely to be somewhat better than that predicted. Measurement of cutaneous temperatures alone, together with clinical observation of color and sweating will permit as useful an evaluation as more complicated methods of recording digital pulsations.

For the investigation of obscure painful syndromes, such as causalgia, or unusual types of visceral pain, paravertebral infection can give a selective block of the sympathetic pathways. From the response to these tests it becomes possible to select certain patients for treatment by sympathectomy without recourse to the more mutilating procedures of cutting posterior roots or the spinothalamic pain pathways in the spinal cord. Procaine block constitutes the most accurate form of diagnostic test and should be used in all doubtful cases.

Vasospasm in Peripheral Vascular Disease. There is a wide variation in vascular tone in the hands and feet of individuals who have

no evidence of occlusive vascular disease. This ranges from the warm dry handed individual, whose extremities maintain brisk circulation in relatively cool surroundings to the person chronically afflicted with cool moist hands and feet. In certain young individuals who are often emotionally unstable the palms of the hands and soles of the feet are continuously wet with perspiration cold and are often cyanotic as well. This may persist even in a relatively warm environment. The hyperhidrosis is due to abnormal activity of the sympathetic sudomotor nerves the coldness and cyanosis of the extremities in part to evaporation of moisture as well as to excessive vasoconstriction. Both hyperhidrosis and acrocyanosis respond in a dramatic fashion to regional sympathetic denervation.

Raynaud's disease The condition first described by Maurice Raynaud in 1862 is characterized by phasic color changes of the fingers and, to a lesser extent, of the hands and feet as well. The extremities are sensitive on exposure to cold and even more so to general chilling of the body. This syndrome includes a number of conditions. The typical Raynaud's syndrome consists of blanching ischemia or cyanosis with numbness of the tips of the fingers. On warming, the digits develop a reddish hue with tingling, followed by return of normal color and sensation. As the condition advances there is a tapering of the fingers with the appearance of superficial ulcers at their tips, which are slow to heal. Sclerodermatous thickening of the skin and stiffness of the fingers may later complicate the picture leading to necrosis of the finger tips and a severely crippled hand. The condition most often develops in young adults, in women more often than men. It usually affects the hands, much more rarely the feet, in a remarkably symmetrical fashion with the thumbs much less involved than the other digits. The radial and pedal pulses are unaffected.

There are a number of factors which lead to the phasic color changes known as Raynaud's phenomenon most of which are related to organic vascular disease. It may be seen in various forms of endarteritis, as an early sign in rheumatoid arthritis, scleroderma, dermatomyositis, lupus, in cases of cervical rib with compression of the subclavian artery and in men who use vibrating tools. Sir Thomas Lewis, who contributed so much to our understanding of this condition, concluded that the primary disorder is hypersensitivity of smooth muscle in the

INNERVATION OF Periph Blood Vessels

INNERVATION OF VISCERA

Sensory and Motor
* indicates Conduction of pain

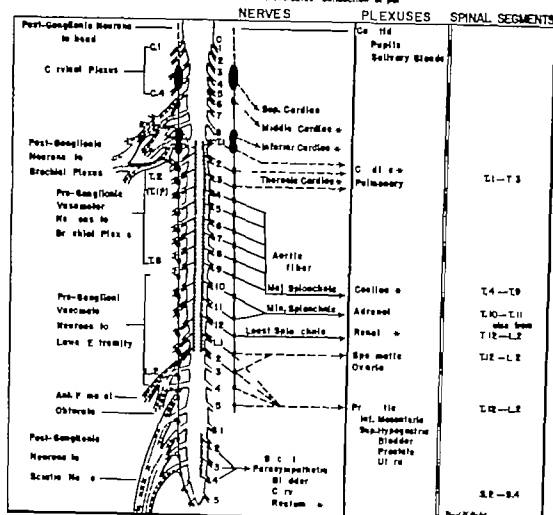


Figure 30 The outgoing vasomotor and somatomotor fibers to the extremities from the lateral horn of the spinal cord are illustrated diagrammatically on the left side. On the right side are shown the mixed visceromotor (sympathetic) and viscerosensory (posterior spinal root) fibers, together with the principal visceral plexuses which they supply and the spinal segments from which they are derived. Pre-ganglionic sympathetic axons are indicated by continuous lines and postganglionic by interrupted lines. Direct pre-ganglionic connections with the stellate ganglion via the first thoracic nerve are marked by dotted lines. Recent anatomic studies suggest that they are often present, possibly in C8 as well. The asterisk indicates the presence of pain fibers running to the posterior roots.

It should be pointed out that there is rarely an actual increase in sympathetic activity. This is seen only in a few younger individuals with cold, clammy cyanotic hands and feet. This condition usually occurs in emotionally unstable individuals and signifies abnormal function at the hypothalamic level. In the more common examples of Raynaud's phenomenon of blanching digital ischemia, and in all cases of obliterative vascular disease, the vasomotor tone is normal. Thus, however by no means precludes a valuable response to sympathectomy especially when the individual happens to have a high degree of normal tone. It is in these patients that laboratory tests to predict the

increase in warming of the cold, uncomfortable extremity prior to a contemplated sympathectomy are most important.

Evaluation of the degree of sympathetic vasomotor activity and particularly of the effect of various procedures or pharmacologic agents on vascular tone is complicated by the great lability and responsiveness of the autonomic nervous system to environmental stimuli, emotional states, digestive activity and many other factors. Study of the peripheral circulatory manifestations of autonomic activity therefore requires initial equilibration of the patient in a basal state in a quiet room with constant temperature and humidity.

This initial steady state may be secured by one hour's exposure in an environment of 68 F and 50 per cent relative humidity. Peripheral autonomic activity is determined by observing the effect of body warming or nerve block on skin temperature and digital plethymographic pulse in the exposed extremity. Sudomotor activity is determined by measuring electrical skin resistance.

Measurement of circulatory impairment produced by organic vascular occlusive disease requires the elimination as far as possible of circulatory decreases from vasoconstriction. The patient studied is warm and his peripheral vessels are maximally dilated. Study of limb circulation during the reactive hyperemia after release of a tourniquet by a method such as venous occlusion plethymography allows the best determination of circulation without vasoconstriction.

Prediction of the degree of warming which can be expected after sympathetic denervation is accurate in Raynaud's disease when there is only a slight degree of endarteritis although the initial increase in temperature of the hands will not be fully maintained. In the advanced case complicated by sclerodactylia, any increase in circulation is limited by the constricting fibrosis of the skin and subcutaneous tissue. In occlusive vascular disease, when lumbar sympathectomy is so often helpful for relief from cold feet with burning discomfort the predicted degree of improvement follows lumbar sympathetic ganglionectomy in seven out of ten individuals. In the others the permanent clinical response is likely to be somewhat better than that predicted. Measurement of cutaneous temperatures alone together with clinical observation of color and sweating will permit as useful an evaluation as more complicated methods of recording digital pulsations.

For the investigation of obscure painful syndromes, such as causalgia, or unusual types of visceral pain, paravertebral injection can give a selective block of the sympathetic pathways. From the response to these tests it becomes possible to select certain patients for treatment by sympathectomy without recourse to the more mutilating procedures of cutting posterior roots or the spinothalamic pain pathways in the spinal cord. Proctaine block constitutes the most accurate form of diagnostic test and should be used in all doubtful cases.

Vasospasm in Peripheral Vascular Disease. There is a wide variation in vascular tone in the hands and feet of individuals who have

no evidence of occlusive vascular disease. This ranges from the warm dry handed individual, whose extremities maintain brisk circulation in relatively cool surroundings to the person chronically afflicted with cool, moist hands and feet. In certain young individuals who are often emotionally unstable the palms of the hands and soles of the feet are continuously wet with perspiration, cold and are often cyanotic as well. This may persist even in a relatively warm environment. The hyperhidrosis is due to abnormal activity of the sympathetic sudomotor nerves the coldness and cyanosis of the extremities in part to evaporation of moisture as well as to excessive vasoconstriction. Both hyperhidrosis and acrocyanosis respond in a dramatic fashion to regional sympathetic denervation.

Raynaud's disease The condition first described by Maurice Raynaud in 1862 is characterized by phasic color changes of the fingers and to a lesser extent, of the hands and feet as well. The extremities are sensitive on exposure to cold and even more so to general chilling of the body. This syndrome includes a number of conditions. The typical Raynaud's syndrome consists of blanching ischemia or cyanosis with numbness of the tips of the fingers. On warming, the digits develop a reddish hue with tingling, followed by return of normal color and sensation. As the condition advances there is a tapering of the fingers with the appearance of superficial ulcers at their tips, which are slow to heal. Sclerodermatous thickening of the skin and stiffness of the fingers may later complicate the picture leading to necrosis of the finger tips and a severely crippled hand. The condition most often develops in young adults, in women more often than men. It usually affects the hands, much more rarely the feet, in a remarkably symmetrical fashion with the thumbs much less involved than the other digits. The radial and pedal pulses are unaffected.

There are a number of factors which lead to the phasic color changes known as Raynaud's phenomenon, most of which are related to organic vascular disease. It may be seen in various forms of endarteritis, as an early sign in rheumatoid arthritis, scleroderma, dermatomyositis, lupus, in cases of cervical rib with compression of the subclavian artery and in men who use vibrating tools. Sir Thomas Lewis, who contributed so much to our understanding of this condition, concluded that the primary disorder is hypersensitivity of smooth muscle in the

digital arteries to cold. It is still a debatable point whether pallid asphyxia of a digit can take place primarily from local susceptibility to cold or whether there must first be endarteritis with superimposed vasospasm. Biopsies have shown striking degrees of occlusion and recanalization of the digital arteries. It is certain, as Lewis pointed out, that neurogenic vasoconstriction of normal arteries can never produce blanching ischemia of a finger.

Occlusive vascular disease Patients with thromboangiitis obliterans and arteriosclerosis may benefit from release of neurogenic vasoconstriction if they have a high degree of normal vascular tone. Patients favorable for sympathectomy can be selected by appropriate clinical and laboratory examination, as described. It should be pointed out, however, that sympathectomy is less commonly used today because of the improvement in technique of reconstructive vascular surgery permitted by arteriography and arterial grafting.

Treatment of Peripheral Vascular Disease by Release of Neurogenic Vasoconstriction. There are no very effective vasodilator drugs for therapeutic use. Raudixin is helpful in 50-mg. doses twice daily especially in the group of younger emotionally unstable individuals, as are estrogens in women at the time of menopause. Priscoline has also been recommended but is ineffective in severe cases. In using ganglionic blocking agents it must be remembered that any generalized vasodilatation may do more harm than good by depriving an extremity with occlusive vascular disease of blood by shunting the blood to other areas of lower vascular resistance.

When patients with favorable cases of peripheral vascular disease have given up smoking, have failed to respond to medication and cannot be protected by wearing warm clothing, mittens and woolen socks, they may often be helped by sympathetic denervation.

Sympathectomy has the great advantage that it can produce relatively local release of arterial tone where it is most needed. The operation must produce complete interruption of the vasoconstrictor fibers to the part involved. This requires an attack on the paravertebral chains of sympathetic ganglia which run along the anterolateral aspect of the thoracic and lumbar vertebrae.

Operative measures designed to interrupt vasoconstrictor tone must take into account the extraordinary proclivity of these fibers,

both preganglionic and postganglionic, to regenerate. Essentially complete recovery of vasoconstrictor tone in the upper extremities occurred in a patient submitted nine years previously to removal of the ganglia from the inferior cervical through the second thoracic on the right side and a preganglionic sympathectomy as described by Smithwick on the left. Time and again regeneration of vasomotor and sudomotor fibers has been observed in the upper extremity and even in the legs when inadequate lengths of lumbar ganglionic chain were removed. Proof that these recurrences are due to regeneration is given by the return of vasodilatation which occurs following procaine block or a secondary more radical sympathectomy.

It has been stated that the phenomenon of sensitization of denervated smooth muscle in the arterial walls to circulating epinephrine prevents satisfactory lasting vasodilatation after cervicothoracic ganglionectomy as this operation results in degeneration of the postganglionic network of vasoconstrictor neurons. While this general principle of sensitization to epinephrine after postganglionic denervation, which was described by Cannon and Rosenblueth, is undoubtedly true in experimental animals, it is relatively unimportant in man. For this reason, the so-called Telford-Smithwick form of preganglionic sympathectomy for increasing circulation in the upper extremity is no more effective than removal of the ganglia, except the patient is spared the mild disfigurement of a Horner's syndrome. Experience with this operation has shown that regeneration of vasoconstrictor fibers is likely to take place. A second objection is that recent anatomic evidence strongly points to the possibility of sympathetic rami containing vasoconstrictor fibers leaving the cord via the first thoracic and possibly in the eighth cervical nerve as well. Some fibers appear to reach the brachial plexus directly through the stellate ganglion, probably also through the middle cervical and vertebral ganglia, as well as coming up from lower levels (Fig. 30). The really essential point is to remove a considerable length of the ganglionated chain, as far down as the fourth rib and preferably upward above the stellate ganglion to include the vertebral and middle cervical ganglia, which lie in relation to the subclavian artery. This operation may be carried out extra-pleurally by resecting the central ends of the first and second ribs or by using a supraclavicular approach. The latter gives the surgeon a better opportunity to remove

the upper portion of the stellate and the small ganglia around the subclavian and origin of the vertebral artery.

For the lower extremity, the second, third and fourth lumbar ganglia should be resected. This is best carried out posteriorly through a modified renal incision. The retroperitoneal space behind the cecum or sigmoid is entered and the lumbar chain is exposed in the gutter formed by the outer edge of the vena cava or aorta, the vertebrae and the psoas muscle.

In experienced hands the risk from thoracic and lumbar ganglionectomy is remarkably slight. The chief essentials to justify surgical intervention are accurate diagnosis and demonstration by procaine injection or other diagnostic procedures that an adequate degree of vasodilatation will ensue. In these circumstances thoroughly satisfactory and lasting improvement has followed resection of the lumbar ganglia in a large series of patients with Raynaud's disease treated at the Massachusetts General Hospital. Surgical attempts to achieve a lasting effective vasoconstrictor denervation of the upper extremity have been beset with many difficulties particularly by regeneration after preganglionic operations or limited ganglionectomy and also because Raynaud's disease in the hands is likely to be more severe than in the feet, as a result of complicating obliterative arteriolar disease and secondary fibrosis of the skin and subcutaneous tissue. Really satisfactory results may therefore be anticipated only in patients with the early uncomplicated form of the disease. In patients with severe attacks of digital blanching and ischemia or in those with advanced scleroderma in whom endarteritic changes have already occurred a really satisfactory permanent recovery of circulation cannot be expected.

Young men with occlusive vascular disease due to thromboangiitis obliterans (Buerger's disease) may derive much benefit from the elimination of superimposed neurogenic constriction, especially those with increased sweating and an active vasomotor tone. These individuals can be selected by appropriate tests. Improvement in circulation generally turns out to be better than the predicted degree. This gives considerable relief from cold extremities and related pain in most cases. It should be pointed out, however, that follow up studies have demonstrated no advantage in collateral circulatory development, in blood flow through the muscles or in measured ability to walk with

out claudication. The resultant vasodilatation occurs mainly in the cutaneous vessels and the digits. In the absence of actual gangrene but with a critically low pedal circulation lumbar sympathetic ganglionectomy should often be performed in the younger group of patients, in whom reconstructive arterial surgical procedures are not feasible, even if all the major arteries have been obliterated. A significant number of major amputations may thus be avoided. However, in the presence of painful digital ulceration or incipient gangrene it is better first to crush the peripheral nerves above the ankle. This operation is far more effective for relieving pain and can be counted on to cause maximal vasodilatation for a period of several weeks. If adequate collateral circulation fails to develop lumbar ganglionectomy can be carried out at a later date with the assurance of producing a corresponding degree of vasodilatation which is likely to be permanent.

Lumbar ganglionectomy has also been used with increasing frequency in recent years in arteriosclerosis, in both its senile and diabetic forms, especially when abnormal sweating and cyanosis indicate a high degree of vasoconstrictor tone. Selection of patients must be made with extreme care, because in some with critically low peripheral circulation gangrene has been precipitated by sympathectomy. It should be emphasized that sympathectomy is of little benefit in cases of rapidly advancing arterial occlusion whether for relief of pain for improvement of deep ulceration or for increasing walking tolerance in cases of intermittent claudication. Individuals with atheromatous occlusion at the aortic bifurcation and others with iliac, femoral or popliteal occlusion should all be submitted to arteriography and treated by arterial grafting whenever limited obliteration makes this feasible.

Prophylactic Sympathectomy in Acute Arterial Occlusion. Sudden occlusion of a main artery to an extremity by embolism, trauma or ligature may lead to gangrene. The risk of this complication is greater in the leg than the arm as its collateral circulation is less competent. In the case of popliteal aneurysms, reconstructive surgery by means of arterial grafting is now a practicable sub-

To secure the maximal increase in circulation in the upper leg, the first lumbar ganglion should be removed along with the second and third, but if this is done on both sides, the power of ejaculation may be lost and the male patient rendered sterile but not usually impotent.

stitute for ligation and excision or for endoaneurysmorrhaphy. In the cases of embolism or penetrating arterial wounds with threatened circulatory insufficiency in the arm or leg, therapeutic efforts should be directed at restoring major vessel continuity whenever this is possible. Some protection however may be gained against critical reduction in collateral blood flow by repeated injections of the stellate or lumbar ganglia with Xylocaine or by means of a fine polythene catheter inserted through a 17 gauge needle. Continuous drip infiltration may thereby be safely maintained over a period of several days if small amounts of penicillin are added to the anesthetic solution.

Pain in Extremities. Pain in the forearm and hand or in the ankle and foot after trauma and amputation of digits is a rare but serious complication.

This usually takes the form of cutaneous hyperalgesia in the extremity or a deep aching or burning sensation which runs up the limb. There is often a patchy decalcification of the bones in the hand or foot. This condition is most commonly seen in individuals with cold cyanotic, moist extremities and can often be relieved by interruption of the underlying *vasospastic state*. Even temporary interruption of sympathetic impulses by paravertebral procaine block of the regional sympathetic ganglia may clear it up especially if repeated on a number of occasions. When pain is relieved, but recurs as the vasodilator response to procaine disappears, thoracic or lumbar sympathectomy can be counted on to give lasting relief. It is evident that the pain is not transmitted by the sympathetic nervous system but that unknown physiologic factors which produce it are activated by the efferent sympathetic discharge.

This is likewise true of another post-traumatic state which came to attention during World War II because of the intense suffering and incapacity which it produces. Typical *causalgia*, as described by Weir Mitchell in 1884, consists of dysesthesia and burning pain with extraordinary exacerbations aroused by emotional stimuli. It develops after partial injury of a peripheral nerve from a penetrating wound. Experience by military surgeons has shown that diagnostic procaine injection followed by regional sympathectomy has resulted in effective relief in the great number of cases reported during the last war. A critical review of sixty-two patients suffering from *causalgia* revealed that they were effectively relieved

of their burning pain for periods of up to seven years, with the exception of a few improperly selected individuals and those with inadequate sympathetic denervation.

The effect of sympathectomy on post-traumatic pain from the extremities is not known. In contrast to visceral innervation, the sympathetic does not appear to be an accessory afferent pathway for pain arising from injury below the shoulder or groin. Only visceral sensation can be evoked on stimulation of sympathetic ganglia in conscious patients, except in *causalgic states*. It seems most likely that *causalgic pain* is produced by efferent sympathetic impulses short-circuiting in the area of the injured nerve to the somatic sensory fibers.

The Autonomic Nervous System and the Regulation of Blood Pressure. The regulation of blood pressure for the varying demands of the body on changes in external and internal environment is mediated by the autonomic nervous system. The central control lies in groups of cells situated in the medulla and diencephalon. Trauma, encephalitis and tumors which injure these areas occasionally produce profound changes in the vasomotor tone.

Carotid sinus syndrome. An important peripheral mechanism for the reflex regulation of blood pressure and heart rate has been found to lie in the aortic nerves and in the carotid sinus. The latter which has nervous connections with the vagus, the cervical sympathetic and the medulla through a branch of the ninth cranial nerve, may become hypersensitive and produce an interesting clinical syndrome. Depending on the efferent pathway involved, three forms of this carotid sinus syndrome are found, carotid-inhibitory vasodilator and cerebral attacks with convulsions. The first type is due to a vagal reflex and can be abolished by atropine, the fall in arterial tension can be prevented by ephedrine, but the cerebral variety can be treated only by denervation of the sinus. This can be carried out either by local periarterial stripping of the carotid bifurcation or by intracranial section of the glossopharyngeal root. Numerous patients with spontaneous epileptic seizures induced by slight pressure on an irritable sinus have been relieved by operation.

Essential hypertension. Aside from rare conditions such as pheochromocytoma, occlusive disease of the renal artery, Cushing's syndrome and coarctation of the aorta, the etiology of hypertension remains unknown. In the common essential form of hyperten-

sion significant lowering of the blood pressure relief of symptoms and modification of degenerative vascular changes have been achieved in the last twenty years by surgery diet and drugs. Following the lead of Adson Peet, and Smithwick extensive resection of sympathetic ganglia and splanchnic nerves has been widely used, though the exact physiologic mechanism by which lowering of the blood pressure may result is unknown. There is no evidence that essential hypertension is related to an abnormal increase of vasoconstrictor activity.

The operations formerly advocated fell into three main groups: bilateral one-stage supradiaphragmatic ganglionectomy and splanchnicectomy subdiaphragmatic resection of the splanchnic nerves and upper lumbar ganglia and the combined, more radical thoracolumbar denervation. The last requires a two-stage operation, performed first on the right side, then on the left. Even more radical operations, by the thoracic transpleural and transdiaphragmatic route or by removal of a portion of the eighth and twelfth ribs with extrapleural and extraperitoneal exposure of the paravertebral trunks, have permitted resection of the entire vasoconstrictor outflow from the upper thorax down through the third lumbar ganglia. Surgical experience has demonstrated that thoracolumbar denervation is in general the most satisfactory procedure and that more radical sympathectomies are likely to be followed by unpleasant complications without a significantly greater or more lasting reduction of blood pressure.

Of suitably selected patients under fifty years of age, and preferably under forty years, who are free from advanced cerebral, coronary and renal damage and who still have a labile blood pressure which falls with bed rest and sedation, it is to be expected that over 80 per cent will have satisfactory symptomatic relief and somewhat under 50 per cent a worthwhile early reduction in blood pressure. Smithwick has reported a significant improvement in retinal lesions, reduction in cardiac hypertrophy with reversal of abnormal electrocardiographic patterns and a notable decrease in headache. The operative mortality is low less than 3 per cent. On the other hand, the operation is followed by much discomfort and requires a long period of convalescence. Long-range studies of the earlier patients operated upon at the Massachusetts General Hospital have shown that less than a fourth

maintained a blood pressure below the critical range after three to five years. Even among the group whose blood pressure has fallen to a normal range for one or more years many have subsequently returned with recurrent hypertension and serious cerebrovascular coronary or renal complications.

In recent years an impressive number of drugs have been developed that lower blood pressure reverse organic vascular changes and relieve symptoms with a high degree of effectiveness. These comprise the derivatives of the *Rauwolfia serpentina* group of alkaloids singly or in various combinations with chlorothiazide and other diuretics, and hydralazine ganglionic blocking agents. Medication with these promising new drugs has almost entirely superseded surgical and dietary treatment. Sympathectomy may now be recommended only for the rapidly advancing malignant variety of hypertension with papilledema in the period before irreversible renal damage has set in.

Other patients in whom operation may be worth while are those who will not or cannot follow the exacting details of medical treatment and a few individuals with angina pectoris or resistant tachycardia. The two latter conditions derive a specific benefit from transthoracic sympathectomy if the entire thoracic chain is removed on each side up through the first thoracic ganglion, as this high denervation interrupts the cardiac sensory and accelerator impulses. As a result of the increasing effectiveness of medical therapy surgical intervention in hypertension is now rarely undertaken.

Sympathectomy in the Treatment of Disordered Visceromotor Activity. Correction of states of abnormal activity on the part of the autonomic nervous system is possible in certain rare but incapacitating conditions. Two of these, hyperhidrosis of nervous origin and tachycardia, are of special interest.

Hyperhidrosis of nervous origin. Excessive sweating limited to the palmar surfaces of the hands and soles of the feet is occasionally seen in young, nervous persons of either sex. It differs from perspiration in a hot environment, in which the whole surface of the body is involved except the palmar and plantar surfaces and is distinctly a reaction on the part of the sympathetic system to psychogenic stimuli. This type of sweating may become so intense under nervous strain that beads of water drip from the fingers. As a result it becomes embarrassing to shake hands, delicate materials

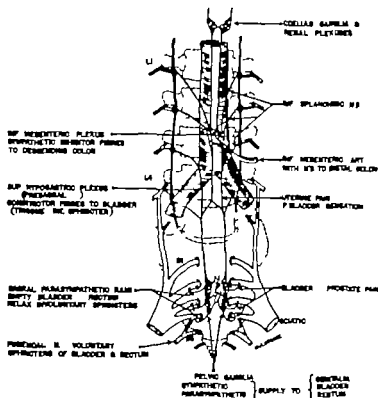


Figure 31 The nerve supply of the pelvic viscera.

are wet in handling and when rubber gloves are worn they become filled with sweat. The same condition may also involve the feet, with the result that the socks and even the shoes are constantly wet through. In these circumstances the skin becomes foul smelling and macerated and is therefore a prey to fungus infections. Psychotherapy, radiation or local applications fail to give relief but the results of sympathectomy are usually satisfactory and frequently give dramatic psychic as well as local benefit. Only the second and third thoracic or the corresponding lumbar ganglia need be removed. It is inadvisable to perform sympathectomy for both upper and lower extremities. This may lead to excessive sweating over the chest and abdomen, which can be as annoying as the original condition.

Control of tachycardia by sympathectomy
Experiments on animals have suggested that many ectopic cardiac rhythms are produced by epinephrine and abnormal activity on the part of the cardiac accelerator nerves. Clinical application of this work has demonstrated that upper thoracic sympathectomy is an effective method of controlling recurrent bouts of paroxysmal atrial tachycardia and fibrillation which cannot be stopped by medication. After bilateral resection of the upper four thoracic ganglia, the frequency and severity of these attacks are greatly reduced, but the pa-

tients may continue to experience asystole and occasional episodes of tachycardia on emotional strain, excessive fatigue or other illness. The attacks, which were formerly so frequent, severe and resistant to medication that the victim was totally incapacitated, become infrequent, mild and readily controlled by quinidine. The majority of patients are able to lead normal lives.

No untoward results have followed complete sympathetic denervation of the heart, as it can still accelerate on reduction of vagal tone or through the chemical response to epinephrine.

In a number of neurogenic disorders associated with impaired emptying of the bladder and rectum, release of "sympathetic inhibition" was formerly advocated. Further investigation has shown that detrusor reflexes are in no way influenced by the pelvic sympathetic nerves in the inferior mesenteric and superior hypogastric plexuses (Fig. 31). Control of micturition and defecation is mediated entirely by the parasympathetic sacral nerves. There is no effective neurosurgical operation for the so-called cord bladder (Megacolon), for which sympathectomy was often performed with slight benefit, can now be cured by resection of the paralyzed rectosigmoidal portion of the large bowel.

It is interesting to recall that cervical and

lumbar ramisection proposed by Royle and Hunter for the reduction of muscular tone although not generally accepted for the treatment of this particular condition led directly to the present operations which are so frequently utilized in peripheral vascular disease. With the possible exception of the much discussed subject of hypertension experience with sympathetic neurosurgery in the conditions discussed has now reached a point where the effects of operation are well known and its therapeutic value in properly selected cases is thoroughly established.

NEUROSURGICAL RELIEF OF PAIN

Severe pain from any source if persistent soon becomes a major problem to both its victim and his physician. There is a group of patients in whom there is no possibility of eliminating the cause of the pain and no other recourse save resort to narcotic drugs or interruption of sensory pathways. Narcotic medication is justifiable only when the patient has a fatal illness advanced to the point that he has but a month or two to live. Relief by neurosurgery has now reached the point where its risks and possible complications are distinctly less than those of narcotic addiction. In patients with malignant disease it is therefore best to consider desensitization at about the time the sufferer first demands medication stronger than codeine but yet has a life expectancy of several months or more. In nonmalignant neuralgias it is important to be sure that the patient has an underlying organic cause for his pain and is not psychoneurotic.

Spinothalamic Tractotomy The fiber tract

which carries pain from the skin deep tissues and viscera runs in the opposite anterior quadrant of the spinal cord. It is most accessible to the surgeon's knife at the level of the second thoracic or second cervical vertebra, where only a very limited laminectomy is required to make a small opening in the dura snip an attachment of the dentate ligament and rotate the cord by this convenient handle. The technique of anterolateral section is illustrated in Figure 32. The pain fibers vary greatly in their distribution, sometimes being relatively compact and superficially placed between the insertion of the dentate ligament and the origin of the anterior spinal roots. This convenient arrangement, commonly illustrated in anatomic textbooks is unfortunately by no means the general rule. In order to obtain consistently a high level of analgesia it is best to make as complete a transection of the anterior quadrant as possible with due respect to the corticospinal tract posteriorly and the anterior spinal artery in the midline ventrally and then to test the level of sensory loss on the operating table. This requires local anesthesia, which may be supplemented by light Pentothal sodium and nitrous oxide anesthesia, allowing time for the patient to awaken for testing.

When this operation has been properly performed, it should result in satisfactory loss of sensibility to pain in 90 per cent of patients. After high thoracic cordotomy this level usually reaches to the xiphoid, at times as high as the nipple, but sometimes only to the umbilicus. Analgesia will not remain complete in an additional 10 to 15 per

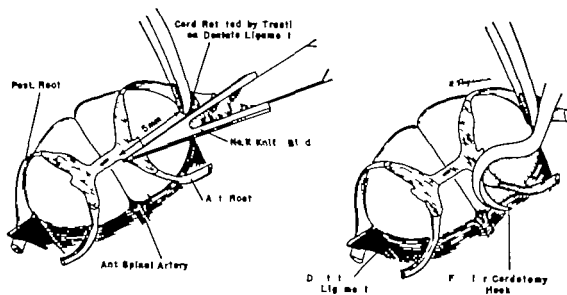


Figure 32. Technique of anterolateral cordotomy at upper thoracic level.

cent of individuals over the course of weeks or months, but relief of pain will be permanent in the remaining 75 per cent. After high cervical cordotomy maintenance of effective analgesia above the upper thoracic level cannot be assured in a high proportion of the subjects. We have been able to maintain relief from pain of brachial plexus distribution in less than half our subjects. A slightly higher level of analgesia can be gained by tractotomy in the medulla, although at an appreciably greater risk. If the pain tract is cut in the mesencephalon, analgesia may be produced over the entire opposite side of the body but at considerable risk of mortality and also of burning dysesthesia which resembles the thalamic syndrome.

When the pain tract is cut unilaterally at the level of the second cervical or second thoracic vertebra, mortality is slight and very few patients will suffer lasting motor or bladder impairment. On the other hand, when bilateral transection is required in malignant disease prolonged urinary retention will be a problem in about 30 per cent of the subjects. This complication is particularly likely to occur when there is pre-existent injury of the detrusor innervation. Other disagreeable sequelae are few as almost no useful sensation is lost save perception of heat and cold.

Experience in 165 cordotomies has taught us that pain in malignant disease is relieved in over 70 per cent of the patients with the exception of those with cancer of the breast and lung. In these the high level of pain and the tendency for the brachial plexus to be invaded make terminal relief a difficult problem. The failures in 30 per cent of our patients who have had cordotomy have been due to incomplete division of the pain fibers or spread of pain into nonanalgesic areas. In nonmalignant conditions, such as the lightning pains of the tabetic, the neuralgias of the lower extremity which follow gunshot wounds of the cauda equina surgical incisions and amputations pain has been controlled in 73 per cent of the patients. Contrary to the experience of those who make more limited incisions in the anterolateral quadrant, we have had impressive success even in patients with phantom pain in the ghost foot or hand has rarely persisted when analgesia has remained complete. Even during high analgesia, however has been difficult to achieve in the upper extremity especially in patients who have had shoulder amputations.

Posterior Rhizotomy Division of posterior spinal roots interrupts all forms of sensation. As a result, the denervated area feels numb if the sensory roots of the brachial or lumbosacral plexus are cut, the anesthetic limb will become a useless appendage lost in space the desensitized bladder is atonic and incontinent. Furthermore, even if appropriate anesthesia is produced, pain sometimes continues. This is the case with postherpetic pain, yet this condition, when the painful scars are favorably situated in the trunk or a lower extremity can be relieved by cordotomy if an adequate level of analgesia is obtained. For these reasons, section of posterior spinal roots has limited usefulness. It can be counted on to relieve angina pectoris and pain of carcinomatous invasion of cervical nodes and also the postoperative intercostal neuralgia which occasionally follows thoracotomy. In the latter condition, at least two sensory roots must be cut above and below the scarred nerve in order to avoid sensory overlap.

Section of Cranial Sensory Nerves. Section of cranial sensory nerves is much more effective and useful than spinal rhizotomy. Pain is carried from the face, nasal passages, internal ear and oropharynx by the trigeminal, nervus intermedius (sensory component of the seventh nerve) glossopharyngeal and vagus nerves. These structures may be cut on one side without too great handicap, except for the lowest vagal rootlets which innervate the muscles concerned with swallowing, closure of the glottis and vocal cords.

Pain in malignant disease of the pharynx. When the posterior tongue, tonsillar region or upper pharynx is invaded by cancer pain may be persistent and severe. It can often be relieved by section of the sensory roots of the fifth, ninth and tenth cranial nerves in the posterior fossa, but when pain is felt deep in the ear it is best to cut the nervus intermedius as well and the upper three cervical posterior roots if the lymph nodes in the neck are involved. Intramedullary section of the descending trigeminal root is an alternate procedure and is often successful when the lower cranial nerves are invaded by malignant disease. In order to interrupt the pain fibers of the seventh, ninth and tenth cranial nerves which accompany the fifth in this descending bulbar tract, it is necessary to make the incision into the medulla at or slightly above the level of the obex (the lower end of the fourth ventricle) and cut to a depth of 8.5 mm. between the nucleus gracilis dorsally and the spinotha-

lamic tract ventrally. Section of the descending bulbar tract should deliberately include a few of the contiguous fibers of each of these structures. A cut of this extent should be followed by some evidence of spinothalamic tract injury viz., slight contralateral hypalgesia of the torso and extremities, in addition to analgesia of the ipsilateral face and oropharynx, but no signs of posterior column involvement. At this level of the medulla some of the fibers entering the retiform body may be injured and the patient may develop a mild degree of lateropulsion. This is usually of brief duration, while the ensuing interruption of pain fibers from the fifth and lower cranial nerves is satisfactory and enduring. It is advisable to carry out this procedure with the patient under local anesthesia with supplementary Pentothal sodium and nitrous oxide anesthesia so that he can be awakened and tested after cutting the tract.

Frontal Lobotomy In patients with certain types of pain specific sensory denervation is extremely difficult, if not impossible. The conditions in which this is most often the case include atypical facial neuralgia, the Pancoast syndrome of carcinoma of the thoracic apex and malignant conditions involving both sides of the pharynx or neck. Other unfortunate individuals in the late stage of malignant disease, in addition to their pain, may suffer extreme mental anguish from the fear of imminent exanguination or strangulation and from the disfigurement and the foul odor of sloughing tissue. In terminal malignant disease, unilateral complete transection of the frontal white matter as proposed by Scarff may give gratifying relief without serious psychologic impairment. As this improvement rarely lasts for more than six months, it is not suitable for atypical facial neuralgias or other benign conditions.

Bilateral frontal lobotomy which stops mental suffering as well as pain, can only be justified if done in a strictly limited way. As originally advocated by Freeman and Watts it results in such grave psychologic deterioration that the nonpsychotic individual will become a hopeless vegetable no longer interested in his friends or able to associate with others. This operation, therefore, cannot be recommended for any person who may survive for a prolonged period or hopes to lead a useful life. For these individuals, Grantham and Spurling have proposed limited destruction of the medial inferior white matter in front of the anterior

tips of the ventricles on both sides by electrocoagulation increasing the area of destruction in stages to the point where the patient is freed from fear of his disease and concern about his suffering.

This procedure has been modified recently by implanting fine wire electrodes under roentgenographic control in the inferior medial quadrants of white matter in each frontal lobe. These are left in place for several weeks while the frontal association fibers are coagulated by radiofrequency current. This is applied in stages as the electrodes are withdrawn with the patient under careful observation. By this means it is often possible to carry out a graded leukotomy just sufficient to eliminate pain and all need for narcotics as well as the fear of imminent death, without serious psychologic deterioration.

Sympathectomy Pain from the heart, aorta and abdominal viscera can be relieved with a high rate of success by regional sympathetic denervation. In order to obtain satisfactory results, the pathologic process must be confined within the capsule of the organ involved. Malignant disease of the lung, gastrointestinal or genitourinary tracts generally is not painful until it has invaded the parietal pleura, peritoneum, bone or the major spinal nerve plexuses. In these circumstances the appropriate posterior spinal roots or the spinothalamic tract must be sectioned.

The classic conception of the autonomic nervous system was that its preganglionic and postganglionic neurons were purely regulators of visceral activity. It is now generally conceded that many afferent fibers capable of transmitting pain and of activating visceral reflexes reach the posterior sensory root system from the cardiac, splanchnic and mesenteric plexuses.* The fibers transmitting visceral pain pass through the paravertebral ganglia, the white rami communicantes and the mixed spinal nerves to reach their cells of origin in the posterior root ganglia. Their dendrites end in the posterior horn of spinal gray matter where they establish synapses with the secondary sensory neurons that decussate in the cord and ascend to the brain in the opposite spinothalamic tract.

Studies of the conduction of pain from the internal organs have demonstrated two main types of afferent conduction.

1. Referred pain of the type described by

On numerous occasions White and Sweet have evoked diffuse abdominal pain on stimulating lumbar ganglia or splanchnic nerves in patients at operation under regional block anesthesia.

Head and Mackenzie This theory assumes that visceral afferent impulses run only as far as the posterior horn of the spinal cord. It is further postulated that impulses reaching this region from a diseased organ set up an irritable focus and thereby reduce the sensory threshold to stimuli from the surface of the body. As a result, the constant bombardment of afferent impulses from the skin which normally fails to reach the level of consciousness, becomes magnified into painful sensation. This type of pain is sharp; it is not necessarily localized over the organ in question but is often referred to such a distant area as the medial side of the arm in angina pectoris. This occurs because some of the cardiac rami enter the spinal cord at the same level as the ulnar and medial brachio-cutaneous nerves.

2. Direct transmission of visceral pain Over sixty years ago Ross wrote that in addition to sharp, well localized referred pain, a diseased organ might also cause a deep, ill-defined sense of discomfort. He called the latter splanchnic or direct visceral pain. This important contribution was lost sight of in later years but there is good evidence that visceral pain may reach the thalamus without any referred cutaneous mechanism, because it can be evoked in animals and in human beings after all cutaneous afferent impulses have been interrupted. This direct type of sensory conduction presumably carries the dull aching form of visceral pain which is vaguely localized in the region of the diseased organ. Lewis has shown that pain from all deep structures, whether skeletal muscle, fascia, periosteum or an internal organ, is fundamentally alike. It is of the slow-conduction type and poorly localized.

The reason for this is the paucity of sensory nerve endings in the viscera as well as in all other deep structures except periosteum. Accurate localization of painful stimuli is present in the skin, and to a lesser extent in the periosteum, because of the large number of overlapping sensory terminals. There is also evidence that there are not sufficient numbers of secondary pain fibers in the spinothalamic tract to supply all the primary neurons in the posterior root system. Hence it seems probable that many primary visceral and cutaneous afferent fibers must share secondary spinothalamic neurons in common, thus accounting for the frequent reference of visceral pain to surface areas which receive their innervation over similar posterior spinal roots.

In the last analysis there is no fundamen-

tal difference between sensation of pain of visceral origin and from deep somatic structures.

Whenever an inflammatory process spreads from a viscus to the neighboring parietal pleura or peritoneum, the intercostal nerves become the chief pathway for conduction of pain, e.g. in pleurisy and appendicitis or cholecystitis with local peritoneal involvement. This is also the case in carcinoma of the abdominal viscera. As long as the disease remains confined within the visceral capsule, interruption of sympathetic innervation will stop pain but once the retroperitoneal nerves and lumbosacral plexuses become involved the posterior spinal roots or spinothalamic tract will have to be severed.

Pain from the bladder, prostate, cervix, uteri and rectum differs from that arising from the abdominal viscera. It is transmitted over the inferior hypogastric plexuses and posterior roots of the second to fourth sacral nerves (Fig. 31). While no form of pain from the heart, pulmonary parenchyma or abdominal organs is conducted over the cranial portion of the parasympathetic outflow, the vagus carries sensation from the esophagus, trachea and main bronchi.

From a knowledge of these mechanisms it is obvious that pain from the heart and abdominal organs can be interrupted by destroying either the regional sympathetic ganglia or their rami. Section of the posterior spinal roots or section of the contralateral spinothalamic tract will also produce a loss of visceral sensation, but at the risk of more serious complications and loss of cutaneous sensibility, whereas sympathectomy is accompanied only by regional vasomotor and sudomotor paralysis.

Table 4 summarizes the segmental afferent innervation of the thoracic and abdominal organs. The affected segments are easily identified by paravertebral procaine injection in patients with intractable visceral pain. When the ganglia through which the pain is transmitted have been accurately located, they can be resected surgically or destroyed by alcohol injection.

The method of treating cardiac pain by paravertebral alcohol injection is the safest method of treating pain in patients with angina pectoris or aortic aneurysm but accurate resection is technically difficult to achieve in about 10 per cent of the subjects. Furthermore, in a small number of patients the alcohol sets up irritating neuritis which may last for a number of weeks. The advantages of injection therapy for the patient

Table 4. Sensory Innervation of the Viscera

ORGAN	SUPERFICIAL AREAS TO WHICH PAIN IS REFERRED	VISCERAL RANGI WHICH CARRY PAIN
Heart	Precordium and inner arm	T_1-T_4 or T_1
Lung	No evidence of pain until parietal pleura and intercostal nerves are involved	
Liver and gallbladder	Right upper quadrant distress with pain referred to right scapular region	T_7-T_9 T_7-T_9
Pancreas	Epigastrium and midback	T_7-T_9
Stomach	Epigastric region	T_9-T_{11}
Small intestine	Umbilical region	$T_{11}-T_{12}$, also S_1-S_2
Colon and rectum	Suprapubic region	$T_{12}-L_4$
Kidney	Loins and groin	L_1-L_2
Ureter	Loins and groin	S_1-S_2
Bladder	Suprapubic region, penis and perineum	$T_{12}-L_4$
Uterus	Suprapubic region and lower back	

who is a poor risk so far outweigh the disadvantages that this method should be used for patients with advanced cardioaortic disease. If it fails to give sufficient relief posterior root section may be undertaken later. For the patient with angina pectoris who is a fair surgical risk, resection of the upper four thoracic sympathetic ganglia is the ideal surgical procedure. In the case of cardiac pain it has been shown that the direct thoracic cardiac nerves, which connect the upper thoracic sympathetic ganglia with the heart, account for the frequent failures of the older operation of cervical sympathectomy. When these ganglia or the upper four posterior thoracic spinal roots are destroyed, pain from the heart is consistently interrupted. White and Bland have reviewed results in eighty five patients with medically intractable angina pectoris who have been treated by paravertebral block or upper thoracic ganglionectomy. These statistics and the results recorded by Lindgren and Olivecrona from their series of seventy three upper thoracic ganglionectomies demonstrate the effectiveness of sensory denervation of the heart.

In addition to its effectiveness in the relief of otherwise intractable cases of angina pectoris regional sympathetic denervation has proved to be equally valuable in the treatment of numerous other forms of visceral pain, such as may be encountered with

Although pain in the precordium and arm is consistently relieved after these operations for medically intractable angina pectoris, it is important to point out that an adequate warning signal is always preserved. On overexertion these patients still have painless sensations of constriction in the throat, palpitation or dyspnea. Occasionally mild pain persists in the neck or jaw presumably mediated by the vagus, as it is not eliminated by the removal of the entire cervical sympathetic trunk.

aneurysm of the aorta, as a complication of surgery of the gallbladder and in pancreatic fibrolithiasis nephralgia and idiopathic dysmenorrhea. In patients with upper abdominal visceral disease it is best to remove the splanchnic nerves and lower thoracic sympathetic ganglia by a paravertebral approach resecting the central portion of the eleventh rib on one or both sides. For relief of chronic renal pain which can be reproduced by ureteropelvic distention and relieved by subdiaphragmatic splanchnic block it is best to perform modified thoracolumbar sympathectomy. This is carried out by removing the twelfth rib and dividing the posterior attachment of the diaphragm. The splanchnic nerves are then seen passing through its medial arcade and can be resected from the celiac ganglion well up into the chest, together with the lower thoracic and first lumbar ganglia on the painful side. For relief of idiopathic dysmenorrhea, the presacral or superior hypogastric plexus is resected from the bifurcation of the aorta and down between the common iliac arteries (Fig. 31). Mortality and annoying complications after these procedures have been exceedingly rare and the incidence of relief from pain most gratifying. Success depends on careful selection of patients who are free from psychoneurosis or severe narcotic addiction and whose pain arises from within a diseased viscus. Relief during a preliminary trial period of procaine block must be clear cut and the denervation anatomically complete.

Pain in disease of the bladder which may be persistent and severe in tuberculosis and chronic interstitial cystitis despite all known methods of treatment, cannot be relieved by sympathectomy. The reason for this is that the afferent pathway is over the second to

fourth sacral nerves as illustrated in Figure 31. These posterior roots cannot be divided with impunity as this takes away all sense of bladder and rectal filling, thereby resulting in overdistention and incontinence. Bilateral anterolateral cordotomy can be utilized as a last resort but may also be followed by lasting retention of urine in a small proportion of cases. This procedure must also be used for the relief of unbearable pain in malignant disease of the viscera. In these circumstances pain does not become a problem until the disease process has invaded the retroperitoneal and lumbosacral plexuses or metastasized to bone, so that interruption of viscerosensory nerves can no longer be effective.

READING REFERENCES

- Cannon, W. B. *The Wisdom of the Body*. New York, W. W. Norton & Company 1932.
- Cannon, W. B. and Rosenblueth, A. *Autonomic Neuro-Effector Systems*. New York, The Macmillan Company 1937.
- Dandy, W. E. An Operation for the Cure of Tic Douloureux, Partial Section of the Sensory Root at the Pons. *Arch. Surg.* 18:687 1929.
- Grantham, E. G., and Spurling, R. G. Selective Lobotomy in the Treatment of Intractable Pain. *Ann. Surg.* 137:602, 1953.
- Hovelacque, A. *Anatomie des nerfs crâniens et rachidiens et du système grand sympathique chez l'homme*. Paris, G. Doin et Cie 1927.
- Kuntz, A. *Autonomic Nervous System*, 4th ed. Philadelphia, Lea & Febiger 1953.
- Lewis, T. *Pain*. New York, The Macmillan Company 1942.
- Mitchell, G. A. *Gs Anatomy of the Autonomic Nervous System*. London, E. & S. Livingstone 1933.
- Pect, M. M. Results of Bilateral Supradiaaphragmatic Splanchnicectomy for Arterial Hypertension. *New England J. Med.* 236:270, 1947.
- Ray, B. S., and Console, A. D. Residual Sympathetic Pathways after Paravertebral Sympathectomy. *J. Neurosurg.* 5:23, 1948.
- Scarff, J. E. Unilateral Prefrontal Lobotomy for the Relief of Intractable Pain; Report of 53 Cases with Special Consideration of Failures. *J. Neurosurg.* 3:30 1950.
- Shumacker, H. B., Jr. *Causalgia*. III A General Discussion. *Surgery* 24:483 1948.
- Sjöqvist, O. Trigeminal Neuralgia, A Review of Its Surgical Treatment and Some Aspects of Its Etiology. *Acta chir. scandinav.* 82:201 1939.
- Smithwick, R. H. The Autonomic Nervous System. In *Operative Technique in Specialty Surgery* edited by W. H. Cole. New York, Appleton-Century-Crofts, 1949 pp. 553-598.
- Taarnhøj, P. Decompression of the Trigeminal Root. *J. Neurosurg.* 11:299 1954.
- Walker, A. E. Mesencephalic Tractotomy: A Method for the Relief of Unilateral Intractable Pain. *Arch. Surg.* 44:953, 1942.
- Walker, A. E. Relief of Pain by Mesencephalic Tractotomy. *Arch. Neurol. & Psychiat.* 43:963, 1942.
- White, J. C. Cardiac Pain. Anatomic Pathways and Physiologic Mechanisms. *Circulation* 18:644, 1957.
- White, J. C., Smithwick, R. H., and Stimson, F. A. *The Autonomic Nervous System*, 3rd ed. New York, The Macmillan Company 1952.
- White, J. C., and Sweet, W. H. *Pain. Its Mechanisms and Neurosurgical Control*. Springfield, Ill., Charles C. Thomas, Publisher 1953.

Congenital Anomalies

By DONALD D MATSON M D

DONALD DARROW MATSON is a New Yorker by birth and was educated at Cornell University and at Harvard Medical School. From the beginning, he directed his training to the field of neurologic surgery in children based upon a wide training in general surgery. The Children's Hospital of Boston offers him a wide experience from which to make his contribution upon the congenital anomalies of the nervous system.

Congenital anomalies of the central nervous system and its coverings include spina bifida and cranium bifidum in their various forms, congenital dermal sinus tracts, craniosynostosis and congenital types of hydrocephalus.

Spina Bifida Spina bifida designates a developmental abnormality of the spinal axis. It is one of the commonest of all embryologic anomalies. Probably more than 20 per cent of all individuals will show some minor defect of a vertebral spine or laminae if survey roentgen ray examinations are made. Most of these defects occur in the lumbosacral area and have no clinical significance.

Whereas the cause of spina bifida is not known, the embryologic circumstances under which it occurs have been widely investigated and documented. In brief this anomaly represents a segmental abnormality of the invagination and separation of the neural ectoderm from the epithelial ectoderm, as well as a simultaneous abnormality in the development of the primitive mesoderm which gives rise to the bone and other supporting structures surrounding the neural tissue of the same segments.

If the fundamental embryologic error is limited to mesodermal derivatives there is no protrusion to the surface of intraspinal contents and the condition is known as spina bifida occulta. If the spine defect is accompanied by protrusion of meninges to the surface to form a saclike structure containing spinal fluid, the condition is known as spina bifida with meningocele. If the embryologic defect includes involvement of the neural tube as well, so that neural tissue protrudes to the surface adherent to the meninges, or if neural tissue fails to develop and function

normally the condition is known as spina bifida with myelomeningocele. If there is complete absence of all of the covering structures so that neural tissue itself presents on the surface of the body the condition is known as spina bifida with rachischisis. These varieties of embryologic fusion defects or spinal dysraphism are diagrammed in Figure 33.

From a surgical point of view the lesions which are of importance include certain types of spina bifida occulta, spina bifida with meningocele, and some of the cases of spina bifida with myelomeningocele. Most patients with spina bifida occulta do not need any treatment and most infants born with rachischisis are stillborn or survive only a very short period.

The presence of spina bifida occulta may be indicated by one of several types of cutaneous defects: an abnormal growth of hair located over the midline of the back and tapering off laterally on either side; irregular cutaneous angiomatous malformations over or just away from the midline; soft, poorly circumscribed, nontender subcutaneous masses of fatty tissue; dimpling of the skin in the midline, or just off the midline, with fixation of the epithelium to the underlying tissues. Children with any of these cutaneous defects should have roentgenograms of the spine made to establish the extent and type of the underlying spina bifida and should be watched carefully throughout growth for evidence of neurologic disturbance.

Spina bifida occulta may first be detected because of a neurologic deficit involving the lower extremities or control of the vesical and rectal sphincters. Often at about the

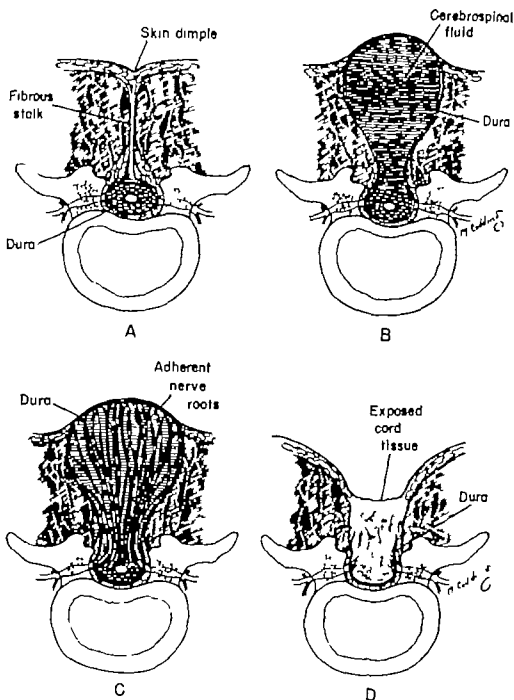


Figure 33 A, *Spina bifida occulta*. The intraspinal contents do not protrude to the surface, although often the site of the underlying bony defect is marked by a cutaneous abnormality. B *Spina bifida with meningocele*. A meningeal sac containing cerebrospinal fluid protrudes on the surface but there is no abnormality of spinal cord or nerve roots. C, *Spina bifida with myelomeningocele*. Nerve roots of the cauda equina are atrophic, displaced and adherent to the exposed membranous sac. D *Spina bifida with rachischisis*. Disorganized and distorted central neural tissue is exposed directly to the surface.

beginning of the second year of life when these youngsters begin to stand and walk, minor degrees of muscle weakness and gait disturbance are noted for the first time. Valgus, varus and cavus deformities of the feet, atrophy of the lower leg musculature, deformities of the toes, real or apparent shortening of one leg, pelvic tilt and limp have all been noted as primary complaints in

patients with spina bifida occulta. Every infant or child who shows a congenital type of scoliosis, dislocation of the hips, pelvic asymmetry, positional deformity of the feet, absence of deep tendon reflexes or evidence of sensory impairment in the lower extremities should have careful roentgenograms made of the entire spine in search of congenital abnormalities. Occasionally the pri-

mary complaint may be incontinence of urine, intractable bed wetting or difficulty in bladder and bowel training.

The diagnosis of spina bifida occulta is confirmed by roentgen ray examination. Anteroposterior films show widening of the spinal canal, absence of the spinous processes, defects of the laminae pedicles or vertebral bodies or possibly the occurrence of anomalous bony masses. Myelography in patients with spina bifida occulta may disclose the presence of fatty tumors, dermoid cysts, intraspinal meningoceles or fibrous cords which distort the subarachnoid space. Spina bifida occulta may occasionally be found at more than one level.

One type of spina bifida occulta of particular interest is that in which a spicule of bone arising from the posterior aspect of a vertebral body protrudes through the spinal canal dividing its contents into two separate compartments. This is known as *diastematomyelia*. It can be recognized on plain x-ray films by the presence of a midline shadow of increased density in an area of fusiform widening of the spinal canal and can be confirmed by contrast myelography in which the subarachnoid space is shown by the opaque medium to be completely split by the midline spicule of bone. Treatment consists of complete removal of the midline spicule of bone, lysis of adhesions from dura to the divided cord and conversion of the double dural compartment into one space.

Surgical treatment in spina bifida occulta is limited to those patients who show evidence of a progressive neurologic disturbance as growth proceeds and who by myelography demonstrate an intraspinal deformity which might lend itself to operative correction or improvement. This includes removal of bony spicules, dermoid cysts and fibrous bands which actually compress neural structures. Overenthusiastic attempts to remove poorly circumscribed masses of fat may result in increased neurologic damage. This should be regarded usually as prophylactic rather than corrective surgery its purpose being to allow further growth of the nervous system to proceed in as nearly a normal fashion as possible.

There is tremendous variation in the anatomic location, morphologic type and clinical importance of *meningoceles* and *myelomeningocele*s associated with spina bifida.

By far the commonest location is in the lumbosacral area, as might be expected, since this region is the last to be closed when

the neural ectoderm is folded inward from the surface (posterior neuropore). The next most common location is in the suboccipital high cervical area (anterior neuropore). Meningocele occur with much less frequency in the low cervical thoracic and upper lumbar segments. They may occasionally be multiple. Anterior meningocele protruding into the thorax, abdomen or pelvis rarely occur.

The visible lesion may be simply a flat partially epithelized membrane overlying a bony defect limited to one vertebral segment or there may be a huge sac, several inches in diameter even at the time of birth, with a thin almost transparent partially ulcerated membrane through which can be seen directly or by transillumination with strong light many nerve elements. A meningocele sac may have a narrow stalklike base so that it is a pedunculated lesion or it may be broadest at its base extending over several vertebral segments. Associated with many meningocele and myelomeningocele there is often an overgrowth of fibrous, poorly circumscribed fatty tissue so that in actuality a large proportion of the mass is not spinal fluid but fat. Except in very small lesions the margins of the bony defect can usually be palpated. It may be impossible to tell on physical examination, particularly when the sac is well covered with skin or thickened, contracted, translucent fibrous membrane, whether nerve elements are present or absent.

In addition to the size, location and covering of the meningocele itself the most important clinical features pertain to the neurologic status of segments caudal to the lesion and to the adequacy of cerebrospinal fluid circulation, that is, the presence of hydrocephalus. In early infancy the limitations of neurologic examination are many. However gross neurologic deficit is readily apparent and the most careful motor sensory and reflex evaluation possible of the lower extremities, as well as estimation of sphincter tone, should always be carried out. Structural deformities of the feet and legs are common in the presence of paraplegia.

Abnormalities of spinal fluid circulation resulting in hydrocephalus constitute complications to the management of infants with spina bifida. This hydrocephalus is due to malformation in the development of the hindbrain (Arnold-Chiari malformation). This anomaly consists of caudal displacement of the cerebellum through the foramen

magnum elongation, distortion and caudal displacement of the medulla oblongata and fourth ventricle and partial to total obliteration of the cisterna magna and surface subarachnoid pathways of the upper cervical spinal cord by dense adhesions. In general the most severe degrees of obstruction of spinal fluid circulation at the level of the foramen magnum due to the Arnold-Chiari malformation are seen with the more severe degrees of paralysis associated with lumbosacral myelomeningocele. Hydrocephalus is rarely seen with spina bifida occulta or simple meningocele.

There are few general rules which can be laid down for the surgical treatment of meningoceles and myelomeningoceles. Each patient's problem must be individualized. Ordinarily simple meningoceles without evidence of loss of neurologic function, should be repaired as soon as it is technically feasible. There is general consensus among neurosurgeons that operation is contraindicated in early life in the presence of total paralysis of both lower extremities and complete loss of bladder and rectal sphincter control, this is true whether or not hydrocephalus is present at the time. It is neither justifiable nor desirable to perform surgical procedures when it is clear that the infant is hopelessly and permanently crippled and that the chances of surviving to adult life in a useful fashion without encountering disastrous infection and gross retardation from progressive hydrocephalus are so limited. It must be explained carefully to all parents that removal of a myelomeningocele can do nothing to bring about recovery of paralyzed limbs or sphincters and, indeed, operation may increase the neurologic deficit if adequate repair is to be achieved. If children with paraplegia survive on nonoperative treatment and appear to be developing normally excision of a myelomeningocele may be indicated later in life as a means of facilitating nursing care and paraplegic ambulation.

In the presence of less than total paralysis associated with myelomeningocele the lesion should be repaired at a time when the judgment and experience of the neurosurgeon indicate that the most favorable result can be achieved. In general, delay in surgical repair increases the possibility of an optimum technical result. It is essential before such repair is performed that hydrocephalus be absent or controlled by surgery or spontaneous arrest. The leaking meningocele of the newborn should probably be repaired at

once if any attempt at all is to be made to preserve life.

Home management, in lieu of or awaiting surgical treatment, consists of regular careful cleansing of the meningocele and the surrounding skin, protection of the sac against friction by a padded circular dress, e.g., physiotherapy to the lower extremities and regular observations for evidence of hydrocephalus.

The purposes of surgical treatment are removal of the protruding, disfiguring mass and prevention of infection. Operation should be performed in such a way as to protect all neural elements and not produce or increase any neurologic deficit. Electrical stimulation of the various roots in and adherent to the sac may be useful to this end. An attempt is made to obtain a tight closure of the meningocele membrane supplemented by firm approximation of the overlying skin and subcutaneous tissues. No attempt is made to repair the bony defect. Cerebrospinal fluid circulation must either be normal or surgically corrected before meningocele repair is performed.

The treatment of spina bifida is not finished with completion of a successful operation. It should be the surgeon's responsibility to see that head measurements are obtained for many months following operation to detect the possibility of delayed onset of hydrocephalus. It should also be his duty to see that orthopedic help is obtained to direct physiotherapy and rehabilitation of musculoskeletal abnormalities and to see that bladder and bowel training are pursued when these are indicated.

Cranium Bifidum. Fusion defects of the cranial vault occur much less frequently than do their spinal counterparts. At the Children's Medical Center in Boston more than six times as many spinal meningoceles have been treated as have *encephaloceles*. An *encephalocele* may occur at any point over the cranial vault or base, usually in the midline or extending to the midline. By far the commonest region is in the occipital area where, in a high proportion of subjects there is associated intracranial malformation of nervous tissue or spinal fluid pathways. Rarely *encephaloceles* may project into the nose, nasopharynx or orbit. Whenever a nasal polyp is discovered in an infant, the possibility that it may represent an anterior *encephalocele* must be entertained to avoid the tragedy of creating a spinal fluid fistula and perhaps meningitis from excision of the lesion transnasally.

It is important that x ray films of the skull be made whenever there is a midline protrusion of any kind on the scalp. If there is an underlying bony defect, the lesion should be considered as an encephalocele and operation carried out with preparations for dealing with intracranial structures should this be necessary.

Morphologic types of encephalocele are comparable to those lesions seen along the spine. The external sac may be tiny or may indeed, be as large as the baby's head itself. It may be pedunculated on a very narrow stalk, passing through a small cranial defect, or the base of the lesion may be its broadest point. The sac may be covered with normal skin or with membrane of varying thickness. The encephalocele may communicate only with the surface subarachnoid pathway and contain simply clear spinal fluid with no neural tissue. It is often obvious on clinical inspection or transillumination that large amounts of brain tissue extend into the sac. Hydrocephalus is commonly associated with encephaloceles in the suboccipital area because of impairment of spinal fluid circulation through the posterior fossa.

Except in instances of obvious irreversible brain damage where fatality appears imminent, surgical removal of encephaloceles is usually indicated in early life. The difficulties of feeding, handling and ordinary nursing care are such with an encephalocele of any size and the psychic trauma to the par-

ents is so great in the presence of this disfiguring and unconcealable mass that the earliest possible excision is highly desirable. Surgical treatment consists in amputation of the membranous sac with watertight closure of the neck supplemented by firm approximation of the skin in two layers. If progressive hydrocephalus is present, this must be controlled before or at the same time the encephalocele is excised. Large bony defects in the frontal and parietal regions should usually be closed by cranioplasty either at the same time or subsequent to excision of the meningocele. It is ordinarily unnecessary to repair the bony defect in suboccipital lesions.

Congenital Dermal Sinus. When the neural ectoderm fails to be pinched off completely from the epithelial ectoderm, a persistent tract or tube of stratified squamous epithelium may extend from the skin surface inward to the central nervous system. Such a congenital dermal sinus may occur at any point along the midline of the central neural axis. It is most commonly found in the same areas where spina bifida is seen, namely in the lumbosacral region and in the suboccipital region.

Because of its persistent communication with the skin surface, such a sinus tract serves as a constant potential port of entry for infection into the central nervous system and its coverings. In addition to the danger of infection, such a dermal stalk may by its

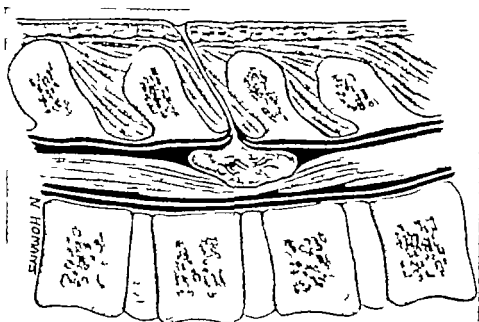


Figure 34. Longitudinal section illustrating congenital dermal sinus in the lumbar region expanding into dermoid cyst within the spinal canal (Ingraham, F. D., and Matson, D. D. *Neurosurgery of Infancy and Childhood*. Charles C. Thomas, Publisher.)

magnum, elongation distortion and caudal displacement of the medulla oblongata and fourth ventricle, and partial to total obliteration of the cisterna magna and surface subarachnoid pathways of the upper cervical spinal cord by dense adhesions. In general the most severe degrees of obstruction of spinal fluid circulation at the level of the foramen magnum due to the Arnold-Chiari malformation are seen with the more severe degrees of paralysis associated with lumbosacral myelomeningocele. Hydrocephalus is rarely seen with spina bifida occulta or simple meningocele.

There are few general rules which can be laid down for the surgical treatment of meningoceles and myelomeningoceles. Each patient's problem must be individualized. Ordinarily simple meningoceles without evidence of loss of neurologic function should be repaired as soon as it is technically feasible. There is general consensus among neurosurgeons that operation is contraindicated in early life in the presence of total paralysis of both lower extremities and complete loss of bladder and rectal sphincter control; this is true whether or not hydrocephalus is present at the time. It is neither justifiable nor desirable to perform surgical procedures when it is clear that the infant is hopelessly and permanently crippled and that the chances of surviving to adult life in a useful fashion without encountering disastrous infection and gross retardation from progressive hydrocephalus are so limited. It must be explained carefully to all parents that removal of a myelomeningocele can do nothing to bring about recovery of paralyzed limbs or sphincters and, indeed, operation may increase the neurologic deficit if adequate repair is to be achieved. If children with paraplegia survive on nonoperative treatment and appear to be developing normally, excision of a myelomeningocele may be indicated later in life as a means of facilitating nursing care and paraplegic ambulation.

In the presence of less than total paralysis associated with myelomeningocele the lesion should be repaired at a time when the judgment and experience of the neurosurgeon indicate that the most favorable result can be achieved. In general, delay in surgical repair increases the possibility of an optimum technical result. It is essential before such repair is performed that hydrocephalus be absent or controlled by surgery or spontaneous arrest. The leaking meningocele of the newborn should probably be repaired at

once if any attempt at all is to be made to preserve life.

Home management in lieu of or awaiting surgical treatment, consists of regular careful cleansing of the meningocele and the surrounding skin, protection of the sac against friction by a padded circular dressing, physiotherapy to the lower extremities and regular observations for evidence of hydrocephalus.

The purposes of surgical treatment are removal of the protruding, disfiguring mass and prevention of infection. Operation should be performed in such a way as to protect all neural elements and not produce or increase any neurologic deficit. Electrical stimulation of the various roots in and adherent to the sac may be useful to this end. An attempt is made to obtain a tight closure of the meningocele membrane supplemented by firm approximation of the overlying skin and subcutaneous tissues. No attempt is made to repair the bony defect. Cerebrospinal fluid circulation must either be normal or surgically corrected before meningocele repair is performed.

The treatment of spina bifida is not finished with completion of a successful operation. It should be the surgeon's responsibility to see that head measurements are obtained for many months following operation to detect the possibility of delayed onset of hydrocephalus. It should also be his duty to see that orthopedic help is obtained to direct physiotherapy and rehabilitation of musculoskeletal abnormalities and to see that bladder and bowel training are pursued when these are indicated.

Cranium Bifidum. Fusion defects of the cranial vault occur much less frequently than do their spinal counterparts. At the Children's Medical Center in Boston more than six times as many spinal meningoceles have been treated as have encephaloceles. An encephalocele may occur at any point over the cranial vault or base, usually in the midline or extending to the midline. By far the commonest region is in the occipital area where in a high proportion of subjects, there is associated intracranial malformation of nervous tissue or spinal fluid pathways. Rarely encephaloceles may project into the nose, nasopharynx or orbit. Whenever a nasal polyp is discovered in an infant, the possibility that it may represent an anterior encephalocele must be entertained to avoid the tragedy of creating a spinal fluid fistula and perhaps meningitis from excision of the lesion transnasally.

The diagnosis should be made in early infancy. It should be suspected from the shape of the infant's head and can be confirmed definitely by x-ray examination. Roentgenograms reveal readily the characteristic contour of the skull and satisfactory films even of newborn infants will demonstrate which have closed prematurely.

The contour of the cranial vault depends upon which sutures are prematurely closed that is, if growth is restricted in any one direction an attempt at compensation results in overgrowth in whatever direction expansion is still possible. If a single suture is involved, the skull expands in a direction parallel to this closed suture. Thus if the sagittal suture is closed at birth the head ex-

pands abnormally in an anteroposterior axis and appears elongated and narrow (Fig. 35). If the coronal suture is fused at birth there is abnormal lateral growth so that the skull appears wide with a broad, flattened forehead (Fig. 36). If the coronal suture is fused prematurely on only one side this flattening is asymmetrical. When there is premature fusion of all the cranial sutures growth is restricted in all directions and the head remains small with a tendency for it to expand upward in the direction of least resistance that is, against the region of the anterior fontanel (Fig. 37).

In addition to cranial deformity there may frequently be facial abnormalities due to involvement of the sphenoid and orbital

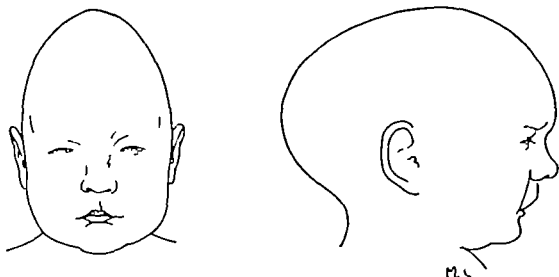


Figure 35 Anteroposterior and lateral appearance of infant with premature closure of the sagittal suture.

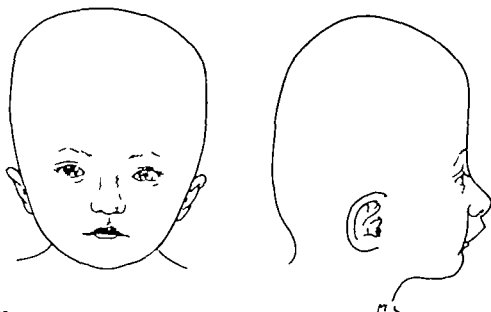


Figure 36 Anteroposterior and lateral appearance of infant with premature closure of the coronal suture. Often the orbital and nasal bones are also involved with much more deformity of the facial structures than is shown in this illustration.

attachment to neural tissue, distort the normal growth and function of the latter. At any point along a congenital dermal sinus and particularly at its innermost extent, there may be expansion of the sinus into a cyst (Fig. 34). Such a cyst may continue to grow in size because of desquamation of the stratified squamous epithelium which lines it and because of collection of sebaceous material from the glands within this skin. When such a mass occurs within the cranial vault or the spinal canal it may act as any other expanding lesion to interrupt neurologic function by local compression of the brain or spinal cord or it may cause interference with the normal circulation of cerebrospinal fluid. In addition, such a cyst may be the site of abscess formation because of the constant possibility of infection from the surface.

Every child who exhibits a sinus tract opening in the midline of the scalp or of the back should have x-ray films made of this area. If there is an underlying defect in the bone of the skull or of the spine, surgical excision of the sinus tract should be carried out forthwith. This should be done by a neurologic surgeon who is prepared to trace the tract into the central nervous system in itself if necessary.

In the presence of an unexplained meningeal infection, particularly due to *Staphylococcus pyogenes* var. *aureus* or *Escherichia coli*, the entire skin of the midline over the neural axis should be examined carefully for the existence of a sinus tract opening. When ever there is a history of meningeal infection or of redness, swelling, tenderness or of thin or purulent discharge from the midline sinus tract opening, complete excision of this sinus tract should always be carried out. The results of surgical treatment after abscess or meningitis has been established in patients with congenital dermal sinus are so poor that it is of the utmost importance to discover and remove these lesions prophylactically. Under the latter circumstances the surgical treatment is simple and the prognosis excellent.

Neurenteric Cyst. A rarer type of embryologic spinal defect is the neurenteric cyst. In this condition there is a cystic structure within the spinal canal usually anterior to the cervical or thoracic spinal cord. The cyst wall itself or a fibrous stalk from it may be intimately attached to the anterior surface of the cord. There is usually a circular defect through one or more vertebral bodies easily visible on x-ray films of the area and there may be an overlying spina bifida oc-

culta as well. The cyst itself is lined ordinarily by mucosa of entodermal origin which may have the characteristic appearance of esophagus, stomach, intestine or respiratory tract. This entity represents a failure of the mesoderm to separate completely the entoderm from ectodermal layers as they arise from the primitive mass of totipotential cells. Treatment consists of intraspinal removal of the cyst or stalk attached to the cord, followed if necessary by transthoracic excision of the extraspinal portion of the cyst wall.

Craniosynostosis. For reasons entirely unknown, but presumably as a result of a genetic defect in the germ plasma, the membranous bones of the skull, which normally are completely separated from one another at the time of birth, occasionally become fused prematurely. This condition, preferably called **craniosynostosis**, may involve any of the cranial sutures alone or in combination.

The human brain normally expands rapidly during the first two to three years after birth and then at a steadily decreasing rate until the individual is twelve years of age. In order for this normal growth of the brain to be possible, the bones of the skull must remain ununited and capable of expansion. Therefore if premature obliteration of the cranial sutures is present at birth, significant restriction of subsequent brain growth may occur.

It is important to differentiate carefully between **craniosynostosis** in which there is primary union between the bones of the cranial vault that restricts growth of the brain, and **microcephaly** in which there is secondary approximation but not union of the bones of the cranial vault which occurs because of failure of the brain itself to grow and push the skull ahead of it. In **craniosynostosis**, the primary defect is in the membranous bones of the skull and the condition is subject to surgical treatment. In **microcephaly** there is primary agenesis of the brain itself which causes secondary failure of the skull to expand normally. The head is small but of normal shape. In this latter situation no surgical treatment is indicated and the prognosis is ordinarily hopeless.

Craniosynostosis appears twice as commonly in males as it does in females. Fusion of the sagittal suture alone is by far the most common variety. Coronal suture closure alone is the next most common and, following this total synostosis of all the sutures is most frequent. Other single suture closure or combinations of various sutures are less frequently seen.

face subarachnoid pathways. It seems clearly established that when the exit of any of the ventricles of the brain is obstructed, that ventricle becomes dilated, and when the surface subarachnoid pathways are destroyed there is enlargement of the cisternae at the base of the brain as well as of the entire ventricular system.

Hydrocephalus refers to a pathologic condition characterized by an increase in the size of the ventricles and sometimes also part of the surface subarachnoid pathways as a result of the cerebrospinal fluid at some time being under increased pressure. The controlling factor in production of hydrocephalus appears to be the rate of reabsorption of cerebrospinal fluid into the vascular system and not its rate of formation. Hydrocephalus is caused therefore by the inability of cerebrospinal fluid to gain access to or be transmitted across the subarachnoid vascular barrier at a normal rate. Common sites of obstruction to spinal fluid flow include the foramen of Monro, the lumen of the third ventricle, the aqueduct of Sylvius, the outlets of the fourth ventricle (foramina of Magendie and Luschka) and the basilar cisternae.

Among the types of hydrocephalus seen as a result of congenital anomalies are those associated with stenosis of the aqueduct of Sylvius (Fig. 38) obstruction of the fora-

mina of the fourth ventricle (Fig. 39) and prenatal obliteration or failure to develop of the surface subarachnoid pathways (Fig. 40) particularly those at the base of the

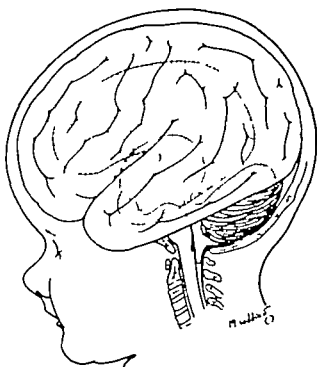


Figure 39 Hydrocephalus due to prenatal obstruction of the foramina of Magendie and Luschka. The fourth ventricle is dilated into a huge cystlike structure filling the posterior fossa and herniating into the upper spinal canal.

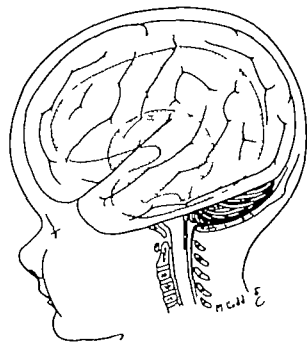


Figure 38 Hydrocephalus due to congenital obstruction of the aqueduct of Sylvius. The lateral and third ventricles are dilated, but there is no demonstrable communication through the midbrain to the fourth ventricle.

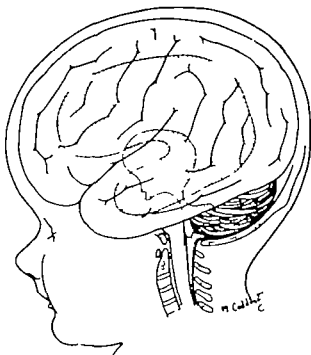


Figure 40 Hydrocephalus due to prenatal obstruction or failure to develop of the intracranial surface subarachnoid pathways. There is dilatation of the entire ventricular system and cisterna magna as well.

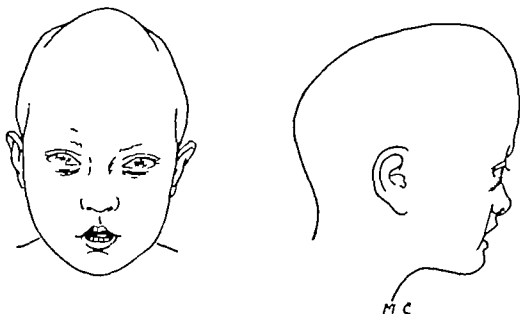


Figure 37 Anteroposterior and lateral appearance of infant with premature closure of all the cranial sutures. Note prominence in the region of prematurely obliterated anterior fontanel.

bones. This occurs particularly in association with fusion of the coronal suture whether alone or in combination with fusion of the other cranial sutures. Exophthalmos and choanal atresia are common findings. Chronic papilledema and optic atrophy with visual loss may also occur under these circumstances.

There is a high incidence of mental retardation among patients with craniosynostosis, particularly when multiple sutures are involved, if surgical treatment is not performed in early life. It is of primary importance, therefore, to make the correct diagnosis and carry out surgical treatment as early as possible not only to minimize the cranial deformity, but to provide the best opportunity for optimum mental development.

Many types of operative procedures have been performed in this disease. In youngsters over three to five years of age any type of procedure which decompresses the intracranial contents is probably satisfactory. In the first months of life, however, when surgery should be carried out if possible, the most physiologic type of treatment would appear to be creation of an artificial suture by linear craniectomy at the site of or parallel to the prematurely closed suture. In the first year of life new bone forms very rapidly from the dura mater and from periosteum and closes such an artificial craniectomy quickly. The use of an inert substance such as polyethylene plastic film placed over the margins of the craniectomy openings has proved satisfactory in delaying the bony closure of these artificial sutures long enough

to allow the brain to expand throughout the early period of rapid growth. It should be emphasized that this is prophylactic surgery in early life and one should not wait for signs and symptoms of increased intracranial pressure to appear before carrying out craniectomy to open up prematurely closed sutures. The prognosis for normal mental development after treatment during the first year of life is excellent when only one or two sutures are involved. After this period the percentage of patients with retardation increases markedly. In premature closure of all sutures, there may sometimes be irreversible brain damage by the time of birth.

Hydrocephalus. The central nervous system is normally bathed in its entirety in a clear colorless fluid which is contained in a series of connected spaces. These include the lateral ventricles within the cerebral hemispheres, the midline third ventricle, aqueduct of Sylvius and fourth ventricle, the large cisternae at the cerebellomedullary angle and base of the brain and the wide spread intricate surface subarachnoid pathways covering the spinal cord and the cerebral and cerebellar hemispheres. There is still much uncertainty about the exact source and mechanism of formation of the cerebrospinal fluid. However it seems clear that its point of principal origin is within the ventricles of the brain and its point of principal absorption is in the diffuse vascular bed of the pia arachnoid investing the cerebral cortex. Because of this, the fluid appears to flow from the ventricular system toward the sur-

surface subarachnoid pathways to a variety of distant locations including the mastoid antrum, the pleural space, the peritoneal cavity, bone marrow, urinary tract and intrathoracic vascular channels. The shunting procedures which have given the most promise have been those into the peritoneal cavity into the ureter and by way of the internal jugular vein directly into the right atrium of the heart. In the latter procedure a competent valve must be placed in the shunt tube which permits only unidirectional flow from the lateral ventricle to the blood stream. Although far from perfect diversion of cerebrospinal fluid through a catheter from an obstructed part of the subarachnoid pathway to some area of excretion or more adequate reabsorption appears to offer today the best treatment of internal hydrocephalus available.

READING REFERENCES

- Dandy, W. E. The Diagnosis and Treatment of Hydrocephalus Due to Occlusion of the Foramina of Magendie and Luschka. *Surg. Gynec. & Obst.* 32: 112, 1921.
- Ingraham, F. D., Alexander, E., Jr., and Matson, D. D.: Clinical Studies in Craniostomosis. Analysis of Fifty Cases and Description of a Method of Surgical Treatment. *Surgery* 24: 518, 1918.
- Ingraham, F. D., and others: *Spina Bifida and Cranium Bifidum*. Cambridge Mass., Harvard University Press, 1913.
- Ingraham, F. D. and Matson, D. D. *Neurosurgery of*

- Infancy and Childhood*. Springfield Ill., Charles C Thomas, Publisher 1951.
- List, C. F.: Intraspinal Epidermoids, Dermoids and Dermal Sinuses. *Surg. Gynec. & Obst.* 73: 525, 1941.
- Matson, D. D.: Hydrocephalus Treated by Arachnoid-Ureterostomy. Report of 50 Cases. *Pediatrics* 12: 320, 1953.
- Matson, D. D.: Ventriculo-Ureterostomy. *J. Neurosurg.* 8: 398, 1951.
- Matson, D. D., and Ingraham, F. D.: Intracranial Complications of Congenital Dermal Sinuses. *Pediatrics* 8: 403, 1951.
- McLaurin, R. L., and Matson, D. D.: Importance of Early Surgical Treatment of Craniostomosis. Review of 36 Cases Treated during the First Six Months of Life. *Pediatrics* 10: 637, 1952.
- Neuhauser, E. B. D., Harris, G. B. C., and Berrett, A.: Roentgenological Features of Neurenteric Cysts. *Am. J. Roentgenol.* 70: 235, 1958.
- Pudenz, R. H., Findlay, E. R., Hurd, A. H., and Sheldon, C. H.: Ventriculoauriculostomy: A Technique for Shunting Cerebrospinal Fluid into the Right Auricle. Preliminary Report. *J. Neurosurg.* 14: 171, 1957.
- Russell, D. S.: Observations on the Pathology of Hydrocephalus. London, His Majesty's Stationery Office, 1919.
- Russell, D. S., and Donald, C.: The Mechanism of Internal Hydrocephalus in Spina Bifida. *Brain* 58: 203, 1935.
- Spitz, E. B. and Nusen, F. E.: The Management of Hydrocephalus by a Valve-Regulated Venous Shunt Presented at the Harvey Cushing Society. April 25, 1957. Detroit, Michigan.
- Torkildsen, A.: Ventriculo-auriculostomy: A Palliative Operation in Different Types of Non-communicating Hydrocephalus. Oslo, Johan Grundt Tanum Forlag, 1947.

brain. In addition, hydrocephalus frequently occurs in association with spina bifida and especially with lumbosacral myelomeningoceles. In these instances the hydrocephalus is apparently always related to a congenital anomaly of hindbrain development which results in the so-called Arnold-Chiari malformation in which there is obliteration of the surface subarachnoid pathways around the brain stem and upper cervical spinal cord at the level of the foramen magnum. There may also be obstruction of the aqueduct of Sylvius and fourth ventricle in severe degrees of this anomaly. Hydrocephalus is also seen associated with encephalomeningoceles in the suboccipital region, usually as a result of obliteration of the surface subarachnoid pathways in the posterior fossa. Hydrocephalus may rarely be due to congenital malformation of the cerebral vascular system; this is usually an arteriovenous malformation which obstructs the posterior third ventricle or aqueduct of Sylvius.

The symptoms and signs of hydrocephalus due to congenital anomalies are those of increased intracranial pressure. These are usually seen in early infancy. They include enlargement of the head, an increase in the normal rate of growth of the head, congestion of the scalp veins, enlargement and bulging of the anterior fontanel, separation of the cranial sutures, displacement of the eyeballs downward and abnormal ocular movements. With severe degrees of increased intracranial pressure there is hyperirritability, increased deep tendon reflexes, ankle clonus and vomiting and there may be convulsions and alterations in respiration and pulse rate. In slowly advancing hydrocephalus there may be little in the way of symptomatology other than an accelerated rate of head growth.

The diagnosis of hydrocephalus is confirmed and its type may be determined by a series of tests. Every hydrocephalic infant should be studied to an extent that an authoritative decision regarding the indications for and type of treatment appropriate can be reached. These studies include measurement of the occipitobregmatic circumference of the head and comparison of this figure with standard charts; x-ray films of the skull; bilateral puncture of the subdural spaces through the coronal suture to rule out the possibility of the presence of subdural hematomas; combined puncture of one lateral ventricle and the lumbar spinal subarachnoid space with simultaneous measurement of pressures, protein and cell content of the

fluid; introduction of a quantitative amount of a tracer dye into the cerebrospinal fluid to test its passage from one compartment to another and to test the rate of absorption of fluid from the subarachnoid space, and ventricular air studies, usually carried out with removal of only a small amount of fluid and replacement by air (30 to 50 cc.). It is only on the basis of clinical experience supported by this group of tests that surgical treatment can be planned intelligently.

If there is reasonable evidence that there is still a chance for normal development and it is established that progressive hydrocephalus is present then the best available surgical procedure should be carried out as soon as possible. In the presence of gross irreversible central nervous system damage which precludes a desirable result no matter how successful operative treatment may be, custodial care only should be recommended. Temporizing operations which permit infants who are blind, decerebrate or otherwise irreversibly damaged to survive, but do not allow for normal brain growth, should be avoided.

The treatment of hydrocephalus is surgical. Dehydration, diuresis and all known forms of drug therapy are of no more than temporary avail. Until the past decade, the surgical treatment of hydrocephalus has been a record of occasional optimism and rare success but mostly of discouragement and failure. More recently a variety of operative procedures in which spinal fluid has been shunted from one compartment of the spinal fluid pathway to another or from the spinal fluid pathway to some other location in the body where it could be reabsorbed or excreted from the body have been employed with increasing success. When obstruction has occurred in the third ventricle, a rubber or plastic catheter has been used to shunt spinal fluid from one of the lateral ventricles into the cisterna magna in the posterior fossa or into the surface subarachnoid pathway within the cervical spinal canal. If the surface subarachnoid pathways are well developed and normally patent, this operation should successfully control increased intracranial pressure. If the surface pathways are inadequate to handle the fluid shunted to them as is usually true in very young infants who have marked hydrocephalus from aqueduct obstruction, then a further procedure must be done to remove the excess fluid from the surface subarachnoid pathways. Fluid has been shunted both from the ventricular system and from the

medicine for hundreds of years. Believing in the supernatural, early physicians applied themselves more energetically to the formulation of incantations designed to cast out demons than they did to the study of physical evidences of disease. Although Galen (131-201 A.D.) was the founder of experimental physiology, his few primitive discoveries so entranced him that he expanded his small array of facts into comprehensive theories to explain every phenomenon of pathology. Garrison stated "Galen with fatal facility and ingenuity proceeded to explain everything in the light of pure theory, thus substituting a pragmatism of medical philosophy for the plain notation and interpretation of facts as taught by Hippocrates. The effect of this dogmatism and infallibility upon after time was appalling for his assumption of omniscience was specially adapted to appease the mental indolence and flatter the complacency of those who were swayed entirely by reverence for authority." The effect of Galen's contributions was more negative than positive, and for two reasons. First, he made the fatal error in research of adopting postulates in advance and directing his investigations to their support rather than permitting his experiments to lead where they would. And second, his systemization of medical knowledge exerted of itself so great an appeal to those who came after him that for fifteen centuries anyone who questioned his conclusions was regarded as a heretic.

Surgery largely escaped such handicaps. As Susruta noted, it was less liable than internal medicine to the fallacy of conjecture. For the most part, early surgeons were practical men and this characteristic continues to distinguish them from speculative philosophers. It also accounts for their interest in and development of the basic sciences of anatomy and pathology and a working knowledge of these subjects became one of the first qualifications of a surgeon. It was perhaps this pragmatism of surgeons which was responsible for their low social status until modern times. The knowledge of Latin and Greek was not essential to the use of the knife and one of the sources of the attacks made upon Ambroise Paré by the physicians of his day was that he wrote in his native tongue rather than in a classical language.

The progress of surgery has been marked with successive barriers to further advancement, each of which was pushed back by a significant discovery. For the most part, these obstacles have been real rather than

philosophical. Three of these were recognized when the first incision was made and others have appeared as the limits of surgical endeavor have been widened. Perhaps the most immediate problem of the primitive surgeon was the control of hemorrhage. In the beginning there was no method of control of bleeding and prevention became the first concern of the surgeon. In the Papyrus Ebers (circa 1550 B.C.) the surgeon is adjured "to treat it with the knife but look out that you avoid the blood vessels." Obviously such necessity greatly restricted the scope of surgery and perhaps the outstanding qualifications of the surgeon of that day were familiarity with the anatomy of the superficial vascular system and caution and courage in proper balance. Pressure was probably the first technique for the control of hemorrhage and this was followed by actual cautery and use of styptics. Although the ligature was used as early as the thirteenth century, boiling oil as a hemostatic continued to be popular for more than 300 years thereafter and the absorbable ligature did not appear until the nineteenth century.

Ability to control hemorrhage markedly increased the scope of surgical practice. However, as soon as this danger was minimized, other problems became limiting factors which had not been very formidable within the restrictions imposed by uncontrollable hemorrhage. One of these was the distantly related pair of reactions, pain and surgical shock, the other was infection. It is moot as to which of these complications was the greater obstacle to further development of surgery. Since the latter continued to plague surgery long after the discovery of anesthesia and since anesthesia extended the limits of surgery into areas in which infection became a much more serious problem, relief from pain may be accepted as the triumph next in importance as well as next in time.

So long as surgical procedures were restricted to the extremities and the superficial layers of the head, neck and torso the intensity and the duration of pain could be favorably influenced by speed of operation. So quick decision and manual dexterity became important qualifications of a surgeon. In this era, except for the rarer massive amputations, surgical shock was not ordinarily so profound as to create serious problems. It was when the surgeon began to meddle with the contents of the peritoneal cavity that shock became a complication to be reckoned with. While anesthesia deadened

THE QUALIFICATIONS OF A SURGEON

By PAUL R. HAWLEY, M.D.

PAUL RAMSEY HAWLEY, the son of a country doctor in Indiana, practiced with his father and entered the regular army medical corps in World War I. Chief Surgeon of the European Theatre of Operations during World War II, he was responsible for the organization of the superior quality of care given to the wounded. He became Chief Medical Director of the Veterans Administration and initiated the participation of the members of medical school faculties in the treatment of veterans. In his present capacity as Director of the American College of Surgeons, Dr. Hawley has made important contributions to the maintenance of the high ethical and professional standards of the surgical profession and to the education of the public.

Just as the qualifications of an aviator have changed with the development of aircraft and the qualifications of a sea captain with the transition from Roman galleys to luxury liners, so have the qualifications of the surgeon changed with the advances in surgery. Certain basic requirements for the surgeon, however, have persisted since the first incision was made for the alleviation of suffering and these will remain unchanged so long as surgery is done.

In part, these essential qualifications are those which apply to every practitioner of the healing arts—integrity, humanity, fidelity to responsibility and freedom from avarice. To these must be added one which applies particularly to the surgeon. This is intrepidity—the quality of calm judgment and decisive action in sudden crises. No one will practice surgery long before he is suddenly faced with a grave emergency in the course of an operation. To hesitate, to become

panicked is fatal. The surgeon must never become bewildered.

While centuries have not altered these basic qualifications of a surgeon, surgical progress has forced the addition of a number of other qualifications. For centuries surgery was the poor relation in the family of medical sciences. The surgeon had little formal education and he occupied a lowly social position. If these were handicaps, they were also advantages. They freed the surgeon from the rigid formalization of learning which impeded scientific progress for centuries. Unable to read Greek and Latin, he studied the patient. Susruta (fifth century A.D.) called surgery "the first and best of the medical sciences, less liable than any other to the fallacy of conjecture and inferential practice, pure in itself, the worthy product of heaven."

In this praise of surgery are implied the obstacles which delayed advances in internal

ing tissue. Surgery is no longer a mechanical art, it is a coalition of several sciences and those who contribute to its advancement in the future must be prepared accordingly.

Refinement of anesthesia has opened an other field of surgery of vast and increasing importance. This is surgery within the chest. By overcoming Nature's intolerance of a vacuum, the anesthesiologist has made it possible for the surgeon to work leisurely upon the lungs, the great vessels and, within rapidly expanding limits upon the heart itself. The end of restrictive limitations upon cardiac surgery is already in sight.

So much for the technical and scientific advances in surgery. Just as each technical advance introduced new problems or magnified existing ones, so have these great strides in surgical usefulness introduced new problems for the surgical profession. Only a few years ago surgery was a dangerous undertaking for the patient—so dangerous that it was considered only in urgent and critical situations in which death appeared to be the only alternative. Under such conditions the reluctance of both the patient and the surgeon offered considerable protection against unjustified surgery.

Now that the danger of surgery has been considerably decreased—although not so greatly as some believe—two problems are increasing rapidly in importance. One of these is that too many physicians are attempting to do surgery without adequate preparation and training and the other is that too many of this group and too many adequately trained surgeons are performing operations which cannot be justified. One mark of a good surgeon is knowing when not to operate and an essential characteristic of an ethical surgeon is refraining from surgery unless there is definite indication for it.

In his presidential address at the 1953 Convocation of the American College of Surgeons, Doctor Fred W. Rankin said that "In many respects surgeons carry the heaviest responsibilities of all physicians, if only because of the harm they can do. For this reason it is essential that they never forget what they are undertaking when they lay hands upon the human body." This locates precisely the boundary of surgical endeavor across which no upright surgeon will step. During World War II a young military surgeon once boasted to Surgeon General Kirk that he had "taken a chance" in some surgery upon a patient and that he had "got away with it." "You took a chance!" replied General Kirk, the surgeon never takes a

chance. It is the patient who takes the chance."

Many definitions have been offered to distinguish between major and minor surgery. None has been completely acceptable and there is the rapidly growing belief that almost all surgery is major in character. Diagnosis yet being an art subject to frequent inaccuracies, one who essays an exploration of the human body must be competent to deal with any situation he may encounter. The projected cholecystectomy may turn out to be an operation for carcinoma of the pancreas. To rule out all possible contingencies inherent in major surgery upon the assurance offered by preoperative diagnosis is no more defensible than venturing far to sea in a cockleshell upon the prediction of continued fair weather. Diagnosis is no more exact a science than meteorology and the granting of surgical privileges on the basis of specified operative procedures indicates total indifference to the safety of patients.

Furthermore, the surgeon is no mere technician, despite the growing tendency to so limit his function. He is a consultant to the point where he advises and the patient agrees to surgery. At this point, the surgeon becomes the physician in charge, responsible for all decisions in the care and treatment of the patient and this relationship is continued until it is terminated by the one or the other. The surgeon must arrive at his own diagnosis and must make his own decision as to what surgery if any is indicated. If his decision to operate is made solely upon the advice or request of another physician, he is in effect engaging in ghost surgery regardless of the patient's awareness of his identity.

These requirements place the full responsibility for surgery upon the surgeon. So long as the surgical condition is paramount, the patient is a surgical patient and his care and treatment remain the obligation of the surgeon. The decisive influence of physiology upon surgical results makes it mandatory that, whenever possible, both preoperative and postoperative care be directed by one specially trained in this field, and that surgeon has shirked his duty who voluntarily relinquishes control of a surgical patient before all danger of postoperative complications has passed. In such a practice lies the chief iniquity of itinerant surgery.

One of the heaviest responsibilities of the family physician is in the choice of surgical consultants for his patients. There is only one defensible rule for him to follow and this is to make the same choice that he

the pain from rough handling of the abdominal viscera, it was less effective in preventing shock and manual dexterity applied particularly to gentleness in handling delicate structures continues to be a qualification of a surgeon. While indecisive putting in is not acceptable technique, speed in recent years has been sacrificed in some degree for a more careful toilet of the operating field. Anesthesia opened the door of the abdomen to the surgeon, but he entered at rare intervals and only for urgent reasons until he learned to avoid soiling the carpet and the furniture.

While hemorrhage and shock were immediate dangers in surgical procedures, an even greater danger had almost always to be faced within a few hours after operation. Almost all wounds, whether accidental or purposeful were infected, even though this was not always apparent to the naked eye. Healing by first intention was so rare as to cause suspicion in some surgeons. Pus was so often a concomitant of wound healing as to be regarded as possibly contributing to that process. The appearance of "laudable pus" was anxiously awaited. Since the character of the pus was some index of the pathogenicity of the invader and the amount some index of the resistance of the host it is readily understandable that certain types of suppuration were regarded as laudable.

It is difficult to conceive of a phenomenon in medicine more baffling to the early surgeon than infection. The cause could not be detected by the unaided senses and the effects were so variable as to defy pure speculation. There is the legend that Ambrose Paré delivered the wife of a shepherd by cesarean section, using the shepherd's knife and operating in the shepherd's isolated cottage. The operation was successful and one can imagine Paré's wonder when an impressive series of his patients without exception died of peritonitis when he attempted the same procedure in a Paris hospital. So to the special qualifications of surgeons was added a heart of steel to bear the recurring discouragement of death and failure which followed in their wake."

The seizure of Lister upon the discoveries of Pasteur inaugurated a revolution in surgical technique. Never in history had surgery taken such a long step forward or added so much to the qualifications of a surgeon. Heretofore largely interested in only the descriptive sciences, the surgeon suddenly found himself an experimental scientist. As Lord Moynihan said, "Every operation in

surgery [becomes] an experiment in bacteriology.

The success of the experiment depends not only on the skill but also upon the care exercised by the surgeon in the ritual of the operation.

With hemorrhage, pain, shock and sepsis controllable if not entirely preventable, the area limits of surgery were almost removed. There remained only the obstacles to entering the thorax, which were overcome by advances in anesthesiology. Had surgeons remained primarily artists with shallow excursions into the sciences these accomplishments might have been considered sufficient.

Fortunately surgery has adopted no such complaisant attitude. With the technical improvements of the last decade or so now permitting operations of a magnitude not dreamed of a generation ago it has become apparent that the ultimate limits of surgery are to be fixed not by the resources of the surgeon or by the accessibility or dispensability of the offending parts, but by the ability of the human body as a whole to withstand the onslaught made upon it and to readjust thereafter. It is in this respect that the animal organism differs most remarkably from the inanimate machine. The machine may be placed in a state of suspended animation while repairs are made, but life must be maintained in the animal organism during and after surgery. Life, insofar as can yet be demonstrated, is a continuous and complex physical chemical operation in rather delicate balance and the survival of a surgical patient often depends more upon the maintenance of this balance than upon the excellence of surgical technique.

For this reason, perhaps the most significant advances in surgery in the past few years have been in the fields of preoperative and postoperative care of the patient. Competence in these fields distinguishes the surgeon from the operator. Preoperative treatment is designed to fortify the physiology of the patient against the terrific insult to his metabolism which cannot be avoided in big surgery. Postoperative treatment is directed toward the rapid re-establishment of normal physiologic processes and their maintenance thereafter. In researches seeking the solution of these problems, surgery has led the way. While it may continue to be possible for the practitioner of surgery to apply techniques of which he has little or no basic understanding, those who in the future will push back the limits of surgery must have a working knowledge of all the sciences involved in the mechanisms of life.

SURGICAL JUDGMENT

By HAROLD LAUFMAN M D

If surgery were an exact science in the strictest sense of the word, the concept of surgical judgment would never have come into being. Facts could be collated to list all possible deviations from the healthy human state and universally applicable formulas could be devised to describe accurately and unerringly the exact procedures for their correction. In such case the student of surgery need but memorize a list of facts and develop through usage a high degree of mechanical skill to achieve pre-eminence in his field. He would never be faced with a host of conflicting variables requiring appraisal and interpretation, he would not need to make a separate decision in each case; he would not have to deal with his patients as people. He would be, in fact no more than a superbly functioning machine.

Surgery is not, however "exact" in that narrow sense. Insofar as the discipline of surgery is based on certain universally applicable principles of anatomy bacteriology physiology and related disciplines, it is a science. Insofar as it must apply these principles to the specific needs of an individual patient under particular circumstances at a given time, surgery is also an art.

Sound training in the fundamentals of surgery and a high degree of technical competence are the basic equipment of the surgeon; they are taken for granted. If he has nothing more a surgeon may be adequate in ordinary situations. If, however, he is to translate his knowledge and apply his skills into their highest potential of healing for each individual, the surgeon must enter into the infinitely complex series of mental, moral

and physical acts which for working purposes is called *surgical judgment*.

Since it is so complex, both as a concept and as a working process, a completely adequate consideration of surgical judgment would have to deal with virtually every aspect of a surgeon's life and the whole of his psychologic structure. Yet surgical judgment is said to be something that a surgeon either has or does not have. It is perennially offered to medical students as a distinguishing excellence they may one day hope to achieve.

Because of the comprehensive nature of surgical judgment, authoritative criteria for it cannot be codified. On the other hand, since it does exist as a therapeutic tool or as a catalyst for all other therapeutic tools, it warrants a more orderly and systematic consideration than has so far been accorded it. It should not remain forever in the realm of a mystique, implicitly acknowledged but always undefined.

Judgment, in general, is not just a static condition or quality though it is regarded as something one "has." It is actually a mental process in which man is always consciously or unconsciously engaged. Daily a number of trivial or important decisions are made based on momentary or prior evaluations. Daily evaluations of one sort or another are made, on which future decisions will be based. Most of the time this process is not recognized and requires little conscious attention. An effort to define it would necessitate looking back on the decisions made over a time. A certain pattern of choices would emerge, a pattern of a fairly consistent nature—the nature of this pattern

would make if he or one of his immediate family were in the position of the patient. This means that no inducement to refer a patient to a specialist should ever be considered other than the quality of patient care expected or desired.

Unethical inducements to refer patients are the product of growing competition and shrinking ideals. They are offered, and accepted, in many guises. The form best known, and probably most common, is the division of the fee collected by the specialist with the referring physician. This practice has long been outlawed by organized medicine, but despite such proscription it has increased in prevalence in many areas. Other forms of unethical inducement are practiced by some who hesitate to divide an individual fee. These are individual billing of alternate patients wherein the specialist collects the entire fee from one patient and the referring physician from the next, the giving of lavish gifts to referring physicians and subsidizing of young men starting practice with the understanding that they will refer their patients to the creditor. The betrayal of the trust of a patient for a few pieces of silver is on a par with the crime of Judas. Even if the choice of a consultant is not influenced by the commission received, the mere yielding to avarice in accepting it brands the physician as deficient in integrity.

Thus, the enormous increase in the capabilities of the surgeon has been accompanied by an equal increase in his responsibilities. His capabilities for good are matched by his capabilities for evil and his most important qualifications are the ability to distinguish between the two and the integrity to choose the right. In no other vocation are the words of Saint Paul more applicable "they that will be rich fall into temptation. For the love of money is the root of all evil."

As often happens in other fields efforts to codify medical ethics have confused rather than clarified these rules. Rules of etiquette among physicians have been interpolated between rules of morality and have become re-

spected as much as if not more than, rules protecting the patient. Webster defines ethics simply as moral principles, quality or practice, the science of moral duty, and, more broadly the science of the ideal human character and the ideal ends of human action. Medical ethics is merely the application of these principles to the practice of medicine. The Great Physician simplified medical ethics still more when He said, "Therefore all things whatsoever ye would that men should do unto you, do ye even so to them. This is all there is to medical ethics."

To summarize, the qualified surgeon must be first a physician, he must be observant, manually dexterous, both courageous and cautious, calm in the presence of disaster, he must acquire an adequate working knowledge of the basic sciences applicable in surgery and, above all, he must have integrity. Guy de Chauliac (1300-1370) wrote a prescription for a surgeon some 600 years ago to which the centuries have added little and from which they have detracted nothing. He said, "Let the surgeon be bold in all sure things and fearful in dangerous things, let him avoid all faulty treatments and practices. He ought to be gracious to the sick, considerate to his associates, cautious in his prognostications. Let him be modest, dignified, gentle, pitiful and merciful, not covetous nor an extortionist of money, but rather let his reward be according to his work, to the means of the patient, to the quality of the issue, and to his own dignity."

READING REFERENCES

- Garrison, F. H. *History of Medicine*. 4th ed. Philadelphia, W. B. Saunders Company 1929 p. 113.
- Maes, U., and Ilgenfritz, H. C. *Lewis Practice of Surgery*. Hagerstown, Md., W. F. Prior Company 1928, chap. 2.
- Major, R. H. *The Papyrus Ebers*. *Ann. M. Hist.* 2: 54, 1930.
- Moyntian, B. *The Ritual of a Surgical Operation*. *Brit. J. Surg.* 8: 27, 1920.
- Sarma, P. J. *Hindu Medicine in Its Antiquity*. *Ann. M. Hist.* 3: 322, 1931.

tained by one surgeon do not necessarily reveal what another surgeon may expect. In order to make a cogent comparison one would need to presume approximate equality of experience, ability, facilities, and judgment between the surgeons as well as comparable lesions and risk components in the patients. A surgeon's use of the literature, therefore, must be discriminating and selective when he attempts to apply what he reads to his own practice. Although knowledge of current statistics may serve as a guiding factor to the surgeon's judgment, it cannot substitute for judgment nor even dominate it. The surgeon cannot operate on a statistical basis alone; for although it will certainly yield numbers, it cannot dependably yield meanings and interpretations.

RAPPORT IN SURGERY

Once these preparatory, preoperative judgments have been made, at least tentatively, by the surgeon, he must inform and prepare the patient and responsible members of the family. A good personal rapport with them will depend on two cardinal qualities in the surgeon, which may at first seem to conflict: honesty and tact. A patient is entitled to know everything pertinent both to his condition and to the probable course of his treatment, which he is capable of understanding intellectually and able to accept emotionally. His questions must be respected and answered fully but simply without recourse to unnecessarily technical language. Professional jargon has no place with a patient; it is designed to impress rather than to inform. Oversimplification is equally out of taste: it is a condescension the patient is quick to sense and which he may justifiably resent. Needless to say, the abrupt dismissal "Let me worry about that," to which an earlier generation of surgeons was prone, insults the patient and argues an insufficiency in the surgeon.

Even as a patient must be respected intellectually, respect must also be accorded his particular emotional system. His tensions and anxieties must be accepted as natural. It is particularly difficult to do so when they are exaggerated or neurotic. A patient comes for help, not for censure; he needs assurance and perhaps the support of an authoritative figure, but the surgeon's "authority" must not be misused to reduce the patient's self-respect and dignity. The patient who is treated as an equal and co-operative adult will tend to be one.

STEREOTYPY AND ROUTINE

The dangers of stereotypy in general are among the most pervasive and pernicious of therapeutic fallacies. The organization of knowledge is part of the business of science; to define separate entities and classify them according to general type is a legitimate intellectual act. Except in rare instances, almost any condition in almost any patient can be placed, for the purposes of classification, into conformance with a familiar disease entity described previously in the literature or familiar to the physician. To establish this is only the first part of the surgeon's obligation. A second, sometimes larger and equally cogent part is to know that there is no such thing as an "average" patient. Even the most "ordinary" patient with the "simplest" lesion has certain sensitivities, stresses, patterns peculiarly his own, requiring the surgeon's individual attention to his particular problem. A surgeon who works in the climate of this basic attitude will be swiftly and sensitively responsive to unexpected reactions and events for which surgeons who think in stereotypes may be unprepared.

Does a usual procedure need to be modified in its nature or degree for a particular patient? This question again must be answered by balancing the nature of the disease against a series of factors peculiar to a given patient. Among other things, these include expected longevity in relation to existing pathology; the presence and severity of associated but not necessarily related infirmities; the degree of urgency deemed essential for preservation of health or life; the success of the physiologic preparation for surgery; an appraisal of the patient's tolerance for the several variations of the available procedural plans; and knowledge of the choice of goals to be sought.

Even as a patient must not be regarded as "average," so the tendency to regard procedures as "routine" must be avoided. The word is often used to indicate a tested and reliable method of approach or to describe a pattern of procedural steps which has proved dependable throughout the years in thousands of individual instances. The word "routine" is allowable, in this context, on practical and pragmatic grounds.

But when dealing, as here, with more precise meanings of concepts in terms of their ultimate implications for intellectual and clinical behavior, the word "routine" is best avoided. It suggests an unthinking, undiffer-

would serve to define the quality of judgment. Judgment can thus be defined as a series of mental acts involving an appraisal of factors leading to decision in the service of a goal.

Surgical judgment is a specialized application of this process requiring greater awareness and subject to more stringent criteria, since the attainment of such important goals depends on it. The goals of surgical judgment upon which all decisions are based and toward which all evaluations are oriented consist in the perennial aims of all therapy: prolongation of life and alleviation of suffering. The manner in which a surgeon employs his personal endowments, his training and his experience in the service of these ends will define him as a surgeon.

PREOPERATIVE JUDGMENT— MOTIVATIONS IN SURGERY

A patient comes to a surgeon because either he or his referring physician thinks he may need surgery. The referral, strictly speaking, is for purposes of a surgical opinion rather than for surgery per se. Even if a given pathologic state is ordinarily amenable to surgery, the decision for or against surgical intervention in a particular instance must be made. Such a decision involves first the accumulation of pertinent data through the indispensable classic sources: a careful history, a thorough physical examination, diagnostic laboratory findings and the surgeon's general or clinical impression of the case. Each factor in the data accruing from these sources must be assessed first individually for its own implications and then in relation to all other factors. This appraisal is made to answer three prime questions: (1) Is a surgical operation advisable at all? (2) If it is advisable is it also feasible in terms of risk to the patient or anticipated result? (3) How might the operation have to be modified to fit a specific set of circumstances?

Often the highest product of seasoned and responsible surgical judgment is the decision not to operate. A number of influences irrelevant to the patient's welfare may be brought to bear upon the surgeon in the formation of this important decision. The surgeon may be eager for experience; he may be influenced consciously or unconsciously by his own psychologic or financial needs; the referring physician may indicate a subtle but influencing preference; the patient himself, for tangential reasons, may urge surgery.

Sometimes a patient has a vague disorder which he hopes may be magically defined and cured, sometimes he has lived with a fear of surgery long enough that he wants to get it over with; sometimes he is just surgery prone. Without playing psychiatrist, a surgeon must remain alert to such distortions if he is effectively to resist them.

The most dangerous artifacts introduced into the area of surgical motivation are, of course, those which may occasionally arise out of the surgeon's own distortions or out of his own self-interest. No matter how well it can be rationalized, any motive that is not based exclusively on a concern for the patient's welfare is an artifact which violates the basic principles of surgical judgment. It is in this area of mixed motive that we encounter such aberrations of surgical behavior as fee splitting, ghost surgery, irresponsible timing of operations and unjustified surgery.

If surgery is feasible at all, the only allowable motivation for it is a conclusion that it is necessary to the patient's welfare. There are, of course, different kinds and degrees of necessity. Surgery may be deemed necessary if (1) the available information indicates that the patient's condition cannot improve in a reasonable time without surgery; (2) the condition may lead to complications or death without surgery; (3) accurate diagnosis leading to more accurate therapy depends upon surgical intervention.

When according to the valid criteria, it does seem advisable to operate, all factors must be weighed to estimate the extent and nature of risk for the particular patient involved, since the next complexus of decisions is made with a view of minimizing, insofar as possible, the attendant risks. This consideration will govern the selection of an optimal time, the most adequate available place, the course of preoperative care, the type of anesthesia, the best anatomic approach and the extent of permissible radicality. Estimation of risk will also involve some knowledge of the possible complications and a reasonably close prediction of probable outcome, based on the nature and urgency of the case in light of the best results to be hoped for. It requires knowledge of the current statistics on morbidity complications and mortality, based not only upon the literature, but upon the surgeon's own experiences with comparable cases.

If his experience is sparse, a surgeon's use of the literature can help him in his estimates, but only up to a point. Results ob-

tained by one surgeon do not necessarily reveal what another surgeon may expect. In order to make a cogent comparison one would need to presume approximate equality of experience, ability, facilities, and judgment between the surgeons, as well as comparable lesions and risk components in the patients. A surgeon's use of the literature therefore must be discriminating and selective when he attempts to apply what he reads to his own practice. Although knowledge of current statistics may serve as a guiding factor to the surgeon's judgment, it cannot substitute for judgment nor even dominate it. The surgeon cannot operate on a statistical basis alone, for although it will certainly yield numbers, it cannot dependably yield meanings and interpretations.

RAPPORT IN SURGERY

Once these preparatory preoperative judgments have been made—at least tentatively—by the surgeon, he must inform and prepare the patient and responsible members of the family. A good personal rapport with them will depend on two cardinal qualities in the surgeon, which may at first seem to conflict: honesty and tact. A patient is entitled to know everything pertinent both to his condition and to the probable course of his treatment which he is capable of understanding intellectually and able to accept emotionally. His questions must be respected and answered fully but simply without recourse to unnecessarily technical language. Professional jargon has no place with a patient; it is designed to impress rather than to inform. Oversimplification is equally out of taste: it is a condescension the patient is quick to sense and which he may justifiably resent. Needless to say the abrupt dismissal "Let me worry about that," to which an earlier generation of surgeons was prone, insults the patient and argues an insufficiency in the surgeon.

Even as a patient must be respected intellectually, respect must also be accorded his particular emotional system. His tensions and anxieties must be accepted as natural. It is particularly difficult to do so when they are exaggerated or neurotic. A patient comes for help, not for censure; he needs assurance and perhaps the support of an authoritative figure, but the surgeon's authority must not be misused to reduce the patient's self-respect and dignity. The patient who is treated as an equal and co-operative adult will tend to be one

STEREOTYPY AND ROUTINE

The dangers of stereotypy in general are among the most pervasive and pernicious of therapeutic fallacies. The organization of knowledge is part of the business of science to define separate entities and classify them according to general type; it is a legitimate intellectual act. Except in rare instances almost any condition in almost any patient can be placed, for the purposes of classification, into conformance with a familiar disease entity described previously in the literature or familiar to the physician. To establish this is only the first part of the surgeon's obligation. A second, sometimes larger and equally cogent part is to know that there is no such thing as an "average" patient. Even the most "ordinary" patient with the simplest lesion has certain sensitivities, stresses, patterns peculiarly his own, requiring the surgeon's individual attention to his particular problem. A surgeon who works in the climate of this basic attitude will be swiftly and sensitively responsive to unexpected reactions and events for which surgeons who think in stereotypes may be unprepared.

Does a usual procedure need to be modified in its nature or degree for a particular patient? This question again must be answered by balancing the nature of the disease against a series of factors peculiar to a given patient. Among other things, these include expected longevity in relation to existing pathology, the presence and severity of associated but not necessarily related infirmities, the degree of urgency, deemed essential for preservation of health or life, the success of the physiologic preparation for surgery, an appraisal of the patient's tolerance for the several variations of the available procedural plans, and knowledge of the choice of goals to be sought.

Even as a patient must not be regarded as "average," so the tendency to regard procedures as "routine" must be avoided. The word is often used to indicate a tested and reliable method of approach or to describe a pattern of procedural steps which has proved dependable throughout the years in thousands of individual instances. The word "routine" is allowable in this context, on practical and pragmatic grounds.

But when dealing, as here, with more precise meanings of concepts in terms of their ultimate implications for intellectual and clinical behavior, the word "routine" is best avoided. It suggests an unthinking, undiffer-

entiatling stereotypy in the surgeon's attitude which will, if adopted, obviate any real potential for what is called surgical judgment.

Flexibility must be preserved in even the most ordinary and familiar circumstance new knowledges insights and techniques must be correlated, unexpected variants of familiar surgical situations must be differentiated from their individual application.

In the well-disciplined surgeon who knows he will not violate basic surgical principles and has no impulse to do so there is no need for undeviating adherence to a rigid routine. Such a surgeon will, of course make use of his experience but he does not make a fetish of it. He will be able to respond to the specific needs of the moment, whether ordinary or deviant within the principles of sound surgical practice.

FEAR AND SURGICAL JUDGMENT

There are generally speaking, two kinds of fear common to the surgeon rational fears which are a necessary tool of responsible surgery and irrational fears which are an unnecessary and sometimes dangerous burden.

Rational fear recognizes realistically all potential danger to the patient it keeps the surgeon alert to physiologic changes before, during and after surgery it makes him respectful of tissue attentive to detail, swiftly responsive to subtle changes, careful to avoid complications. He uses rational fear to quicken his purposeful responses and sharpen his skills so that he may avoid catastrophe and meet new problems flexibly when they arise.

Irrational fears are those genuine not to the patient, but to the surgeon. They may immobilize him into a repetition of familiar and "safe" routines insufficient to or irrelevant to the problem at hand. They may spur him to irresponsible innovations or deviations from sound surgical principles. The surgeon may suffer from fears based on an insufficient knowledge of the kind of problem he is dealing with or an insufficient experience of it. In such instance he would do well to obtain assistance from a surgeon better versed in the problem or to refer the patient.

There are a host of other fears arising out of threats to the surgeon's own professional or personal welfare, but having no real pertinence to the condition of the patient fear of criticism, fear because the patient is "important," fear of extensive surgical procedures, fear conditioned by interpersonal

difficulties with colleagues. The presence of any such irrational fear may distort the surgeon's motives presence of mind and coordination to such a degree that his best surgical judgment is impossible. When fear of this sort is a major motivating force it threatens the logical performance of what otherwise might be acceptable surgery.

TECHNICAL SKILL AND SURGICAL JUDGMENT

A surgeon's confidence with tissue comes out of his basic physical coordination, his training and his experience. It is developed by work done in the experimental laboratory, at the autopsy table and in the operating room. Within the limits set by his manual dexterity muscular coordination and total integration as an individual, each surgeon learns when to be gentle how to be strong, he knows or senses the balance of strength and delicacy to be applied in a given instance. The sum of his relationship to tissue may be called a surgeon's technique. There are, of course, different technical styles having equal effectiveness and validity. It is interesting to observe two skilled surgeons, one with a slow plodding manner the other with spectacular rapidity of motion. Whatever their differences they will have in common respect for tissue, meticulous attention to detail and a subordination of their physical procedures to the demands of their surgical judgment.

For although technique is of the greatest importance, it is so not as an end in itself but only as a highly developed instrument of the surgeon's intention. During the war it became evident that technical skill at the operating table can be acquired rather easily by well-coordinated individuals. Often outstanding surgical assistance was rendered by medical technicians who had been stock brokers saloon keepers or salesmen in civilian life. But without a surgeon's education and experience, even these especially skilled people could not be expected to estimate operative risk, to prepare a patient physiologically or psychologically for surgery to know when or when not to operate, to adapt their skills to unfamiliar situations or to carry a patient through a stormy post-operative course.

Recently C. W. Mayo gave clear expression to the relationship between surgical skill and surgical judgment.

Surgical judgment differs from surgical skill which implies the quality of technique that, for the most part, is confined to the time a patient is un-

deregoing an operation. It is during this period that the quality of judgment should be most dominant over the best of technical skills. This statement I put made with any intent to belittle the value of dexterity but rather to place it in proper perspective.

One does not inherit either judgment or skill. They are attained in varying degrees by individual effort. Only in the "top flight" surgeon do they approach eventual perfection in balance. Good surgical judgment has its foundation in a knowledge not only of the basic sciences but also of the social sciences and the arts. The rocks on which to build are obtainable in premedical and medical schools. It is what one does with one's time after finishing medical school that determines one's rating in judgment. Some acquire good judgment the hard way by trial and error at particular expense in all senses, to the patient others acquire it by continuing study of the written word from reliable sources by association with and observation of those of proved good surgical judgment, and by unending constructive criticism of their own work and experience. There is nothing that will hasten one's surgical advancement more amply than will a closed mind.

The surgeon whom I would select to tend my family or one must first know when not to cut then when and where to cut, how to cut, and when to stop cutting.

JUDGMENT IN CERTAIN SPECIFICS OF SURGICAL MANAGEMENT

Experienced surgeons are aware of their responsibility in managing certain aspects of surgical care which may appear less consequential than in fact they are. Other aspects merge into related or allied disciplines and are therefore often left to the responsibility of colleagues or house staff. Whatever the necessary and desirable collaboration with such colleagues as the anesthetist, the internist or members of the house staff the surgeon must regard himself as the fulcrum of responsibility to the patient in a surgical situation.

Decisions about mode and type of anesthesia, for example, cannot be left entirely to the anesthetist; ultimately the surgeon must decide. Unusual reactions to the induction of anesthesia demand the surgeon's decision whether to proceed or to send the patient back to his bed without surgery. Certainly many types of surgery can be postponed advantageously under these conditions.

Problems in judgment associated with so-called standard operative procedures are not necessarily inherent in application of the procedure, but in encountering, recognizing and acting upon unexpected findings necessitating change from the planned attack.

Such postoperative measures as bandaging, splinting and grading of motion may make the difference between success and

failure. In many instances the most trying days for both the patient and the surgeon only begin with the day of surgery. It is in the postoperative period that some of the most perplexing problems in judgment arise. Since the surgeon assumes the responsibility for the patient's welfare by subjecting him to surgery, he must by the same token see the patient through the subsequent period. Nor should discharge from the hospital necessarily be the end of patient-surgeon relations.

In the immediate postoperative period the surgeon must cope with the intricacies of fluid and electrolyte balance; precautions against complications arising either from surgery or from unrelated infirmities by keeping in constant touch with the status of all the vital functions of the body through appropriate physical and laboratory examination; the judicious employment of medications; the timing and grading of alimentation and ambulation, and not least, the encouragement of the patient to cooperate properly. All require judgment.

In the late postoperative period, it is the surgeon's obligation to the patient to himself and to his profession to obtain follow-up information especially for types of surgery in which such information is not well known or in which it may possibly alter his future surgical approach to comparable problems. Much of the present-day knowledge of what to expect from surgery has come from careful judgment exercised in appraisal of results. In turn such information is bound to affect the judgment of other surgeons.

NEWNESS IN SURGERY

The concept of newness often is confused with one of unfamiliarity. One need only reflect on how often rediscoveries are made. Yet the rediscovery is new to the rediscoverer. Familiarity by association constitutes a foundation of experience each surgeon must acquire for himself. Every skill grasped by the young surgeon in his graded program of training is in a sense a rediscovery. For the young man it is a discovery for himself a realization that he is capable of doing something he was not sure he could do the day before.

One type of newness has to do with novel situations confronting a surgeon, perhaps unexpectedly which may require a novel approach to be accomplished by variations of old and established techniques. Occasionally discoveries are made by chance rather than by sagacity but such advances

ordinarily require a background of experience, ability to recognize and utilize a new turn and the skill to employ the discovery to advantage. Chance discovery and pragmatism have their place in surgery as they do in other fields but they merely complement and do not replace sound judgment.

Most surgical advances employ standard techniques with some variations but are made possible by advances in sister fields of physiologic knowledge. Surgeons readily acknowledge the debt owed to other sciences for surgical advances. The gamut spans scientific fields from physiology to physics from immunology to electronics, from chemistry to metallurgy.

A second variety of newness is seen in certain procedures which have proved satisfactory in the originator's hands but nonetheless require a fresh experimental and experiential background by every new team of surgeons, no matter how experienced they may be in other types of surgery. Such is the case for example in the use of oxygenators in open heart surgery.

Experimental types of surgery undertaken under carefully controlled conditions are often reported from large university centers. The surgeon who reads reports of this kind of work can easily become overstimulated and overenthused. Despite warnings by the reporting surgeons on the experimental nature of the work, the reader may tend to accept the new procedure too readily.

It is often difficult to distinguish newness, bigness or firstness from betterness. Also it is important to realize that certain types of surgery cannot easily be done except within certain well-controlled surroundings. To proceed with new surgery on the basis of the printed word alone is injudicious. One cannot expect to acquire from the printed word every detail of technical know-how and every nuance of each phase of judgment. Only by a sensible use of skepticism and by his own observations, experimental work and experience with problems and pitfalls can the surgeon arrive at a surgical philosophy acceptable to himself. Nor is experience with numbers alone a guarantee of correctness. A numerically large experience will have been qualitatively small unless it yields changes for the better by the incorporation of changing concepts, aims toward simplification, safety precautions and advancement of knowledge.

A third type of newness involves the planned employment of an operative procedure for which there is no precedent. It

is assumed that any planned operation which is to be undertaken as an experimental procedure upon a human being is preceded by its performance over and over again in animals or at the autopsy table. To undertake a life-endangering new procedure without a background of experimental work could be considered an unprincipled act. But once all phases of the technique, timing, use of special equipment and teamwork are mastered in experimental trials, and once "dry runs" in the operating room run smoothly a new procedure may be considered for application to a human being. It goes without saying that the patient or legal guardian must be apprised of the experimental nature of the procedure and must agree to its performance.

The odds for success based on the life-improving prospects of the proposed surgery, the surgeon's confidence in himself, his team, and his equipment, an estimate of the patient's chances of withstanding the surgery and the physiologic soundness of the procedure must all be assured within reasonable limits of expectancy. Even after successful performance, new procedures must be considered experimental until long term follow-up studies have proved their value.

CONFLICTING GOALS

But though there are rewarding advances, some defeating or essentially insoluble problems must be faced. Ordinarily therapeutic goals such as the prolongation of life and the alleviation of suffering are adequate criteria and we need only concern ourselves with how best to meet them. However, it is often necessary in surgery to make decisions when these two goals are not confluent at all, but antithetical, when the goals conflict with each other.

If the only known method of alleviating pain involves a radical alteration or destruction of the personality, is it justifiable to act to alleviate it?

If it is known that death can only be postponed in the face of unalterable, intractable pain, is it justifiable to act only to postpone it?

These are among the gravest decisions surgeons are called upon to make. They are not properly matters for surgical judgment so much as they are matters for the judgment of the surgeon. Such problems will remain in the province of philosophy until better tools are developed—mechanical, chemical or intellectual—with which to solve them. Until then surgeons can only . . . for a

leavening of human wisdom with which to season their surgical judgment

Surgical judgment is a distillate of physical, emotional, intellectual and moral capacities of the training and experience which informed and disciplined them of an individual's potential to grow, learn and create of the capacity to apply known principles to new specific situations and of dedication to the welfare of the patient

No one can lay claim to superlatives in these things; one may hope to have some of each and to maintain an effectively pro-

ductive balance among them. If one recognizes with rigorous honesty his own special abilities and limitations, if he remains sensitive to the needs of patients and the demands of the profession, he may lay some claim to participation in the development and understanding of his own surgical judgment.

READING REFERENCE

Mayo, C. W. There Is No Substitute for Surgical Judgment (editorial) *Surgery* 39:1013, 1956

INDEX

- Abscess** examination in primary tract disease, 859
incision, ideal, 555
lymphatic drainage, 1366
nodes, anterolateral, myoaponeurotic junctions, 474
pain in, in appendicitis, 693
cramping, in intestinal obstruction, 739-74
surgery antibiotic prophylaxis in, 60
pneumoperitoneum and, 511
surgical dressings for, 74
umbilical region, embryology 477
upper in lower thoracic injuries, 387
viscera, lymph vessels of 1368
wall, 471-484
abnormalities, congenital, 476
anatomy 474
desmoid tumors, 482
pathologic features, 483
embryology 472, 473
examination, importance of 471
infections, 479
masses vs. peritoneal cavity masses, 481
muscles. See *Muscles of abdominal wall*
musculoaponeurotic deficiencies as factor in hernia, 521
spasm, causes, classification, 480
trauma, 479
tumors, 482
benign, 482
malignant, 483
primary 483
secondary 484
metastatic, 484
wounds, classification, 479
nonpenetrating, 480
penetrating, 480, 507
wounds of, penetrating, 507
surgical treatment, 508
Abductor paralysis, bilateral, 311
Abernathy 17
Abortion, 940
bleeding in, 940
classification, 940
complete, 941
definition, 940
incidence, 940
Abortion incomplete 941
treatment, 94
inevitable 940
infections following, 958
missed, 941
treatment, 94
threatened, 940
treatment, 94
treatment, 941
Abrasion(s) 80
in coronary artery disease 461
Abscess, aheolar 28
anal. See *Anal abscess*
appendiceal, formation, 695
brain. See *Intracranial abscess*
breast, 337
cerebellar 1432
vs. subdural abscess, 1433
cervical, 300
cold, 1139
treatment, 1140
collar-button, of hand, 1187
of foot, 1794
extradural, 1432
vs. subdural abscess, 1433
extraperitoneal, following delivery or abortion, 958
facial, 255
frontal lobe, 1431
intra-abdominal, as cause of postoperative fever 122
intracranial. See *Intracranial abscess*
intraperitoneal, 300
in peritonitis, 493
jaw 235
liver. See *Liver abscess*
lung, 405
in bronchogenic carcinoma, 417
postoperative atelectasis and, 116
mediastinal, 372
nasal septal, 226
of Bartholin's gland, 927
of foot, 1293
orbital, as complication of sinusitis, 229
surgical treatment, 231
pelvic, 501
gonorrheal, 957
operative treatment, 501
rupture, "spontaneous," 501
Abscess, periapical 281
periappendiceal, 500
drainage, 501
operative objectives, 501
symptoms, 501
treatment, 501
peritonsillar 234
pharyngeal, lateral, 234
prostatic 910
retropharyngeal, 234 303
splenic 851
subcutaneous, on dorsum of finger 1187
subcuticular of hand, 1187
subdiaphragmatic, supportive necrosis of diaphragmatic hernia due to, 564
subdural, 1433
etiology 1433
symptoms, 1433
treatment, 1433
vs. intracranial abscess, 1432
subpectoral, 390
subphrenic, 501
clinical picture, 503
diagnosis, 504
etiology 503
in appendicitis, 696
in peritonitis, 493
incidence, 503
pathology 503
postoperative 119
prophylaxis, 505
symptoms, 504
treatment, 505
subcapular 390
subungual, 1189
temporal lobe, 1431
tonsillar 234
Académie Chirurgique de Paris, 15
Accidental injuries, classification, 79
definitive care in, 82
Accidents, care of patient following, 79
mortality 1392
Acetabulum, fractures, 1057
Achalasia, esophageal. See *Megacoelephagus*
Achlorhydria, prolonged, relation to gastric carcinoma, 670
small intestine and, 668
Acid, carbonic, concentration, 130

- Amputation, early advantages 170
postoperative 170
in surgical conditions, 13 4
in thoracic surgical conditions,
13 4
- Anchorage, 70.
hepatic abscess following, 798
incidence 798
- Anchorage, colonic 707
- Ameloblastoma, 83
- Amenorrhea, observation of 77
- Amenorrhea, endocrine, 9 0
- Amblyopia, quaternary in peptic ulcer
6, 2
tertiary in peptic ulcer 6, 2
- Ammonia intoxication syndrome 794
- Annesia following head injury 1397
- Ampulla of Vater carcinoma, 786,
811
- Aspiration(s) 1177 1181
artificial extremities in, 13 1
bandaging of stump in, 13 1
conducting exercise in, general,
13, 2
cap type, elective 1179
for vascular gangrene 1179
pneumothorax technique 1177
heat in, 13, 2
in children, 1180
leak, 1177 1178
non-vascular 1179
of arm, congenital, 1 48
of cervix in chronic endocervicitis,
926
of fingers, congenital, 1 48
of finger tip, 1213
of hand, congenital, 1 48
open, technique, 1177
physical medicine and rehabilita-
tion in, 1370
objectives, 1320
procedures, 1320
positioning in, proper 1370
refrigeration in, local, 1347
research in, 1322
surgical principles, 1177
therapeutic exercise in, 1321
ultrasound therapy in, 1322
vascular 1179
- Amniotic, 700
- Amylase, pancreatic, 807
serum, determination in acute
edematous pancreatitis, 805
- Anabolism, 143
corticoid-withdrawal phase 144
endocrinology in, 143
- Anal abscess, 722
diagnosis, 723
drainage 723
etiology 722
symptoms, 722
- Anal canal, 714-731
anatomy 714
gross, 715
imperforate, 590
fistula associated with, 590
591
infection, 718
in anal abscess and fistula,
722
in anal contracture, 724
in anal fissure 721
in hemorrhoids, 719
stages, 718
lymph vessels, 1368
pruritus, 727
- Anal contracture, 724
- Anal contracture acquired, 724
prevention 725
congenital, 724
types, 7 4
- Anal epithelioma 729
- Anal fissure 721
diagnosis, 721
etiology 721
palliation, 722
surgical treatment, 722
symptoms, 7 1
- Anal fistula, 7, 2
definition, 72, 2
diagnosis, 723
etiology 7
symptoms, 7, 2
treatment 724
- Anal sinus, definition, 722
etiology 72, 2
- Anal stenosis. See *Anal contracture*
- Anal structure. See *Anal contracture*
- Analgesia stage of anesthesia, 160
- Andrews, 12
- Androgens, function of 1 4
in breast carcinoma treatment,
164
in endometriosis, 964
in male infertility 918
in prostatic carcinoma, 915
- Anemia, Cooley's, 852
erythroblastic, 85
hemolytic acquired, 850
primary 846, 848
management, preoperative, 10
Mediterranean, 85
pernicious, relation to carcinoma,
670
postgastrectomy 676
- Anesthesia, caudal, 174
classification, 157
closed technique, 160
conduction, 169
drugs for 170
types, 169
depth of shock and, 93
endobronchial, 166
endotracheal, 166
epidural, 174
disadvantages, 174
field block, 169 175
in fracture treatment, 985
general, precautions, 178
history 10
in burns, 193
in congenital hypertrophic pyloric
stenosis, 576
in esophageal stricture repair 573
in fractures, 984
in gastrointestinal hemorrhage, 632
in shock patients, 94
infiltration, 169 175
inhalation, 162
insufflation technique, 158
intratracheal, history 12
intravenous, history 12
in fracture treatment, 985
local, 169 170
nerve block, 169 175
in fracture treatment, 985
nonvolatile, 157
administration, methods, 159
operating room deaths due to, 176
peridural, 174
disadvantages, 174
peridural block, 169
planes, 160
pneumoperitoneum and, 511
- Anesthesia, premedication in, 177
preparation of patient for 177
rebreathing technique 160
rectal ether history 12
refrigeration, 175
regional, 169
drugs for 170
types, 169
resuscitation in, 179
sacral, 174
saddle block, 177
semiclosed technique 159
signs, 160
spinal, 169 177
advantages, 173
deaths due to, 177
disadvantages, 173
high, 172
history 12
in fracture treatment, 985
low 172
medium, 172
physiologic disturbances in, 173
stages, 160
surgical, stage 160
technique, postoperative atelec-
tasis and, 116
topical, 170
- Anesthesiology 157-181
- Anesthetic(s) absorption, 161
characteristics, 159
choice of for shock patients, 95
elimination, 161
general, 157
administration, methods, 158
local, 170
detoxification, 170
reactions to, 171
central nervous stimulation
type, 172
depressant, 172
volatile, 157
administration, methods, 158
- Aneurysm(s) 1355
arteriosclerotic, of thoracic aorta,
462
arteriovenous, 1435
congenital, of hand, 1242
treatment, 1 43
classification, 1355
congenital, 1433
of sinus of Valsalva, 46, 2
diagnosis, 1356
dissection, of aortic arch, 463
465
false, 1355
in coarctation of aorta, 433
intracranial, 1433
demonstration, 1434
rupture, 1433
mycotic, of aortic arch, 463
of aortic arch, 462
congenital, 462
symptoms, 463
of cerebral arteries, 1433
of circle of Willis, 1433
of sinus of Valsalva, surgical re-
pair 459
of splenic artery 851
of thoracic aorta, 462
symptoms, 1356
syphilitic, of aortic
truncus, of aortic arch, 462
treatment, 1356
true, 1355
ventricular 462
surgical correction, 462

- Acid, duodenal, inhibition, in inhibition of gastric acid secretion, 612
gastric, formation, regulation, 612
secretion, inhibition, 61
stimulation, 610
hydrochloric, secretion, 608
homeostatic in pulmonary tuberculosis, 405
para-aminosalicylic, and streptomycin in pulmonary tuberculosis, 405
phosphatase, blood, in prostatic carcinoma, 913
undecylenic in dermatophytosis, 1295
- Acid-base equilibrium, disturbances, 129
- Acidosis, chloride, 132
hyponatremic in pancreatic fistula, 132
in acute hematogenous osteomyelitis, 1118
metabolic postoperative, 132
renal tubular, 872
respiratory, 13
- Aciduria, paradoxical, of metabolic alkalosis, 131
- Acoustic nerve disorders, 1449
- Acrocyanosis, 1350
- Acromioclavicular joint, dislocations, 1046
- ACTH in acquired hemolytic anemia, 830
in hyperadrenocorticism, 829
in peptic ulceration, 614
in primary thrombocytopenic purpura, 843
in rheumatoid arthritis, pyarthrosis and, 1130-1137
- Actinomycosis bovis infection, 46
- Actinomycosis of chest wall, 391
of jaw, 283
pulmonary, 413
- Adamantinomas, 283
- Adentia, lingual, 908
mesenteric, 513-587
vs. appendicitis, 695
septic, repetitive, 1384
- Adenocarcinoma, gastric, 666
survival rate, 676
- Adenocarcinoma of abdominal wall, 483
of appendix, 698
of bladder, 878
of cervix, 935
of colon, 710
of corpus uteri, 939
of duodenum, 679
incidence, 680
of epidiadymis, 904
of extrahepatic bile ducts, 786
of gallbladder, 784
of kidney, 880
of lung, 418
of ovary primary solid, 969
of pancreatic body and tail, 816
of prostate, 913
of rectum, 729
of small intestine, 690
of stomach, survival rate 676
of testis, 901
of thyroid, 321-322
- Adenofibrosis of breast, 344
- Adenofibrosarcoma of breast, 367
- Adenoid, aplasia, 232, 233
- Adenoidectomy complications, 236
- Adenoidectomy indications, 235
polymyositis susceptibility and, 236
technique, 236
- Adenoma, basophil, 1426
in Cushing's syndrome, 822
bronchial. See *Bromochus adenoma*.
chromophil, 1425
chromophobic, 1425
of adrenal cortex, 830
of biliary tract, 784
of breast, 346
of colon, 706
of duodenum, 679
of hypophysis, 1420
of islets of Langerhans, 816
of kidney, 880
of liver, 795
of pancreatic islet cells in peptic ulceration, 614
of pituitary, 1425
of rectum, relation to rectal carcinoma, 729
of stomach, simple, 663
of thyroid, 321-322
papillary, of colon, 706
perineurial, 912
- Adenomatosis of breast, 341
pulmonary, 419
- Adenomyosis of uterus, 963
- Adenosis sclerosing, of breast, 341
- Adenotomy in adenoidectomy, 236
- ADH, excretion, postoperative, 126
- Adhesions, intestinal obstruction by, 756
peritoneal, 505
clinical picture, 506
etiology, 505
pathology, 506
prevention, 506
treatment, 506
- Adrenal cortex. See also *Adrenal glands* and *Adrenal medulla*
anatomy, 821
extracts in shock prevention, 93
hormones produced by, 124-139
in adrenocortical surgery, 832
postoperative increase in, 124
hyperfunction in Cushing's syndrome, 822
hyperplasia. See *Hyperadrenocorticism*.
hypertensive vascular disease and, relation, 833
in breast cancer treatment, 836
in catabolism, 139
in irreversible shock, 91
in prostatic cancer treatment, 836
inefficiency, 833
tumors, 830
adrenalectomy in, 833
classification, 830
in sexual precocity, 919
pathology, 830
- Adrenal glands, 820-841. See also *Adrenal cortex* and *Adrenal medulla*.
anatomy surgical, 820
embryology, 820
failure, as cause of hyponatremia, 149
postoperative, 154
hyperfunction in peptic ulceration, 614
- Adrenal medulla. See also *Adrenal cortex* and *Adrenal glands*.
anatomy, 820
- Adrenal medulla, in catabolism, 140
tumors, 838
- Adrenal-pituitary relations in Cushing's syndrome, 822
- Adrenalectomy "biologic," in regression of metastatic mammary carcinoma, 351
in breast carcinoma, 364-377
results, 838
in hyperadrenocorticism, 837
in hypertension, 834
changes in heart size following, 835
in prostatic cancer, 836
results, 837
subtotal in adrenal cortical hyperplasia, 833
- Adrenogenital syndrome, 825
congenital, 918
Cushing's syndrome and, mixed syndrome, 826
diagnosis, 827
differential diagnosis, 827
etiology, 825
symptoms, 826
- Aero-otitis, 243
- Aeroculaplasia, 2
- Agammaglobulinemia, effect on wound healing, 38
- Age as factor in hernia, 520
- Aged, burns in, treatment, 196
- Air cold, movement, in cold injury, 198
draft, 399
intraoperative, following surgery, 311
passages, foreign bodies in, 315
residual, studies, 401
studies in cranio-cerebral injuries, 1414
swallowed, in intestinal obstruction, 736
- Alcohol, ethyl, 167
injection in trigeminal neuritis, 1446
restriction in peptic ulcer, 622
- Aldosterone, mobilization, 125
postoperative increase in, 124
- Aldosteronism, primary, 827
- Alexandrian School, 2
- Alimentary canal, 571-758
duplications, 583
diagnosis, 585
location, 585
symptoms, 586
treatment, 587
malformations, congenital, 571-597
- Alkalosis, hypochloremic, 130
hypokalemic, in postoperative patients, 148
in primary aldosteronism, 827
metabolic, postoperative, 130
- Allen's test for occlusion of radial or ulnar artery, 1346
- Allis method in hip dislocation, 1094-1096
- Alpha-prothion, 169
- Aluminum hydroxide in peptic ulcer, 622
- Alveolar cell carcinoma, 419
- Alveolar, dental, abscess, 282
carcinoma, 270
jaw invasion by, 284
upper structures, 274
osteomyelitis, 282
- Ambulation, crutch, 1310

- Appendicitis, differential diagnosis, 694
epiploic, 915
peritonitis and, 489
postoperative care, 697
profound features, 693
treatment, 696
varieties, 696
vs. acute gonorrheal salpingitis, 936
with abscess formation, treatment, 696
with general peritonitis, treatment, 696, 697
without gangrene and rupture, treatment, 696
- Appendix, 692-698
adenocarcinoma, 698
carcinoid, 698
location, 692
lymph vessels, 1368
mucocoele, 698
tumors, 698
- Appendix testis, torsion, 900
vs. acute epididymitis, 903
- Arachnodactylum in hand, 125
- Arachnoiditis, 1430
- Arch(es) aortic. See *Aorta, arch*.
of foot, 1259
zygomatic, fractures, 277
- Arched in intracranial operations, 1429
- Arm(s). See also *Extremities*
amputations, congenital, 1248
levels, 1177-1178
artificial, 1321
constrictions, congenital, 148
function, prosthetic replacement and, 1180
fusion of elements of, 1250
- Arnold-Chiari malformation, 1477-1484
- Arrest, cardiac. See *Heart arrest*
- Arthroblastoma, 966
differential diagnosis, 966
symptoms, 966
treatment, 967
- Arterial diseases, obliterative, chronic, treatment, 1354
- Arterial stipes, 1351
- Arteriosclerosis obliterans, 1352
- Arteriosclerotic aneurysms of thoracic aorta, 462
- Arteriovenous fistulae. See *Fistulae, arteriovenous*
- Artery(ies) 1344-1361
abdominal wall, 476
brachial, injury in Volkmann's contracture, 1221
bronchial, anatomy 400
cardiac, great, transposition, 446
carotid, common, left, origin from innominate artery 436
originating from right aortic arch, 436
cerebral, aneurysms, 1433
coronary disease, 460
surgical correction, 461
innominate, aberrant, esophageal constriction by 574
left common carotid artery origin, 436
beating from, 436
large, wounds, 1348
mammary implantation into myocardium in coronary artery disease, 461
internal, ligation in coronary artery disease, 461
- Artery(ies) obstruction, organic, 1351
pulmonary anatomy 399
radial, occlusion, Allen's test in, 1346
splenic aneurysm, 851
subclavian, right, origin from left of aortic arch, 436
ulnar, occlusion, Allen's test in, 1346
- Arthritis of foot 1291
pyogenic acute. See *Pyarthrosis, rheumatoid of foot*, 1291
therapy pyarthrosis and 1130-1137
vs. pyarthrosis, 1133
tuberculosis, of foot, 1294
- Arthrodesis in tuberculosis, of elbow 1146
of foot and ankle 1145
of knee 1145
of shoulder 1146
of wrist, 1147
triple in calcaneal fractures, 1083
in flatfoot, 1277
in pes cavus, 1278
- Arthrodesis in pyarthrosis, 1135
- Articular cartilage in pyarthrosis, 1179
- Ascleus in fibroma of ovary 968
- Asthenia, history 4
- Asphyxia during anesthesia, fatalities due to, 176
traumatic 388
- Aspiration, joint, in pyarthrosis, 1134
of gastrointestinal contents, postoperative atelectasis and, 116
suction, direct, in intestinal obstruction, 750
- Astereognosia in head injuries, 1402
- Astrocystoma, 1423, 1424, 1425-1442
- Astypole, management, 468
- Atelectasis, 386-408
in bronchogenic carcinoma, 416
postoperative 115
Atlas, dislocation on axis, 1053
- Atroventricular communis, 442
surgical treatment, 444
- Atrium, absent septum, 441-442
embryology 442
septal defects, 441
classification, 441
development aspects, 442
surgical treatment, 443
symptoms, 442
single, 442
- Atropine, in craniofacial injuries, 1417
in peptic ulcer 622
sulfate in thromboembolism, 1342
- Atropinization, excessive, postoperative atelectasis and, 116
- Audiograms, in measurement of threshold of hearing, 247
in Meniere's disease, 49
speech, 47
- Audiometry speech, in Meniere's disease, 250
- Auer, 12
- Aureomycin. See also *Tetracyclines*.
in gonorrhea, 927
in surgical infections, 57
- Auriculotemporal syndrome, 275-1450
- Autografts in burns, 195
- Anticonvulsant blocking agents in irreversible shock prevention, 90
- Averin, 167
- Avulsion fracture of extensor tendon of fingers, 998
- Avulsions, 81
- Axilla, metastasis of mammary carcinoma to 340
- Auxiliary nerve, injuries, 1456
- Axial, dislocation of atlas on, 1053
of balance of foot, 1264
of leverage of foot, 1264
- Azygos lobe 409
- Babinski sign in head injuries 1404
- Bacillary dysentery 707
- Bacillus anthracis infection, 44
- Bacillus, gram-negative, in infection 45
tubercle, in infection, 44
- Bacteracin, toxic reactions of 56
- Bacteria, antibiotic resistant, 53
drug-resistant, reduction in control of hospital infections, 58-60
escape in intestinal obstruction, 738
gram-positive, in infection, 43
in bile in cholecystitis, 771
role in wound infection, 5
toxins of, in peritonitis, 489
in wound healing, 33
- Bacterial factor in circulatory insufficiency 90
- Bacteriophage typing of *Staphylococcus aureus*, 44
- Baker's cyst, 1171
- Baker exclusion operation, 644
- Balance axis of foot, 1264
maintenance, 1264
- Balanitis, 894
- Balanoposthitis, 894
- Balanus sign in splenic rupture, 845
- Balloon sphygmomanometer test in recurrent ulcer 654
tamponade in bleeding esophageal varices, 793
- Ball-valve prosthesis in aortic insufficiency 459
- Bancroft exclusion operation, 644
in peptic ulcer 646
- Bandaging of amputation stump 1321
- Bands, intestinal obstruction by 756
peritoneal, 505
- Banti's disease 852
- Bar median, 912
- Barber-surgeons, 14
- Barber's hair granuloma of, 1204
- Barbiturates, 168
classification, 168
- Barium enema in intestinal obstruction, 743
in volvulus, 701
- Barker Fordyce, 12
- Bartholin's gland, abscess, 927
cyst, 927
- Bartholinitis, 973
- Basal cell carcinoma. See *Carcinoma, basal cell*
- Baseball finger 1218
- Basophil adenoma, 14.6
in Cushing's syndrome, 822
- Basophilic, pituitary. See *Cushing's syndrome*
- Bassini inguinal hernioplasty 544
- Baths, contrast, 1305
paraffin, 1305
- Battle's sign in head injuries, 1406

- Angina, Ludwig's, 256, 304
pectora, pain relief in, 1472
Plaut Vincent, 234
- Angiocardiography in aortic arch aneurysms, 463
in atrial septal defects, 443
in tetralogy of Fallot, 440
in transposition of great vessels, 446
in ventricular septal defects, 445
- Angiography in craniocerebral injuries, 1414
in intracranial aneurysms, 1434
in intracranial tumors, 1428
- Angiomas, capillary of hand, 1242
treatment, 1243
cavernous, of hand, 1242
treatment, 1243
of lip and mouth, 265
gastric, 664
intracranial, 1435
of nose, 1170
of colon, 708
of epididymis, 904
of mouth, 264
of salivary glands, 287
- Angiosarcomas of colon, 798
- Angle, cerebellopontine, tumors, 1425
- Angulation in intestinal obstruction, 736
- Anhydrous, carbonic, in hydrochloric acid secretion, 609
- Anhydrohydroxyprogesterone in amenorrhea, 940
- Anilerdine, 169
- Animal bites, care of, 65
- Ankle, dislocations, 1098, 1107
fractures, 1076
abduction, 1078
adduction, 1078
compression, 1080
tuberculosis, 1145
- Ankylosis of jaw, 283
false, 283
- Anoci-association, 12
- Anorectum, 714-731
anatomic relationships, 717
anatomy, 714
developmental, 715
blood supply, 716
defecation, 715
inflammatory disease, etiology, 718
pathogenesis, 718
lymphatics, 716
malformations, 589
fistulae associated with, 591
symptoms, 590
treatment, 590
types, 590
nerve supply, 716
neurophysiology, 714, 717
surgical spaces, 717
venous drainage, 716
- Anosmia in head injuries, 1403
- Anrep, 12
- Antacids as peptic ulcer, 622
- Antibiotic-parasite relationship, 54
- Antibiotics, 52
action, 52
in vitro, factors influencing, 53
bacterial resistance to, 53
broad-spectrum, in preoperative bowel preparation in colonic cancer, 711
cross-resistance, 53
- Antibiotics, diffusion, 54
host-drug relationships and, 55
host-parasite relationships and, 55
immunologic response alterations by, 55
in accidental wounds, 85
in appendicitis, 495
in burns, 59
in fever of undetermined origin, 504
in infection, 51
in intestinal obstruction, 749
in intestinal stenosis or atresia, 579
in irreversible shock prevention, 90
in osteomyelitis, acute hematogenous, 1118
in pulmonary tuberculosis, 405
in sinusitis, 229
in surgical infections, importance, 56
in traumatic wounds, 59
microbial flora alteration by, 55
prophylactic, in abdominal surgery, 60
in genitourinary surgery, 60
in head and neck surgery, 59
in obstetric and gynecologic surgery, 60
in surgery in clean fields, 60
in surgery in contaminated fields, 59
in thoracic surgery, 49
indications, 59
therapy combined, indications for, 41
principles, 51
topical, 54
three response alterations by, 55
toxic reactions of, 56
- Anticholinergic agents in peptic ulcer, 622
- Anticoagulants, postoperative, 1340
rectus sheath hematoma and, 481
- Antidurems, postoperative, 140
- Antidiuretic hormone secretion, postoperative, 126
- Antilumen globulin test in acquired hemolytic anemia, 850
- Antimicrobial agents, 52
- Antimicrobial therapy. See *Antibiotics*.
- Antisepsis, history, 4
- Antithyroid drugs in hyperthyroidism, 319
- Antituberculous drugs, 405
- Antral acid inhibition in inhibition of gastric acid secretion, 612
- Antral phase of gastric acid secretion, 610
- Antrectomy in chronic gastric ulcer, 655
in peptic ulcer, 638
physiologic considerations, 643
surgical considerations, 643
with vagotomy in peptic ulcer, 643
- Antrotyphonic glands, physiology, 632
- Antrostomy in maxillary sinus inflammation, 230
- Antrum, importance of, 610
- Anuria, electrolyte deviations in, 134
in urinary tract disease, 856
- Anus. See *Anal canal*.
- Aorta, arch, aneurysms, 462
rupture, 465
surgical treatment, 463
symptoms, 463
types, 462
- Aorta, arch, anomalies, 435
double, 435, 436
esophageal constriction by, 574
right, left common carotid artery originating from, 436
with ligamentum arteriosum, 436
coronation, 433
adult, 433
aneurysms in, 433
collateral circulation in, 434
etiology, 433
infantile, 433
murmurs in, 434
operation, indications, 435
postductal, 433
preductal, 433
symptoms, 434
treatment, 435
results, 435
types, anatomic, 433
insufficiency, 457
aortic stenosis and, 455
classification, 457
free, 457
surgical correction, 459
criteria, 457
regurgitation, 457. See also *Aortic insufficiency*
aortic defects, 472
bicuspid, in, 433
murmur in, 433
pulmonary congestion in, 433
treatment, 433
stenosis, 454
acquired, surgical correction, 456
aortic insufficiency and, 455
calcification in, 455
diagnosis, 455
mitral disease and, surgical correction, 457
surgical correction, 456
open vs. closed technique, 457
symptoms, 455
types, 454
valvular congenital, 454
surgical correction, 456
thoracic, aneurysms, 462
valve, anatomy, 454
valvulitis, rheumatic, 455
Aortic rock, 457
- Aortography, retrograde, in aortic arch aneurysms, 463
- Ape hand, 1454
- Apakia, 222
- Aphasia, catatonic and, in head injuries, 1402
in head injuries, 1401
- Apnea of hand, 1248, 1250
- Apnea, occurrence in anesthesia, 161
- Aponeurosis, plantar, 1461
- Apophysitis, calcaneal, 1290
- Appendectomy in acute nonspecific mesenteric lymphadenitis, 114
in mesenteric adenitis, 687
- Appendices epiploicae, 515
- Appendicitis, 692
acute, incidence, 694
peritonitis in, generalized, treatment, 694
local, 694
localized, treatment, 694
cause, 692
chronic, 694
clinical picture, 693
complications, 695

- Rosetti's metastatic deposits, 1167
 saccular. See *Varicellar home of foot, accessory* 1277
 sarcomas of hand, 146
 reticulum, primary 1161
 structure 1112
 tuberculosis. See *Tuberculosis of bones and joints*.
 tumors, 1148
 benign, of fibro-osseous origin, 1157
 shes, 1149
 by inclusion or direct invasion, 1148-1169
 cartilagenous, 1148
 classification, 1148
 malignant, shes, 1150
 vs. acute hematogenous osteomyelitis, 1117
 metastatic deposits, 1148
 of hand, 144
 of osseous origin, 1148, 1163
 of osseous origin, 1148-1149
 osseous, 1148
 primary types, 216
 response 1148-1160
 Bony prominences, protection during operation, 106
 Boot, pax, in peripheral vascular diseases, 1323
 Ulna's pax, in chronic venous insufficiency 1341
 Borborygmi in intestinal obstruction, 739
 Borrelia vincenti infection, 44
 Bottom, tailor's, 1109
 Bouchacourt's sign, 483
 Bougie esophageal perforation with, 594
 Bowditch in chemical burns of esophagus, 600
 Bowel, care in spinal injuries, 1440
 large, injuries, treatment, 510
 obstruction, 732
 small, injuries, treatment, 510
 obstruction, 737
 in peritonitis, 493
 sounds, absence in intra-abdominal penetration, 507
 in nonpenetrating abdominal wounds, 509
 Brachial artery injury in Volkmann's contracture, 1221
 Brachial plexus, block, 175
 injuries, 1456
 Bradycardia in head injury 1397
 Brain, abscess. See *Intracranial abscess*.
 arteriovenous anomalies, 1435
 concomitant, 1394
 contusion, 1394
 in head injuries, 1393
 edema, in cranio-cerebral injuries, 1394
 laceration, 1394
 in head injuries, 1393
 stem, herniation in head injuries, 1402
 syndromes in head injuries, 1395
 tumors, 1425
 vascular lesions, 1433
 Brachial cleft anomalies, embryologic derivation, 292
 Branchiogenic carcinoma, 299
 diagnosis, 300
 treatment, 300
 Branchiogenic cysts, 29. See also *Neck, anomalies lateral*
 Branchiogenic fistulae, 292. See also *Neck anomalies lateral*
 Branchiogenic sinuses, 292. See also *Neck anomalies lateral*
 Breast(s) 36-368
 abscess, 337
 adenofibroma, 344
 adenoma 346
 adenomatous, 341
 anatomy 328
 anomalies, developmental, 336
 carcinoma. See *Carcinoma of breast*
 changes in menstrual cycle 379-383, 333
 cystadenocarcinoma, papillary 347
 cystic disease 326, 339
 disorders due to abnormal physiology 338
 dysplasia, 339
 carcinoma and, relation, 342
 cyst removal in, technique 343
 cystic phase 341
 ductal papillomatosis in, 342
 exudative phase 342
 hyperplastic changes, 341
 pseudoneoplastic, 344
 incidence, 327-328, 341
 phases, 340
 physical findings, 341
 proliferative changes, 341
 symptoms, 341
 treatment, 343
 enlargement, male, 339
 examination, tactus eroditus in, 377
 technique 330
 fat necrosis following trauma, 337
 fibroadenoma, 376, 344
 incidence, 377-328
 foreign bodies in, 337
 hyperplasia, acinar atypical, 344
 neonatal, 338
 prepubertal, 338
 pseudoneoplastic, 344
 hypertrophy 339
 intraductal papilloma, 345
 lesions, incidence, 327
 inflammatory 337
 lipomas, 346
 lump, dominant, absence breast dysplasia treatment in, 343
 breast dysplasia treatment in, 343
 lymphatics, 1371
 cutaneous, 329
 intramammary 329
 neoplasms, benign, 344
 common, 345
 physiology 329
 pyogenic infections, 337
 removal. See *Mastectomy*
 roentgenography 335
 sarcoma, 367
 self-examination, 327
 technique, 335
 supernumerary 336
 transillumination, 335
 trauma, 346
 cancer and, 337
 tuberculosis, 338
 tumors, biopsy excisional, 360
 malignant, 346
 Breathing, capacity maximum, 401
 exercises in thoracic surgical conditions, 13-4
 Breathing, mouth-to-mouth, in artificial respiration, 179
 Brenner tumor, 964
 Bronchidrosis, 1351
 Bromophalein test in peptic ulcer hemorrhage 670
 Bronchiectasis, 410
 acquired, 410
 bilateral, 412
 congenital, 410
 postoperative atelectasis and, 116
 pseudo, 411
 reversible, 411
 types, 410
 Bronchiolar carcinoma, 419
 Bronchogenic carcinoma. See *Carcinoma, bronchogenic*
 Bronchography in bronchiectasis, 411
 in bronchogenic carcinoma, 419
 Broncholithiasis, 408, 414
 Bronchopleural fistula in empyema, 39
 Bronchoscopy in bronchogenic carcinoma, 419
 Bronchus(s) adenoma, 421
 carcinoid, 471
 cylindromatous, 471
 types, 41
 anatomy 398
 arteries, anatomy 400
 diseases, 398-425
 middle lobe, syndrome, 406
 veins, anatomy 400
 Brooke Army Medical Center for mules in burn shock, 189
 Brown-Sequard syndrome in intraspinal tumors, 1443
 Bruises. See *Contusions*
 Brunner's glands, physiology 63.
 Bubo, chancroidal, 908-974
 Buccopharyngeal fascia, 302
 Buck's fascia, 891
 Buck's traction in intertrochanteric fractures, 1068
 Boerger-Allen exercises in peripheral vascular disorders, 1323
 Boerger's exercises in ischemia of extremities, 1348
 Bude exteriorization operation in pilonidal disease, 728
 Bulbocavernosus reflex in urinary tract disease, 858
 Bull and Cooley inguinal hernioplasty 544
 Bullae emphysematous, 409
 Bullet wounds, of head, 1408
 of larynx and trachea, 308
 of neck, 307
 Benson, 1278
 etiology 1279
 treatment, 1280
 Bunionectomy Keller in osteoarthritis of foot, 1287
 Bunionette, 1281
 Burns, 187-197
 anesthetics in, 193
 antibiotic therapy in, 59
 autografts in, 195
 blood transfusions in, 192
 cardiovascular system in, changes in, 186
 chemical, of esophagus, 599
 chronic, 194
 classification, 183
 clinical course 185
 Curling's ulcer in, 186

- Beaumont, 63^o
 Bell, John, 18
 Bell's palsy 1448
 Belly jelly 513 698
 Bennett's fracture-dislocation of thumb 1000
 Berkow barn scale, 185
 Bezerra, 627
 vs. gastric carcinoma, 670
 Bicarbonate, plasma, alterations in, 130
 Bier's hyperemia in fractures, 994
 Bigelow Henry J 11
 Bigelow method in hip dislocation, 1095 1096
 Bile, bacteria in, in cholecystitis, 771
 chloride losses in, 129
 composition, 760, 761
 duct, atresia, congenital, 764
 common, exploration at cholecystectomy incidence of stones, 780
 stones in. See *Cholelithiasis*
 extrabiliary, carcinoma, 786
 formation by liver 762
 in peritoneal cavity 499
 metabolism, gallstone formation and, 767
 potassium losses in, 129
 sodium losses in, 129
 stasis, gallstone formation and, 767
 Biliary atresia, 764
 Biliary fistula, postoperative, 814
 Biliary system, 759-799
 anatomy 760
 trauma to, 764
 Biliary tract disease, gallstones and, relationship, 766
 infection, gallstone formation and, 767
 hematogenous, chronic cystitis and, 771
 inflammation, strictures due to 782
 injury strictures due to, 782
 operations in relapsing pancreatitis, 807
 strictures due to inflammation and injury 782
 tumors, 784
 benign, 784
 malignant, 784
 Bilirubin, formation by liver 762
 Billroth, 19 25 700
 Billroth I, gastric resection in gastric carcinoma, 672, 673
 gastrooduodenostomy 639 640, 641
 operation in gastric carcinoma, 671
 in peptic ulcer 625
 Billroth II operation. See *Gastrojejunostomy*
 Biopsy, excisional, in breast tumors, 360
 gastroscopic, in gastric atrophy 629
 in gastric, 628
 in gastric ulcer 618
 incisional, in breast carcinoma, technique, 340
 needles, in breast carcinoma, 356
 technique, 356
 pericardial, in pericarditis, 449
 scalene node, in bronchogenic carcinoma, 419
 Biopsy, suction, in fibroids, 944
 surgical, in breast carcinoma, 359
 incisions, 369
 Bitex, animal, care of, 65
 human, infection, 34 1199
 Bladder anomalies, 870
 care in spinal injuries, 1440
 disease pain relief in, 1473
 exploration in urinary tract disease, 862
 eustrophy 479 870
 injuries, 871
 in pelvic ring fractures, 1056
 lymph vessels, 1369
 neck obstruction, types, 913
 neurogenic, 881
 automatic, 884
 diagnosis, 886
 treatment, 886
 types, 886
 autonomous, 886
 characteristics, 885
 in spinal shock, treatment, 883
 motor 887
 sensory 887
 uninhibited, 884
 obstruction, causes, 864
 pain in urinary tract disease, 855
 perforation, 871
 tumors, 878
 classification, 878
 diagnosis, 879
 etiology 878
 pathology 879
 symptoms, 879
 treatment, 879
 wounds, treatment, 510
 Blast injury of lung, 388
 Blastomycosis, pulmonary 413
 Blebs, emphysematous, 409
 Bleeding. See *Hemorrhage*
 Blind rehabilitation therapy definition, 1298
 Blister purulent, of hand, 1187
 Block, 700
 Block anesthesia, 169
 drugs for 170
 types, 169
 brachial plexus, 175
 epidural, 174
 disadvantages, 174
 extrabiliary, in portal hypertension, 791
 field, in fracture treatment, 945
 nerve, 169 175
 in fracture treatment, 985
 pendular, 174
 disadvantages, 174
 Blood, administration in nonpenetrating abdominal injury, 509
 chemistry determinations in peritonitis, 493
 dyscrasias, rectus sheath hematomas and, 481
 flow. See *Circulation*
 in burns, chemical changes in, 187
 in peritoneal cavity 499
 increased flow in wound healing, 24
 intravenous, in shock treatment, 93
 loss during operation, measure ment, 106
 estimation, 92
 replacement, 97
 plasma, composition, 127
 pressure in head injuries, 1398
 regulation, autonomous nervous system and, 1466
 Blood, replacement in wounds of large arteries, 1348
 study in preoperative routine 97
 supply local, wound healing and, 35
 of anorectum, 716
 wound healing and, 37
 transfusions in burns, 192
 in gastrointestinal hemorrhage, 651
 ultraviolet radiation of 1313
 vascular system in cold injury 200
 vessels in wound cleansing, 64
 tumors 1420
 of hand, 1 41
 volume, normal, shock prevention and, 97
 whole, injection in fractures, 994
 Blood acid phosphatase in prostate carcinoma, 913
 Blow-bottles in thoracic surgical conditions, 1324
 Bochdalek, foramen of hernia through, 562
 treatment, 566
 Bodily depletion, preoperative, effect on convalescence, 150
 Body(ies) carotid. See *Carotid body*
 foreign. See *Foreign bodies*
 osteocartilaginous, loose, in knee joint, 1089 1092
 rice, in tuberculous tenosynovitis, 1200
 temperature, 198
 weight, in assessing hydration, 127
 in post-traumatic metabolism, 142
 Boeck's sarcoid pulmonary 414
 splenomegaly in, 831
 Bohler traction in calcaneal fractures, 1085
 Bohler walking iron in fractures, 995
 Boll of hand, 1186
 Bone(s) 978-1181
 atrophy in fractures, 979
 carcinoma, metastatic 1167
 cortex, drilling in fractures, 995
 cuboid, fractures, 1085
 cuneiform, fractures, 1085
 cyst, benign, 1160
 of hand, 1246
 aneurysmal, 1246
 disease in hyperparathyroidism, 324
 giant cell tumor 1162
 grafts, autogenous, 995
 dial, 995
 homogeneous, 995
 in femoral neck fractures, 1063
 in fractures, 994 995
 in osteomyelitis, 1127
 inlay 994 995
 intramedullary 995
 onlay massive, 994 995
 osteopneumal, 995
 sliding, 995
 to jaw 284
 grammatous lesions, 1166
 healing of 40
 in cold injury 201
 infections, acute and chronic, 1111-1147
 lunate aseptic necrosis, spontaneous, 1008
 dislocation, 1006
 metastases of mammary carcinoma to, 350

- Carcinoma, gastric, exploratory laparotomy in, 677
 family history of, 669
 gastric ulcer and, relation, 620
 incidence, 663-665
 age, 667-668
 sex, 667-668
 location in relation to survival rate in total gastrectomy, 674-676
 mucinous, 666
 survival rate, 676
 operability, 671
 operation, 671
 mortality, 675
 palliative treatment, 675
 pathology, 667
 precursors, 669
 survival rates, 676
 suspected, in peptic ulcer surgical treatment, 675
 symptoms, 668
 earliest, 669
 treatment results, 676
 vs. gastric ulcer, 656
 vs. peptic ulcer, 617-618
 in radiation injuries, 206
 irradiation, 1237
 treatment, 1233
 of abdominal wall, 483
 implantation, 484
 of adrenal cortex, neoadenoma, 831
 of ampulla of Vater, 786, 811
 of bladder papillary, 879
 squamous cell, 879
 of bone metastatic, 1167
 of breast, 326-346
 acute, 347
 axillary metastasis, 350
 levels, 354
 biologic predeterminism, 351
 biopsy in, incisional technique, 360
 surgical, 359
 incisions, 360
 cell types, 347
 clinical in extirpation, initial, 355
 clinical laboratory procedures in, 357
 clinical-pathologic correlation, 346
 comedo, 347
 distribution in breast, 349
 ductal, 346
 dysplasias of breast and, relation, 342
 etiologic factors, 346
 grading, 348
 growth, local, evolution, 349
 patterns, 348
 in lactation, treatment, 362
 in male, 366
 in pregnancy, treatment, 362
 incidence, 327-328
 inflammatory, 347
 inoperability, 359
 inoperable artificial menopause induction, 363
 hormone treatment, 363
 management, 363
 steroid metabolism alterations in, 363
 internal mammary metastasis, 350
 lobular, 348
 mastectomy in, radical, 360
- Carcinoma of breast, mastectomy in, radical, postoperative care, 361
 radiation therapy following, 361
 routine postoperative roentgenotherapy in, 348
 simple intensive roentgenotherapy in, 358
 McWhorter technique in, 358
 medullary, 347
 metastatic, distant, 350
 patterns, 348
 regional, 350
 patterns, 348
 subareolar lymphatic, 350
 needle biopsy in, 356
 technique, 356
 of acinar origin, 348
 operability, 359
 Paget type, 347
 physical evaluation of patient, 357
 primary size, axillary metastasis and, relation, 352
 psychologic evaluation of patient, 357
 radiographic studies, 356
 retrobus, 347
 stages, 352, 354
 clinical, 354
 incidence, 352
 pathologic, 354
 trauma and, 357
 treatment, adrenal cortex in, 836
 adrenalectomy in, 837
 results, 838
 cortisone acetate in, 836
 "curative" results, 365
 end results, 365
 in lactation, 362
 in pregnancy, 362
 methods, 357
 palliative, 362
 results, 366
 untreated, natural history, 352, 353
 of cervix, 933
 bleeding in, 933
 classification, 934
 control, 933
 diagnosis, 936
 discharge in, 979
 factors in, predisposing, 934
 grading, 934
 histologic changes, 934
 incidence, 933
 metastasis, 934
 Papanicolaou technique in, 936
 pneumative, 934
 treatment, 937
 squamous cell, 934-935
 symptomatology, 935
 treatment, 936
 of cheek, 270
 of colon, 708
 adenomatous polyps and, relation, 706
 blood-borne metastases, 710
 colloid, 710
 diagnosis, 709
 direct designation of cancer cells in, 710
 epidermoid, 710
 etiologic, 709
 following ileostomy, 704
 incidence, 708
- Carcinoma of colon, intramural extension, 710
 location, 710
 lymphatic metastases, 710
 multiplicity of, 710
 spread, 710
 surgery, historical aspects, 700
 surgical extirpation, 711
 results, 713
 symptoms, 709
 vs. diverticulitis, 705
 of common bile duct, 786, 938
 of corpus uteri, diagnosis, 939
 discharge in, 979
 histopathologic changes, 939
 incidence, 938
 survival rates, 940
 symptoms, 938
 treatment, 939
 of cystic duct, 786
 of esophagus, 602
 diagnosis, 602
 surgical treatment, 603
 results, 604
 of external auditory canal, 251
 of fallopian tubes, discharge in, 979
 of female genitalia, 979
 of gallbladder, 784
 diagnosis, 784
 incidence, 784-786
 symptoms, 784
 treatment, 785
 of hand, 1, 32
 etiologic factors, 123
 incidence, 1232
 of jaw, 270, 284
 metastatic, 284
 of kidney, 880
 diagnosis, 880
 embryonal, 880
 pathology, 880
 prognosis, 881
 symptoms, 880
 treatment, 881
 of larynx, 313
 of lip, basal cell, 267
 "field-fire", 267
 "invaded", 267
 of multicentric origin, 267
 raised, 267
 types, 267
 basosquamous, 268
 squamous cell, 267-268
 treatment, 268
 of liver, 796
 metastatic, 796
 primary, 796
 of lymphatic system, 1388
 vs. Hodgkin's disease, 1387-1388
 of middle ear, 251
 of nose, 238
 of ovary, 969
 incidence, 969
 metastatic, 972
 primary cystic, 970
 symptoms, 969
 types, 969
 of pancreas, 811
 nonoperative, 814
 operation, results, 814
 treatment, 813
 vs. hepatitis, 811
 of pancreatic body, 815
 of pancreatic head, 811
 of pancreatic tail, 815

- Burns, deep dermal. See *Burns*, *second degree*
- depth, 183
- dressings, 193
- endocrine changes in, 186
- extent, 184
- examination, 185
- first-aid treatment, 193
- first degree, 183
- clinical course, 185
- flame, of face, 196
- flash, 183
- fluid in, 186
- full thickness. See *Burns*, *third degree*
- gastrointestinal tract changes in, 186
- hematologic changes in, 187
- homografts in, 195
- in convalescence, endocrine and metabolic features of, 193
- in elderly, 196
- in infants, 196
- infections in, 187, 192
- lymph vessel injuries in, 1373
- nutrition in, 191
- of eyeball, 223
- of eyelid, 218
- of face, 196
- tracheotomy in, 193
- of hand, 196, 1220
- abrasive, 1213
- of perineum, 197
- partial thickness. See *Burns*, *second degree*
- radiation. See *Radiation injuries*
- respiratory tract changes in, 186
- scars, in carcinoma of hand, 1235
- second degree, 183
- clinical course, 185
- of hand, 1220
- treatment, late, 194
- shock, 188
- fluid requirements in, estimation, 189
- treatment, 188
- initiation, Brooke Army Medical Center formula in, 189
- Evans formula in, 189
- skin grafting in, 195
- superficial, 183
- partial thickness, 183
- treatment, late, 194
- surfaces, pathologic changes, 183
- systemic alterations following, 186
- tetanus in, 192
- third degree, 183
- clinical course, 185
- of hand, 1220
- treatment, exposure method, 193
- initial, 192
- later, 194
- special, 196
- urinary tract changes in, 187
- vittulus in, 191
- whole thickness, treatment, late, 194, 195
- Bursa(e) pharyngeal, 232
- radial, 1191, 1192
- in tenosynovitis, treatment, 1196
- trauma to, 1109
- tuberculous, 1147
- tumors, 1109, 1171
- ulnar, 1191, 1192
- in tenosynovitis, treatment, 1196
- Bursitis, hydrocortisone in, 1110
- suppurative, 1109
- Buttonhole dislocation of hand, 1219
- By-pass, cardiopulmonary. See *Cardiopulmonary by-pass and Circulation extracorporeal*
- CAFFEINE sodium benzoate in cranio-cerebral injuries, 1417
- Calcaneonavicular ligament, 1261
- Calcaneovalgus, congenital, 1270
- Calcaneus, fractures, 1082
- Calcification in aortic stenosis, 455
- of gallbladder in chronic cholecystitis, 773
- Calcium, carbonate, in gallbladder in chronic cholecystitis, 773
- in peptic ulcer, 622
- deposition in fractures, 990
- deposits in burnae, 1110
- metabolism, parathyroid hormone in, 323
- source, local, in fracture healing, 993
- Calculus(1) formation, causes, 871
- 872
- pancreatic, 808
- prostatic, 915
- renal, recurrence, 877
- treatment, 876
- salivary system, 286
- stag-horn, treatment, 876
- ureteral, treatment, 875
- urethral, treatment, 875
- urinary, 871
- clinical findings, 874
- colloid-crystalloid imbalance and, 872
- colloid effects in, 873
- concentration of solutes and, 872
- diagnosis, 874
- dissolution, 877
- epithelial changes and, 872
- etiologic study, 873
- foreign body and, 872
- infection and, 873
- mucelle formation and, 872
- nitric, 871
- prophylaxis, 877
- recurrence, 877
- prophylaxis, 873, 877
- stasis and, 873
- subepithelial injury and, 871
- treatment, 875
- vs. appendicitis, 694
- vesical, treatment, 873
- Caldwell-Luc procedure in maxillary sinusitis, 231
- Calf muscles, paralysis, foot deformities in, 1248
- Callouses, plantar, 1282
- Callos formation, 40
- in fractures, 990, 991
- Caloric tests in Meulens disease, 250
- Calorie(s) intake, in anabolic phase of convalescence, 146
- post-traumatic, 142
- Canal, anal. See *Anal canal*
- Cancer. See *Carcinoma*
- Candida albicans in thrush, 928
- Canker sore, 256
- Cannulation, venous, technique, 69
- Capillaries, lymphatic, anatomy, 1362
- Capillary circulation, testing, reactive hyperemia in, 1345
- Capitulum, fractures, 1029
- Capsule, joint, bared tumors arising from, 1234
- Capsulitis, adhesive, contubernalous, tuberculous of shoulder and, 1146
- Carbohydrate, absorption, alterations following gastrostomy, 626
- balance in postoperative patient, 124
- metabolism, liver and, 742
- Carbon dioxide, excess, prevention during operation, 107
- tension, determination, 401
- Carbonic acid concentration, 130
- Carbonic anhydrase in hydrochloric acid secretion, 609
- Carbonic of hand, 1187
- Carcinoids, malignant, of colon, 708, 710
- of appendix, 698
- of colon, 793
- of small intestine, 690
- Carcinoma, adenocystic basal cell, of scalp, 215
- alveolar, 270
- jaw invasion by, 284
- alveolar cell, 419
- bronchiogenic, 299
- diagnosis, 300
- treatment, 300
- bronchiolar, 419
- bronchiogenic, 414
- adenocarcinoma, 418
- anaplastic, 419
- bronchiolar, 419
- cellular type, clinical course and, 419
- cigarette smoking and, relationship, 415
- classification, 418
- diagnosis, 419
- epidermoid, 418
- etiology, 415
- extension to contralateral side, 420
- incidence, 414
- inoperability signs, 420
- origin, multicentric, 419
- pathology, 418
- physical findings, 416
- roentgenologic diagnosis, 416
- squamous, 418
- symptoms, 415
- treatment, 411
- results, 421
- vs. chronic suppurative pneumonia, 408
- buccal, 470
- chimney-sweep, 897
- death in U.S., relation to total deaths, 667
- epidermoid, of nasopharynx, 241
- of skin of abdominal wall, 483
- gastric, 666, 667
- classification, pathologic, 666
- death due to, 667, 676
- sex incidence, 676
- diagnosis, 669, 670
- differential diagnosis, 670

- Children, supracondylar humeral fractures in, 1032
- Chinney-sweep' cancer 897
- Chiruria Alama*, 8
- Chlorophenicol, in osteomyelitis, acute hematogenous 1118
- in surgical infection, 58
- toxic reactions, 56
- Chloride acidosis, 132
- balance in postoperative patient 124, 126
- cobalt, test in sweating pattern alterations, 1303
- in gastrointestinal discharges, evaluation, 147
- losses, in bile 129
- in gastrointestinal fluids 128, 129
- in pancreatic juice, 129
- metabolism, post traumatic, 142
- Chloroform, 164
- history 11
- Chloroma, 1165, 1388
- Chloroprocaine 171
- Chlorpromazine in shock prevention, 91
- Chlortetracycline See also *Tetracyclines*
- in surgical infections, 57
- Cholangitis, 764
- Cholecystectomy combined with common duct exploration, incidence of choledocholithiasis, 780
- in acute cholecystitis, 777
- in chronic cholecystitis 773
- medical, in gallbladder carcinoma, 783
- Cholecystitis, acute, 770, 774
- diagnosis, 774
- etiology 775
- operation, choice of 777
- production, experimental, 776
- treatment, 776
- vs. appendicitis, 693
- chronic, 769
- bacteria in bile in, 771
- calcification of gallbladder in, 773
- chemical irritation and, 773
- cholesterosis and, 772
- diagnosis 770
- differential diagnosis, 773
- hematogenous biliary tract infection and, 771
- hydrops of gallbladder in, 773
- infection in, role of, 771
- lymphatic spread of infection in, 772
- metabolic disturbances in, role, 772
- milk of calcium gallbladder and, 773
- pathogenesis, 770
- symptoms, 769
- treatment, 773
- Cholecystography in cholecystitis, 770
- in gallstones, 769
- Cholestyramin, 762
- Cholelithotomy in acute cholecystitis, indications, 777
- Choledochofistulotomy as obstructive jaundice, 813
- Choledocholithiasis, 766, 778. See also *Gallstones*
- gallbladder disease and, 778
- Choledocholithiasis, gallstones and 778
- incidence 778
- at cholecystectomy combined with common duct exploration, 780
- surgical removal, 779
- Choledochotomy combined with cholecystectomy incidence of choledocholithiasis, 780
- in acute cholecystitis, 777
- Choledochus, carcinoma, 786
- Cholelithiasis. See *Gallstones*
- Cholesteatoma, 1425
- Cholesterolemia in chronic otitis media, 246
- Cholesterol metabolism, chronic cholecystitis and, 777
- gallstones and, 767
- Cholesterosis in chronic cholecystitis, 773
- Chondroblastoma, benign, 1153
- malignant, 1153
- Chondroma, 1151
- of chest wall, 396
- Chondromalacia patellae, 1089
- Chondromatosis of joint synovium 1171
- Chondromyxoid fibroma, 1153
- Chondromyxosarcoma, mediastinal 378, 380
- primary 1155
- Chondrosarcoma, 1154
- of jaw 284
- secondary 1155
- Chordee, 893
- Chordoma, bone involvement in, 1170
- Chordotomy anterolateral, 1469
- in chronic obliterative arterial disease 1355
- Chorioepithelioma of testis, 901
- Choroid, injuries to, 222
- plexus, papilloma, 1424
- Chromaffinoma. See *Phaeochromocytoma*
- Chromophil adenoma, 1425
- Chromophobe adenoma, 1425
- Chromoxal in neuromuscular disorders, 1302
- tests in peripheral nerve injuries, 1457
- Chyle thorax, 1373
- Cicatricial stenosis of larynx, 31
- Cigarette smoking, and lung cancer relationship 415
- postoperative atelectasis and, 116
- Circle of Willis, aneurysms, 1433
- Circulation, capillary testing, ree the hyperemia in, 1343
- collateral, in coarctation of aorta, 434
- Main test of, 1345
- distribution of, oxygen consumption and, 89
- extracorporeal, 427. See also *Cardiopulmonary bypass*
- cardiac arrest induction in, 429
- complications following, 428
- hyperinflation in, 428
- hypothermia and, 430
- in aortic arch aneurysms, 464
- in aortic insufficiency 459
- in aortic septal defects, 433
- in great vessel transposition, 447
- partial, 429
- ventricular fibrillation in, 429
- Circulation insufficiency bacterial factor in, 90
- balance between vasoconstrictor material and vasodilator material and 90
- irreversible shock following, 88
- of extremity increasing, regional sympathetic denervation in, 1347
- response to stress, 88
- reversal, in coronary artery disease, 461
- status, in fracture healing, 993
- Cirrhosis, gallstones and, 766
- in portal hypertension, 790
- portal, effect on wound healing, 39
- vs. extrabiliary block, 792
- Clinoids intracranial, 1435
- Clinical chyl, anatomy 1364
- Classification, intermittent, 1344
- Clavicle fractures, 1045
- first aid, 982
- Clavus, of toes, 1283
- Clawfoot, 1277
- Cleft, branchial, anomalies, embryologic derivation, 292
- Cline, 17
- Cloquet, lipoma theory 518, 451
- Clostridia in wounds, 34, 45
- toxigenic, 44
- Closure delayed, of contaminated wounds, 312
- skin, materials for 112
- Clothing, mechanism in wound healing, 24
- Clubfoot, congenital, 1266
- treatment, 1268
- Coagulation, abnormalities, rectus sheath hematoma and, 481
- necrosis, of muscle in cold injury 201
- Coal spots in irradiation carcinoma, 1234
- Coarctation of aorta. See *Aorta, coarctation*
- Cobalt, chloride test in sweating pattern alterations, 1303
- radiation in cervical cancer 937
- radioactive, in bladder tumors, 880
- Cocaine history 12
- Coccaloidomycosis, pulmonary 413
- Coccydynia, 1061
- Coccyx, fractures, 1060
- Codeline, 169
- Cocoon, 472, 484
- Coffee, restriction in peptic ulcer 622
- Coin lesions in bronchogenic carcinoma, 416
- Cold, susceptibility to 198
- Cold abscess, 1139
- treatment, 1140
- Cold fingers, hereditary 1350
- Cold injury blood vascular system in, 200
- bone in, 201
- cartilage in, 201
- classification, 199
- degrees, 199
- freezing type, 199
- local, 198-204
- predisposing factors in, 198
- muscle in, 201
- nerve tissue in, 201
- nonfreezing type, 199
- pathology 199
- racial incidence, 199
- signs, 202
- skin in, 201

- Carcinoma, of paranasal sinuses**, 238
 surgical treatment, contraindications, 240
 of penis, 895
 of peritoneum, metastatic, 51
 of pharynx, 235
 of prostate, 913
 benign prostatic hyperplasia and, 915
 differential diagnosis, 914
 prostatectomy and, 915
 symptoms, 913
 treatment, 914
 adrenal cortex in, 836
 adrenalectomy 836
 results, 837
 corticosteroid acetate in, 836
 of rectum, 729
 anal, 730
 diagnosis, 729
 extirpation, historical aspects, 700
 palliative treatment, 730
 pathology, 729
 symptoms, 729
 treatment, 730
 of salivary gland, jaw invasion by, 284
 of scalp basal cell, 215
 squamous cell, 215
 of seminal vesicles, 916
 of skin of abdominal wall, 483
 of testes, 901
 of thyroid, angioinvasive, encapsulated, 321 322
 treatment, 322
 classification, 321
 in children following radiation treatment of Graves disease, 319 320
 medullary, 321 322
 treatment, 322
 nonpapillary, 321 322
 papillary, 321
 treatment, 322
 undifferentiated, 321 322
 treatment, 322
 of tongue, 271
 of trachea, squamous cell, 314
 of urethra, male, 895
 squamous cell, 878
 treatment, 878
 of vulva, 975
 leukoplakia of vulva and, relation, 976
 radiation, 1232
 treatment, 1233
 simplex, gastric, 666
 survival rate, 676
 rectal, 729
 verrucous, 270
Carcinomatous, peritoneal, 710
Cardiac glands, physiology 632
Cardiopulmonary by-pass, 427 See also *Circulation, extracorporeal*
 in cardiac tumors, 467
 in pulmonary stenosis, 439
 in subaortic stenosis, 456
 in ventricular aneurysm, 462
Cardiovascular disease, acute cholecystitis and, treatment, 777
Cardiovascular system in burns, pathological changes in, 186
Caries sicca, 1139
 of shoulder 1145
Carotid artery common, left, origin from innominate artery 436
 from right aortic arch, 436
Carotid body tumors, 298
 clinical features, 298
 diagnosis, 299
 embryologic derivation, 298
 pathology, 298
 treatment, 299
Carotid cavernous fistula, 1435
Carotid sheath, 306
 infection, 306
Carotid sinus syndrome, 1466
Carpal tunnel, median nerve compression in, 1458
Cart, surgical dressing, standard equipment for 71
Cartilage(s) articular in pyarthrosis, 1129
 healing of, 40
 in cold injury, 202
 semilunar fractures, 1089 1090
 mucoid cysts, 1089
Cast, hanging, in humeral shaft fractures, 1037
Castration in carcinoma of prostate, 914
Catabolism, 139
 endocrinology in, 139
Catastrophic states in head injuries, 1402
Catheterization, cardiac. See Heart catheterization.
 endobronchial, 166
 in male technique, 862
 in urinary tract disease, 862
Cauda equina injury 1459
Caudal anesthesia, 174
Causalgia, 1452, 1466
Cauterization in chronic endocervicitis, 926
Cavernous, 894
Cecostomy historical aspects, 700
 in carcinoma of colon, 712, 713
 in intestinal obstruction, 751
Cecum, lymph vessels, 1368
Celiotomy ideal, 545
Cells, chief, physiology 631
 interstitial, of Leydig, 897
 parietal, 608
 physiology, 631
Cellular shock, hyponatremia as manifestation of 150
Cellulitis, acute, following thrombophlebitis, 1338
facial, 255
 of paratubercular soft tissues vs. pyarthrosis, 1132
 orbital, as complication of sinusitis, 229
 post-traumatic, of breast, 336
 vs. acute hematogenous osteomyelitis, 1117
Celux, 8
Cephalhematoma, 214 1406
Cephalic phase, of digestion, 632
 of gastric acid secretion, 610
Cerebellopontine angle, tumors, 1425
Cerebellum, 1419-1439
 abscess, 1432
 tumors, 1424
Cerebral arteries, aneurysms, 1433
Cerebral hemispherectomy in focal epilepsy 1438
Cerebral hemispheres, tumors, 1423
Cerebritis, 1413
Cerebrospinal fluid, accumulations, subdural, 1411
 treatment, 1418
 diversion in hydrocephalus, 1484
 findings, in cerebrospinal fluid, 1405
 in head injuries, 1405
 in intracranial tumors, 1426
 in intraspinal tumors, 1443
Cerebrospinal injuries, cerebrospinal fluid findings in, 1405
Cerebrum 1419-1439
 abscess, vs. subdural abscess, 1433
 Cervical abscess, 300
 Cervical cord, injuries, 1439
 Cervical fascia, anatomy, 301
 Cervical glands, enlargement, in focal infection, 235
 tuberculous, 1384
 Cervical infections, deep, 300
 etiology, 300
 Cervical rib, 1458
 Cervical spine, fractures and dislocations, 1051
 Cervical traction, 1314
 Cervix, amputation, in chronic endocervicitis, 926
 cancer. See *Carcinoma of cervix.*
 dilation, in dysmenorrhea, 937
 lymph vessels, 1369
 nabothian cysts of, 925
 structure, in chronic endocervicitis, 925
Chain, wheel, 1311 1319
Championnier, Lucas, 5
Chancro in female, 974
Chancroid in female, 974
Chancroidal bubo, 908, 974
Charaka, 2
Charcot, 779
Cheek, carcinoma, 270
Chelosis, 256
Chemical burns of esophagus, 599
Chemical irritation, chronic cholecystitis and, 773
 in carcinoma of hand, 1235
Chemodectoma, of carotid body. See Carotid body tumors.
 of middle ear, 251
Chemotherapy in intestinal stenosis or atresia, 579
 in pulmonary tuberculosis, 405
 in radical mastectomy, 361
 in sinusitis, 229
Cheselden, William, 4, 16
Chest. See also Thorax.
 "fall," 385
 funnel, 388
 surgical dressings for, 73 74
 wall, unstable, 385
Cheyne-Stokes respirations in head injuries, 1398
Chian's disease in portal hypertension, 791
Chief cells, physiology 631
Children, acute hematogenous osteomyelitis in, 114
 amputations in, 1180
 fractures of radius and ulna in, 1017
 Graves disease in, radiation treatment and, 319
 lateral humeral condyle in, fractures, 1030
 pyarthrosis in, 1138

- Craniofacial injuries, classification, 1395
 clinical manifestations, 1395
 clinical-pathologic correlations in, 1406
 consciousness, state of and alterations in, 1396
 diagnostic aids in management, 1413
 dizziness in, 1400
 headache in, 1399
 infection in, 1412
 intracranial hemorrhage in, 1408
 laceration, penetration in, 1405
 management, diagnostic aids in, 1413
 mechanism, 1393
 neurologic manifestations, 1401
 operation in, indications, 1415
 management, 1418
 pathologic conditions in, summary 1396
 pathology 1394
 treatment, 1416
 vital functions in, 1397
 vomiting in, 1400
- Craniofacial dysmorphism, 15
 Craniofacial fistula, repair 1418
 Craniofacial syndrome, 14.5 1426
 Craniofacial syndrome, 215 1480
 diagnosis, 1481
 surgical treatment, 1482
- Craniofacial lesions, surgical drainage for 73
- Cranium bifidum, 1478
- Cricocarytoides, paralysis, 311
- Cries, night, 1139 1141
- Cric, 12
- Cric procedure as peptic ulcer 642, 643
- Cross-resistance of antibiotics, 53
- Cruentous ligaments of knee. See *Ligaments, cruentous*
- Cruentous injuries, open, 81
- Cruentous in wound healing, 30
- Crotch ambulation, 1310
- Crotch gait, four-point alternate sequence, 1311
 sequence, 1311
 swinging, 1311
 three-point, sequence, 1311
 tripod, sequence, 1311
 two-point alternate, sequence, 1311
 types, 1311
- Crutches, selection, 1310
 types, 1310
 use of, training, 1311
- Crouchfield tongue in cervical vertebrae fractures, 1051
- Cryptorchidism, 898
 cause, 898
 diagnosis, 899
 orchiectomy in, 900
 orchiopexy in, 900
 treatment, 899
- Crypts, paraurethral, infection, 927
- Cryptoscopy multiple, in anal fissure, 722
- Crystalline concentrations, high, as cause of hyponatremia, 149
- Cuboid bone, fractures, 1083
- Cul-de-sac puncture in ectopic pregnancy 946
- Culdocopy in ectopic pregnancy 946
- Cullen's sign in ectopic pregnancy 946
- Cuneiform bones, fractures, 1083
- Cups, metal, in femoral neck fractures, 1064
- Curare 169
- Curvature, in carcinoma of corpus uteri, 939
 medical, 950
- Curling ulcer 635
- Current, direct, in neuromuscular disorders 1301
 faradic, in neuromuscular disorders 130
 tetanizing, in neuromuscular disorders, 1301
- Curve strength-duration, in neuromuscular disorders, 1302
- Cushing head dressing, 73
- Cushing's syndrome, 822
 adrenogenital syndrome and, mixed syndrome 876
 diagnosis 823
 etiology 822
 symptoms, 823
 treatment, 832
- Cut-downs, venous, in esophageal atresia repair 573
 technique, 69
- Cut wounds of neck, 307
- Cut-throat wounds, 307
- Cyanosis, in atrial septal defects, 442
 in pulmonary stenosis, 437
 in tetralogy of Fallot, 440
 in transposition of great vessels, 446
- Cycle, redox, in hydrochloric acid secretion, 609
- Cyclopropane, 163
- Cylindromas of scalp, 214
- Cyst(s) adrenocortical, 831
 air giant, 409
 as cause of osteomyelitis of jaw 81
 Baker's, 1171
 blue-domed, 341
 branchogenic, 292. See also *Nest, anomalies lateral*.
 cartilaginous, of joints, 1171
 colloid, 1423
 corpus luteum, 966
 dental, 283
 dentigerous, 283
 dermoid, inclusion, submental, 296
 of epididymis, 904
 of lips and mouth, 264
 of mesenteries and omentum, 514
 of ovary 969
 of peritoneum, 512
 echinococci, pulmonary 412
 endometrial. See *Endometriosis*.
 enteric. See *Alimentary tract duplications*.
 eosinophilous. See *Alimentary tract duplications*.
 epidermoid, of hand, 1231
 epithelial, of scalp, 214
 follicle, pilaris, 966
 hydatid of mesenteries and omentum, 514
 of spleen, 850
 pulmonary 412
 hygroma. See *Hygroma coli cyclic*.
 in breast dysplasia, removal, technique, 343
- Cyst(s) mucoid of semilunar cartilage 1089
 mucous, of hand, 146
 subcutaneous, of cervix 925
 neuroenteric, 1480
 of Bartholin's gland, 927
 of bone benign, 1160
 of hand 1246
 aneurysmal, 146
 of esophagus, 601
 congenital, 574
 reduplication, 574
 of lips and mouth, 264
 of lung, congenital 409
 echinococcus, 412
 hydatid 412
 of mesenteries, 514. See also *Alimentary tract duplications* and *omentum, dermoid*, 514
 enterogenous, 514
 lymphatic, 514
 congenital, 587
 of omentum, 514
 congenital, 587
 of ovary twisted, vs. appendicitis, 695
 of pancreas. See *Pancreas cysts* of spleen. See *Spleen cysts* of pericardial, 447
 phloidal, 727
 etiology 727
 symptoms, 728
 treatment, 728
 radicular 283
 retention, of pancreas, 809
 root, 283
 sebaceous, of hand, 1231
 of lips, 264
 spring water of mediastinum, 377
 378
 synovial, 1171
 theca-luteum, 962
 thyroglossal, 294
 clinical course, 295
 embryologic derivation, 294
 pathology 795
 treatment, 795
 vs. ectopic thyroid tissue, 296
 urachal, 478
 vitelline, 478
- Cystadenocarcinoma, of pancreas, 809
 papillary of breast, 347
 pseudomucinous, of ovary 971
 serous, of ovary 970
- Cystadenoma lymphomatous, papillary of salivary glands, 287
 288
 of ovary. See *Ovary cystadenoma*, of pancreas, 809
- Cystic disease of breast, 326
- Cystic ducts, carcinoma, 784
- Cystic hygroma. See *Hygroma coli cyclic*
- Cystitis, vs. acute prostatitis, 910
 vs. appendicitis, 694
- Cystocele, 951
 etiologic factors, 951
 symptoms, 952
 treatment, 952
 vs. enterocoele, 568
- Cystosarcoma phyllodes, 367
 origin, 345
- Cystoscopy in neurogenic bladder 883
- Cytologic studies in esophageal carcinoma, 602

- Cold injury symptoms, 202
tendons in, 202
treatment, 203
- Cold therapy 1306
in orthopedic disorders, 1317
- Colectomy and polypectomy in adenomatous polyps, 707
in carcinoma of colon, 713
in chronic ulcerative colitis, 703
- Colic, abdominal, vs. intestinal obstruction, 745
definition, 735
gastroic, 768
- Cold, chronic, in amebiasis, 70
ulcerative chronic 703
clinical course, 703
diagnosis, 703
treatment, 703
segmental, 704
- Collagenase, production of 34
- Collar-button abscess, of hand, 1187
- Colic fracture, 1009
reverse, 101
rupture of extensor pollicis longus in, 1217
- Colloid carcinoma of colon, 710
- Colloid cyst, 1425
- Colon, 699-714
amebiasis, 702
anatomy surgical, 699
asthenia, 577
carcinoma. See Carcinoma of colon
diverticula, 704
inflammatory diseases of, 702
injuries, 700
lacerations, 700
lymph nodes, 1368
metastatic tumors, 708
obstruction, 741
acute, treatment, immediate, 749
diagnosis, 744
in diverticulitis, 705
treatment, 751
volvulus in, 701
perforation, 700
symptoms, 701
treatment, 701
physiology 699
resection and anastomosis in colonic cancer 711
segmental, in adenomatous polyps, 707
stenosis, 577
subdivisions, 699
surgery historical aspects 699
tuberculosis, 702
tumors, benign, 706
classification, 706
malignant, 708
- Colostomy historical aspects, 700
in advanced rectal structure, 725
transverse, in carcinoma of colon, 713
in intestinal obstruction, 751
- Colpotomy posterior in ectopic pregnancy 946
- Colostoma, short, in cleft lip, repair of, 262
- Comedo carcinomas of breast, 347
- Condomastitis, 338
- Commodo operation, 170
- Complement deficiency wound healing and, 38
- Complement-fixation test, in inflammatory rectal structure, 725
- Complement fixation test, in lymphogranuloma venereum, 908
- Compound F in interdigital neuroma, 1283
- Compression, jugular test of, 1432
- Compression dressings, 73, 74
- Compression fracture, definition, 979
of ankle, 1080
- Concussion, definition, 1396
of brain, 1394
- Conditioning exercises, general, in amputations, 1322
- Condyle, lateral, of humerus, fractures in children, 1030
mandibular fractures, 275
- Condylomata acuminata, 894
latum, 974
- Conization, high frequency in chronic endocervicitis, 976
in cervical cancer 937
surgical, in chronic endocervicitis, 976
- Cornell procedure in peptic ulcer 644
- Consciousness, states of and alterations, in craniocerebral injuries, 1396
- Constantine, 13
- Constantinus Africanus, 14
- Constriction in peptic ulcer 616
- Consultants, surgical, recommendation, 1489
- Contamination, definition, 42
operative, peritonitis and 489
peritonitis secondary to 489
surgical procedures in, 59
antibiotic prophylaxis in, 59
wound, care of 112
- Contraction, muscle, isometric, 1107
of scar phase of wound healing, 23
- Contracture Dupuytren's. See Dupuytren's contracture
- Contracture, of hand, 1221
treatment, 1222
- Volkman's, in supracondylar humeral fractures, 1034
treatment, 1222
vs. Ledderhose's disease, 1293
- Contrast baths, 1305
- Continuity, 80
facial, 252
of brain, 1394
in head injuries, 1393
of frontotemporal junction, 1402
of scalp findings, 1406
- Convalescence, abnormalities, common, 146
alkalosis in, 148
effect of burns on, 153
effect of extensive wounds on, 152
effect of fractures on, 152
endocrine disease in, 154
endocrinology and metabolism in, 138-156
extrarenal losses in, 147
hypokalemia in, 148
infection in, 152
intravenous feeding in, prolonged, 150
late, clinical management, 146
endocrinology of 146
metabolism in, 146
low sodium syndrome in, 149
potassium loss in, 148
renal failure in, 155
surgical, anabolic phase, 144
- Convalescence, surgical, anabolic phase, clinical management, 145
clinical management, 143
- Convulsions, in head injuries, 1404
in intracranial surgery, 1422
in local anesthesia, 172
- Convulsive disorders, 1437 See also Epilepsy
- Cooley's anemia, 85
- Coomb's test in acquired hemolytic anemia, 840
- Cooper Astley 17 18, 1223
- Cord, spermatic. See Spermatic cord
- Cord, spinal. See Spinal cord
- Cordeotomy anterolateral, 1469
in chronic obliterative arterial disease, 1355
- Cordus, Valerius, 11
- Cornea, perforation, 221
- Corns of toes, 1283
- Coronal suture, premature closure, 1481
- Coronary artery disease 460
surgical correction, 461
- Coronary occlusion, 460
- Coronary sinus, ligation in coronary artery disease 461
- Coronary vein, great, ligation in coronary artery disease, 461
- Coronoid process involvement in ankylosis of jaw 283
- Corpora cavernosa, 891
- Corpus Hippocampus, 2
- Corpus luteum, cyst, 966
rupture, vs. appendicitis 685
- Corpus spongiosum, 891
- Corrective therapy definition, 1298
- Cortex, adrenal. See Adrenal cortex
- bone drilling, in fractures, 995
cerebral, excision, in focal epilepsy 1438
ulceration, 1413
- Corticoid-withdrawal phase of anabolism, 144
- Corticotropin in peptic ulceration, 614
- Cortisone, in acquired hemolytic anemia, 850
in chronic gonorrheal salpingitis, 957
in craniocerebral injuries, 1417
in peptic ulceration, 614
in peritonitis, 496
in rheumatoid arthritis, pyarthrosis and, 1130 1137
- Cortisone acetate, in adrenocortical surgery 837
in breast cancer treatment, 836
in hyperadrenocorticism, 829
in prostatic cancer treatment, 836
- Cough, in bronchogenic carcinoma, 415
rectus sheath hematoma and, 481
- Cough-pain test in venous thrombosis, 1337
- Courvoisier's law 812
- Cowper's glands, 916
- Cranial nerves, 1446-1450
injury in head injuries, 1403
section, 1470
- Craniofacial linear in craniosynostosis, 1482
- Craniofacial fistula, repair 1418
- Craniofacial injuries, 1392 1419
associated injuries and, 1405
bullet, 1408

- Downed, 9, 13
 Dorsal spine, fractures, 1033
 Dorsey 18
 Dorsiflexion test in venous thrombosis, 1337
 Dorsiflexor muscles of foot, paralysis, 1285
 Douglas, James, 486
 Douglas, linea semicircularis of 475
 Drain(s), incisional hernia and, 554
 postoperative use 114
 Drainage in hand infections, 1185
 in infection, 50
 in osteomyelitis due to direct contamination, 1126
 in subphrenic abscess, 505
 lymphatic, of abdomen and pelvis, 1366
 of abdominal wall, 476
 of thorax, 1369
 prophylactic, in peritonitis, 493
 sump, as replacement for heavy dressings, 74
 Dressings, burn, 193
 compression, 73 74
 head, Cushing type, 73
 surgical, application, technique, 71
 discarding, 72
 removal, 72
 special types of 72
 Dribbling in urinary tract disease, 856
 Drift, air 399
 Dromedary, 169
 Drop flager 998
 Drop phalangette, 1218
 Drug(s), antithyroid, in hyperthyroidism, 319
 antituberculosis, 405
 perfusion, local, by means of car diopulmonary by-pass, 429
 reactions as cause of postoperative fever 122
 Drug-host relationships, 55
 Drug-resistant bacteria, reduction in control of hospital infections, 58, 60
 Drummer's palsy 1216
 "Dry sponge technique of estimating blood loss in surgery 92
 Duerer infections of vulva, 974
 Duct(s) bile See *Bile duct*
 extrahepatic, anomalies, congenital, 763
 lymphatic, injuries, 1373
 right, 1364
 mammary ectasia, 338
 omphalomesenteric, 472
 anomalies, 477
 persistent, 583 See also *Diver ticulum, Meckel's*
 salivary 285
 fistula, 285
 Skene's, gonorrheal infections of 974
 thoracic, anatomy 1364
 injuries, 1373
 vitelline, 472
 anomalous, 477
 persistent, 583
 Ductus arteriosus, patent, 430
 cardiac catheterization in, 431
 mechanism, 430
 murmurs in, 431
 symptoms, 431
 treatment, 432
 contraindications, 432
 Ductus arteriosus, patent, treatment, mortality rates in, 432
 untreated, complications, 432
 tetralogy of Fallot and 440
 Dumping syndrome, following gastrectomy 626
 following peptic ulcer surgery 660
 following subtotal gastric resection, 639
 Duodenal acid inhibition, in inhibition of gastric acid secretion, 61
 Duodenal secretion, 612
 Duodenopancreaticostomy in annular pancreas, 588
 retrocolic, in duodenal obstruction, 578
 Duodenostomy following gastric resection, 647
 Duodenum, 677-685
 anatomy 607 678
 atrophy, 577
 diverticula, 681
 incidence 681
 sex 681
 location, 681
 surgical treatment, 683
 symptoms, 682
 treatment, 682
 types, 681
 fistulae, 684
 external, 684
 internal, 685
 skin protection in, 684
 injuries, treatment, 510
 lesions, obstructive, 678
 requiring surgery frequency 678
 lymph vessels, 1368
 obstruction, acquired, 678
 annular pancreas and, 803
 congenital, mongolism and, 579
 surgical treatment, 578
 pancreatic juice drainage into, in relapsing pancreatitis, operation to improve 807
 physiology 608
 stasis, 679
 stenosis, 577
 tumors, 679
 benign, 679
 diagnosis, 680
 incidence, 679
 sex, 680
 surgical treatment, 681
 malignant, 680
 classification, 680
 symptoms, 680
 ulcer chronic, treatment, 637
 gastric ulcer and, 635
 differences, 620
 incidence, 606
 location, 615
 perforated, treatment, 649
 vs. appendicitis, 694
 resection, anastomosis following, 639
 roentgenography in, 618
 surgical procedure in, clinical considerations, 645
 results, 625
 selection, factors in, 645
 technical problems, 645
 symptoms, 617
 treatment results, 626
 vagotomy in, 642
 Dupuytren, 16, 1223
 Dupuytren's contracture 1223
 etiology 1225
 pathology 1225
 symptoms, 1, 26
 treatment, 1227
 Durand-Fardell, 786
 Duret 700
 Dynamometer in measurement of muscle strength, 1300
 Dyachondroplasia with hemangioma-tosis, 1152
 Dysentery amoebic, acute, 702
 bacillary 702
 Dysgerminoma, 966
 Dymenorrhea 931 933
 characteristics, 932
 idiopathic, pain relief in, 1473
 primary 931
 secondary 932
 treatment, 93
 Dyspareunia in chronic gonorrheal salpingitis, 957
 Dyspepsia, functional, vs. peptic ulcer 618
 Dysphagia, in esophageal carcinoma, 602
 in esophageal ulceration, 600, 617
 in megacophagus, treatment, 596
 leukemia, 575
 Dysplasia, fibrous, 1161
 diffuse 1161
 monostotic, 116
 multiple 1161
 Dyspnea, in bronchogenic carcinoma, 415
 laryngeal, obstructive, 311
 Dysuria, in urinary calculous disease 874
 in urinary tract disease 856
 EAR(s) 247-252
 agenesis, congenital, 251
 drum, inflammation, myringotomy in, 43
 external, tumors, 251
 malformations, 251
 middle reconstruction, 251
 tumors, 251
 Ebers Papyrus, 2, 1487
 Echinococcosis in fractures, 990
 Echinococcus cysts, pulmonary 412
 Eczema, varicose 1332
 Edema, hyponatremia in, treatment, 150
 in intestinal obstruction, 736
 in stoma ulcer 617
 in varicosities, 1331
 of brain in craniocerebral injuries, 1394
 of glottis, 311
 of larynx, acute inflammatory 311
 noninflammatory 31
 pulmonary in head injury 1397
 scrotal, 896
 wound healing and, 35
 Educational therapy definition, 1298
 in orthopedic disorders, 1317
 Effluage 1309
 Elinhorn, 12
 Eiseberg exclusion operation, 644
 Eiseberg gastrectomy 639
 640
 Eiseberg operation in gastric cancer 671

Cytology exfoliative in broncho-
genic carcinoma, 419
in gastric ulcer 621

Dark Ages, surgery in, 3

Davy Sir Humphrey 10

Deafness, 247

acquired, 248

adhesive, 248

conductive, causes, 247

surgeal treatment, 48

hearing tests in, 247

perceptive, causes, 247

congenital, 247

types, 247

tonic 48

types, 247

vasomotor 248

Deaths, operating room 176

Debridement, in facial wounds, 253

in infection, 50

Decamethosum, 169

Decerebrate rigidity in head injuries,

1404

de Chauliac, Guy 3 9 15 1490

Decompression, intestinal in intes-

tinal obstruction, 746

De-epicardialization in coronary ar-

tery disease, 461

Defibrillation, procedure 109

Defibrillator in cardiac arrest, 468

Degeneration, reaction of, 1301

red, of submucous fibroids, 943

Delusiveness, wound, 113

Dehydration, in acute hematogenous

osteomyelitis, 1118

in obstruction of small intestine,

748

of exposed viscera during opera-

tion, protection from, 106

surgeal, 147

Delirium stage of anesthesia, 160

DeLorme's progressive resistive ex-

ercises, 1307

DeLorme's progressive resistive ex-

ercises, 1307

Demerol, 169

de Mondeville, Henri, 3 8

Denervation, sympathetic. See *Symp-*

parietectomy

Dental cysts, 283

Dentigerous cysts, 283

Depletion, bodily preoperative, effect

on convalescence, 150

Dermatitis, infected, following

thrombophlebitis, 1338

in chronic venous insufficiency

treatment, 1341

Irradiation, 1232

prevention, 1233

varicose, 1332

Dermatophytosis, 1294

Dermoid cyst. See *Cyst dermoid*

Dermoid, mediastinal, 376

Dermoid tumors of abdominal wall,

482

Dexamethasone acetate in

adrenocortical surgery 832

de Stoll, Maximilian, 784

Devegan in trichomonads, 924

Devine atrial excision, 644 645

Devine exclusion operation in pep-

tic ulcer 646

Diabetes, effect on wound healing,

39

Diabetes, postoperative, in obstruc-

tive jaundice, 814

Diaphragm, anatomy 562

elevation, postoperative atelectasis

and 116

eventration, vs. esophageal hiatus

hernia, 566

pelvic, hernias, 567

respiratory hernia. See *Hernia,*

diaphragmatic

Diarrhea, following gastrectomy 626

in stoma ulcer 617

Diastomatomyelia, 1477

Diathermy 1306

apparatus, 1306

Dibucaine 171

Dieffenbach, 18

Diet, in burn patients 191

in peptic ulcer treatment, 622

in preoperative management of

malnourished patient, 98

liquid, for malnourished patients,

99

ulcer 622

Diethylstilbestrol in breast carci-

noma, 564

Digestion, phases, 632

stomach functions in, 613

Diptus quintus varus, 1281

Dihydromorphine, 169

Dilatation, gastric, acute, postopera-

tive, 117

and curettage in carcinoma of

corpus uteri, 939

in ectopic pregnancy 946

in endocrine bleeding, 951

Dilaudid, 169

Diphtheric tonsillopharyngitis, 234

Diplopia in Moiré's disease, 249

Diplopia in intracranial tumors,

1421 1423

Direct current, in neuromuscular dis-

orders, 1301

tetanus ratio in neuromuscular

disorders, 1302

Disarticulation. See *Amputation*

Discharge from female genitalia, 922

Disease, Chauri's, in portal hyperten-

sion, 791

Fothergill's. See *Neuralgia, trigem-*

inal

Gaucher's, splenomegaly in, 852

Graves. See *Graves' disease*

Hirschsprung's, 589

Hodgkin's. See *Hodgkin disease*

Koenig's, 1008

Köhler's, 1290

Ladderbone's, 1293

Letterer-Siwe, 216, 1166

Memere's, 1449

Milroy's, 1376

Niemann-Pick, splenomegaly in,

852

Paget's, of skull, 116

Peyronie's, 895

Dupuytren's contracture and,

1226

Raynaud's, 1349 1463

Thomwaldt's, 232

Disk, intervertebral. See *Interverte-*

bral disk

Dislocation () fractures and, 997-

1100

metatarsophalangeal, 1100

of acromioclavicular joint, 1046

of ankle, 1098, 1107

of atlas on axis, 1053

Dislocation(s) of cervical spine,

1051

of elbow joint, 1027

of foot, midtarsal, 1099

subtalar, 1099

of foot joints, 1099

of hand, buttonhole, 1219

of hip joint, 1094

of knee joint, 1096

of lunule bone, 1006

of metacarpophalangeal joint of

thumb, 999

of patella, 1097

of shoulder 1106

recurrent, 1044

of shoulder joint, 1043

of sternoclavicular joint, 1048

of talus, 1099

of wrist, perilar, 1008

physical medicine and rehabilita-

tion in, 1316

tarso-metatarsal, 1099

Disruption, wound, 112

causes, 113

treatment, 114

Dissecting aneurysms of aortic arch,

463 465

Dissection, and snare method of ton-

sillectomy and adenoidectomy

236

introduction of, 15

neck, in carcinoma of hp 269

suprathyroid, in carcinoma of hp

270

Distention, in intestinal obstruction,

736, 739 740, 742

treatment, 749

postoperative, 117

Dours's, postoperative, 140

Diverticulitis, acute, peritonitis in,

treatment, 494

colonic obstruction in, 705

intestinal obstruction in, 757

location, 704

of colon, 704

medical treatment, 706

surgeal treatment, 706

symptoms, 704

vs. cancer 705

surgery in, indications, 705

Diverterculosis, incidence, 704

location, 704

of colon, 704

etiology 704

symptoms, 704

Diverticulum, duodenal. See *Duo-*

denum, diverticulum

epiphrenic, 597

esophageal. See *Esophagus, div-*

ertic

giant. See *Alimentary tract dupli-*

cations

Mackel's, 477 583

Inflammation, vs. appendicitis,

695

Intestinal obstruction in, 756

symptoms, 583

treatment, 584

of epiphrenic, 904

of pericardium, 447

pathosis, 135

Dizziness in head injuries, 1400

Dys, bases, care of, 65

Dumont, experimental ulcer in,

634

Mann-Williamson, experimental ul-

cer and, 634

- Erythrocyte production by liver 763
- Erythromycin, in osteomyelitis, acute hematogenous, 1118
- toxic reactions, 56
- Fenarch, 9
- Esophagectomy partial, in chemical burns of esophagus, 600
- Esophagitis, 618
- chronic, 600
- nonspecific, 600
- surgical treatment 659
- Esophagogastric anastomosis in treatment of esophageal carcinoma, 603
- Esophagomyotomy in megacophagus, 596
- Esophagoplasty in megacophagus, 596
- Esophagoscope, esophageal perforation with, 598
- Esophagotomy in esophageal carcinoma, 601
- in esophageal lesions, 594
- in esophageal ulcer 617
- Esophagus, 592-605
- achalasia. See *Megacophagus*.
- anatomy 597
- atresia, congenital, 571
- diagnosis, 572
- surgical repair 572
- feeding following, 573
- gastrostomy in, 573
- types, 571, 572
- carcinoma. See *Carcinoma of esophagus*.
- chemical burns, with cicatricial stenosis, 599
- compression, from aortic arch anomalies, 435
- constriction by anomalous vessels in mediastinum, 574
- cysts, congenital, 574
- reduplication, 574
- dilatation and hypertrophy idiopathic. See *Megacophagus*.
- diverticula, 596
- false, 597
- pseudo, 597
- pulsion, 596
- traction, 597
- true 596
- types, 596
- foreign body impaction in, 598
- lesions, esophagotomy in, 594
- inflammatory 599
- location, diagnostic methods, 594
- roentgenography in, 594
- lymphatic vessels, 1371
- perforation, in mediastinitis, 374
- 375
- with esophagoscope or bougie, 598
- reaction and excision in carcinoma, 603
- rupture, spontaneous, 598
- shortness, congenital, 573
- stenosis, 658
- topographic relations, 592
- trauma to, 598
- tumors, benign, 601
- malignant, 601
- ulcer 600, 615
- peptic, 658
- surgical treatment, 658
- roentgenography in, 618
- Esophagus, ulcer surgical treatment, results, 625
- symptoms, 617
- varices, 601
- bleeding, balloon tamponade in, 793
- in portal hypertension, 792
- Estrogens, in amenorrhea, 950
- in breast carcinoma, 364
- in endocrine vaginitis, 928
- in male pseudobermaphroditism, 919
- in prostatic carcinoma, 914
- Etter 164
- history 11
- lens, 163
- Ethics, medical, 1490
- Ethmoid sinuses, displacement of secretion in, 230
- Ethyl alcohol, 167
- Ethyl chloride, 164
- Ethylene 161
- Eunuchoidism, 919
- Eulipistes of Alexandria, 8
- Evans formula in burn shock, 189
- Evertor muscles of foot, paralysis, 186
- Evipal, 168
- Evisceration, wound. See *Blood evisceration*.
- Ewald tube, passing technique, 76
- Ewing's sarcoma, 1163
- vs. primary reticular sarcoma of bone 1164
- Ewing's tumors of hand, 146
- Examination, emergency 63
- Excision, wound, 83. See also *Debridement*.
- Exerche's active, 1306
- anasthetic 1307
- resistive, 1307
- breathing, in thoracic surgical conditions, 134
- Buerger's, in ischemia of extremities, 1348
- conditioning, general, in amputations, 1322
- passive, 1308
- progressive anasthetic, De Lorme's, 1307
- resistive De Lorme's, 1307
- static, 1307
- therapeutic, 1306
- in amputations, 1311
- in lower motor neuron disorders, 1320
- in orthopedic disorders, 1317
- in surgical conditions, 1324
- in thoracic surgical conditions, 1324
- in upper motor neuron disorders, 1318
- Exocoelom, 47
- Exophthalmos, 219
- of hyperthyroidism, cause, 318
- severe progressive treatment, 221
- Exostosis. See also *Osteochondroma*.
- of hand, 1245
- cartilaginous, multiple, 1252
- "Exposure" method of burn treatment, 193
- Extensor muscles of toes, paralysis, 1285
- rupture, 1216
- Extensor pollicis longus tendos, re pair 1254
- Esterification, in colon injuries, 701
- in intestinal obstruction, 751
- in pilonidal disease 728
- Extracuticular fluid, compartment, peritoneum and relation, 488
- composition, 127
- Extradural abscess, 1432
- vs. subdural abscess, 1433
- Extracapsular block in portal hypertension, 791
- vs. cirrhosis, 792
- Extracapsular ducts, anomalies, congenital, 763
- Extremity(ies). See also *Arms and Legs*.
- artificial, 1321
- training in use of 1321
- blood flow increasing, regional sympathetic denervation in, 1347
- heberic management, 1346
- lower amputation levels, 1177
- 1178
- artificial, 1371
- fractures, 1056
- first aid, 983
- function, prosthetic replacement and, 1180
- joints, dislocations of 1093
- lymphatic supply 1366
- lymphedema in, 1374
- spasms, in spinal injuries, 1440
- spontic paralysis, foot deformities in, 1289
- thrombophlebitis, prognosis, 1337
- thrombosis in, sites of 1335
- pain in, relief 1466
- soft parts, tumors, 1148, 1173
- classification, 1173
- malignant, 1173
- surgical dressings for 73
- upper amputation levels, 1177
- 1178
- artificial, 1321
- fractures and dislocations, 997
- 1055
- function, portable pulley apparatus in, 1308
- prosthetic replacement and, 1180
- lymphatic supply 1365
- lymphedema, 1381
- congenital, 1382
- thrombosis, 1342
- veins, 1328
- Eye(s), 17, 224
- anatomy 217
- foreign body in, magnetic, removal, 221
- nonmagnetic, removal, 221
- inspection, 218
- in proptosis, 219
- surgical conditions of emergency 218
- eyeball, burns, 13
- perforation, 222
- eyelids, burns, 218
- lacerations, 118
- Fack, burns, 196
- contusions, 252
- injury compound, 252
- lacerations, 253
- surgical dressings for 74

- Elbow fractures, 1024
 first aid, 982
 joint dislocation, 1027
 Mayer's, 1109
 sprained, 1108
 tuberculosis, 1146
- Elderly burns in, treatment, 196
- Electrical skin resistance measurements in neuromuscular disorders, 1304
- Electrical stimulation in lower motor neuron disorders, 1370
 of denervated muscles, 1312
- Electrocardiography in aortic insufficiency, 457
 in atrial septal defects, 443
 in mitral stenosis, 450
 in transposition of great vessels, 446
- Electrocoagulation in chronic endocarditis, 926
- Electrodiagnosis, data guide for, 1301
 procedures in physical medicine and rehabilitation, 1301
- Electroencephalography in craniocerebral trauma, 1415
 in determining depth of anesthesia, 161
 in intracranial tumors, 1426
- Electrolyte(s), alterations in pancreatic fistula, 132
 balance in peritonitis, 491
 deviations in anuria, 135
 fluid and, management, 123
 loss in intestinal obstruction, 735
 737
 serum, metabolism, post-traumatic, 142
 therapy in peritonitis, 496
- Electromyography in neuromuscular disorders, 1302
 tests in peripheral nerve myoses, 1457
- Electroshock, in defibrillation, 109
 in ventricular fibrillation, 468
- Electrosurgery in skin cancer, 240
- Elephantiasis. See also *Lymphedema neuromatosa*, 1247
- Embolectomy in arterial embolus, 1352
 pulmonary in thromboembolism, 1342
- Embolus, arterial, 1351
 intestinal obstruction in, 754
- Emergency care, preoperative, 97
- Emergency room procedures, 63
- Emphysema, in rib fracture, 1050
- Mediastinal, 369
 387
 obstructive, in bronchogenic carcinoma, 416
 subcutaneous, 387
- Erythema, encapsulated, 391
 pleural, acute, 391
 chronic, 395
 staphylococcus, 392
 subdural. See *Abcess subdural*, thoracic, 391
 tuberculosis, 395
 mixed, 395
- Encephalocele(s), 1478
 morphologic types, 1479
- Encephalography in craniocerebral injuries, 1414
 in intracranial tumors, 1428
- Enchondroma of hand, 1244
- Endarterectomy in coronary artery disease, 461
- Endocarditis, 924
 acute, 924
 chronic, 924
 pathology, 924
 symptoms, 925
 treatment, 925
- Endocervix, hypersecretion, endocrine, 928
 physiology, 925
 removal, 976
- Endocrine-cervical hypersecretion, 928
- Endocrine changes in burns, 186
- Endocrine disease, in convalescence, 154
 in female, 927
- Endocrine factors in gastric acid secretion, 611
- Endocrine system, metabolic response to surgery and, 125
- Endocrine vaginitis, 927
- Endocrinology in metabolism, 139
 in metabolism, 143
 metabolism and, in surgical care, 138-156
 of late convalescence, 146
- Endocrinopathy due to ovarian failure, 947
- Endolymphatic hydrops. See *Meningitis*
- Endometrial cyst. See *Endometriosis*
- Endometriomas of colon, 708
- Endometriosis, 963
 classification, 963
 etiology, 963
 external, 963
 internal, 963
 of umbilicus, 479
 symptoms, 963
 treatment, 964
- Endoscopy peroral, 315
 techniques, 316
- Endothelioma, 1388
 pleural, 397
- Endotracheal anesthesia, 166
- Endotracheal intubation. See *Intubation, endotracheal*
- Enteric, barium, in intestinal obstruction, 743
 in volvulus, 701
- England, surgery in, history, 16
- Enophthalmos, 219
- Enteric cyst. See *Alimentary tract duplications*
- Enteritis, regional, 683
 diagnosis, 689
 operation, type, 690
 symptoms, 688
 treatment, 689
- Enterocoele, 568
 surgical repair, 570
 vs. cystocele, 568
 vs. rectocele, 568
- Enterocolitis, acute, staphylococcal, 702
 regional, 702
 tuberculosis, 702
- Enterogastrostomy, 614
 in inhibition of gastric acid secretion, 612
- Enterogenous cyst. See *Alimentary tract duplications*
- Ependymoma, 14, 4, 1442
- Epicondyle, medial, of humerus, fracture, 1031
- Epidermoid carcinoma, of colon, 710
 of lung, 418
- Epidermoid cyst of hand, 1231
- Epididymectomy in chronic epididymitis, 904
- Epididymis (epididymides), 902
 anatomy, 902
 anomalies, 903
 diverticulum, 904
 infection, 903
 physiology, 903
 trauma to, 903
 tumors, 904
- Epididymitis, acute, 903
 vs. mumps orchitis, 900
 vs. torsion of spermatic cord, 907
 chronic, 903
 gonococcal, 904
 nonspecific, 903
 tuberculosis, 904
- Epididymotomy in acute epididymitis, 903
- Epididymovasectomy in chronic epididymitis, 904
 in male infertility, 918
- Epidural anesthesia, 174
 disadvantages, 174
- Epidual hematoma, treatment, 1418
- Epidual hemorrhage, 1408
- Epigastric hernia. See *Hernia, epigastric*
- Epigastrium, anatomy, 550
- Epilepsy, 1437
 cryptogenic, 1437
 essential, 1437
 focal, 1437
 cortical excision in, 1438
 localization of lesion in, 1438
 idiopathic, 1437
- Epinephrine, elaboration, 821
 function, 838
 in cardiac arrest, 468
 test, in hyperplasmism, 847
 in primary splenic neutropenia, 848
- Ephreptic diverticulum, 597
- Ephyphry, damage, in acute benign toxicous osteomyelitis, 1120
 radial, distal, fracture-dislocation, 1014
- Ephedias, 893
- Epistaxis, 226
- Epithelioma, anal, 729
- Epithelium, healing, 39
 new pigmentation, 30
 regeneration after injury, 30
- Epithelization, 30
- Epulis, 267
 giant cell, 1162
- Equation, Henderson-Hasselbalch, 150
- Equipment mechanical, care of, 69
- Erb's palsy, 1456
- Erection, mechanism, 892
- Erector, 2
- Erosion, acute. See also *Stress ulcer*
 chronic peptic ulcer and, relationship, 636
 gastric, 635
 in peptic ulcer, 613
- Erysipeloid of hand, 1202
- Erythema nodosum vs. pyarthrosis, 1133
- Erythrocyte, count in radiation injuries, 11

- Flatfoot, 1272
hypermobile 1774
spastic peroneal, 1275
treatment, 177
- Flexor pollicis longus tendon, repair 1253
- Flexor tendons of hand, rupture 1220
- Fluorquin in trichomoniasis, 94
- Fluid, balance assessment body weight in, 177
in peritonitis, 491
burn, 186
cerebrospinal. See *Cerebrospinal fluid*.
compartments, 126
boundaries, 126
constituents, 177
electrolytes and management, 123
extracellular compartment, peritoneum and, relation, 488
composition, 127
sodium concentration disturbances, 133
volume disturbances, 132
water, composition, 127
volume sodium ion concentration and, 177
loss during operation, replacement, 106
peritoneal, 486
requirements in burn shock, estimation, 189
in surgical patients, 127
spermatic, examination in infertility 918
synovial, study of in pyarthrosis, 1137
therapy in peritonitis, 496
thick, biochemical status in fracture healing, 993
- Fluorohydrocortisone in adrenocortical surgery 832
- Fluoroscopy emergency in gastrointestinal hemorrhage, 657
- Floodlight, 165
- Focal seizures in head injuries, 1404
- Fold, gastrosplenic, 843
presplenic, 843
- Follicle, graafian, cyst, persistent, 962
rupture, vs. appendicitis, 695
- Folliculitis, of hand 1186
- Foramen, 972, 973
- Foot, 1259-1296
abscess, 1293
agranulosis, 1272
anatomy functional, 1259
arches, 1259
arthritis, 1291
axis, of balance, 1264
of leverage, 1264
bones, accessory 1772
burns, infection, 1293
disorders, 1266
congenital, 1266
fractures, 1080
hind, problems, 1284
immersion, 199
in gout, 1291-1292
infections, 1293
joints, dislocations, 1099
ligaments, 1261
medial dislocation, 1099
motion, 1259
muscles, 1262
dorsiflexor paralysis, 1285
extensors, paralysis, 1286
intrinsic, paralysis, 1288
- Foot, muscles, inventor paralysis, 187
plantar flexor paralysis, 1288
osteoarthritis, 1291-1292
osteochondritis, 1790
osteomyelitis, 1794
paralytic deformities, 1284
positions, 1259
shear 199
subtalar dislocation, 1099
trench, 199
tuberculosis, 1145
tumors, 1293
vascular disease, 1795
- Foramen, of Bochdalek, hernia through, 562
treatment 566
of Winslow 486
ovale patent, pulmonary stenosis with, 437
persistent, with incompetent valve 441
without valve 441
- Forceps, hemostatic, history 9
- Forearm, fracture, first aid, 982
- Forefoot, affections, 1278
- Foreign bodies, definition, 34
impaction of esophagus, 598
in air passages, 315
in breast, 337
in joints, 1136
in peritoneal cavity 499
in pharynx, 235
in stomach, 627
diagnosis, 677
symptoms, 627
treatment, 627
in thorax, 387
intraocular magnetic, removal, 221
paramagnetic, removal, 221
pharyngeal, 235
urinary calculi and, 877
wound healing and, 34
- Formosa, Evans, in burn shock, 189
- Foxbay intradermal test in tularemia, 1203
- Fothergill's disease. See *Neuralgia, trigeminal*.
- Fothergill's sign, 481
- Fowler's solution, carcinoma of hand and, 1235
- Fracture(s) active function and, 991
anesthesia in, 984
evulsion, of extensor tendon of fingers, 998
Bier's hyperemia in, 994
blood, whole, injection in, 994
bone cortex in, drilling, 995
by direct violence, 980
by indirect violence, 980
by muscular action, 980
calcium deposition in, 990
callus formation in, 990, 991
closed, definition, 978
Colles' 1009
reverse, 1012
rupture of extensor pollicis longus in, 1217
committent, definition, 979
complete, definition, 979
complicated, definition, 979
compound. See *Fracture open*.
compression, definition, 979
of ankle, 1000
depressed, definition, 979
dislocations and, 997 1100
- Fracture(s) double vertical of Malgaigne 1059
ecchymosis in, 990
etiologic 979
fatigue 1087
first aid in, 980 991
fixure, definition, 979
greenstick definition, 979
healing, all in, 992, 993
immobilization, post reduction, 991
impacted, definition, 979
in convalescence endocrine and metabolic features, 15
incomplete definition, 979
injury in, treatment, 991
intertrochanteric, of femur 1067
local changes following, 989
march, 1087
marginal, of distal end of radius, 1013
Monteggia 1022
nail, 227
seeding, 994
nonunion, 992
calcium source in, 993
granulation tissue in, 993
local pathologic condition in, 992
prevention, 993
of acetabulum, 1057
of alveolus, upper 276
of ankle, 1076
of calcaneus, 1082
of capitulum, 1079
of cervical spine, 1051
of clavicle 1045
first aid, 982
of coccyx, 1060
of condyle of mandible 275
of cuboid bone, 1085
of cubiform bones, 1085
of distal phalanx, 997
of dorsal spine, 1053
of elbow 1024
first aid in, 982
of femur 1061
head, 1061
lower end, 1071
neck, 1062
shaft, 1069
of fibula, 1073
of foot, 1080
of forearm, first aid, 982
of hand, 997
first aid in, 982
of humerus. See *Humerus, fractures*.
of jaw lower first aid, 982
osteomyelitis and, 281
upper 276
of larynx, 310
of lower extremity 1056
first aid, 983
of lumbar vertebrae transversus process, 1054
of mandible 274
of metacarpal of fingers, 1001
of metacarpal of thumb 1000
of metatarsals of foot, 1087
of middle phalanx, 997
of navicular bone. See *Navicular bone fracture*.
of nose, 279
of odontoid process, 1053
of olecranon process, 1025
of patella, 1071
of pelvic rim, 1056

- Face, tears, 253
wounds, débridement, 253
- Facial nerve, disorders, 1448
preservation in removal of salivary gland tumors, 287
trauma to, 1449
- Factor bacterial, in circulatory insufficiency, 90
- Fallopian tubes, cancer discharge in, 929
lymph vessels, 1369
- Fallop, tetralogy, 439
diagnosis, 440
ductus arteriosus and, 440
surgical treatment, 440
symptoms, 440
- Parade current in neurovascular disorders, 1301
- Fascia, buccopharyngeal, 302
Buck's, 891
cervical, anatomy, 301
digital, anatomy, 1224
excision of in wound cleansing, 84
of abdominal wall, 475
of hand, anatomy, 1224
palmar anatomy, 1224
pelvic, anatomy, 952
pretracheal, 301
prevertebral, 302
Scarpa's, 475
- Facial spaces, of hand, anatomy, 1191
infections, 1191
of neck, 302
- Fasciotomy in wound cleansing, 84
- Fat, in burn diet, 191
in convalescence, gain, 146
metabolism, liver and, 762
pancreas and, 803
necrosis, of breast following trauma, 337
- Fatigue, fractures, 1087
in cold injury, 198
- Focalith, perforation of sigmoid by, 700
- Feces, impaction, intestinal obstruction in, 757
passage, in intestinal obstruction, 739-742
- Feeding, intravenous, prolonged, in convalescence, 150
postoperative, in congenital hypertrophic pyloric stenosis, 577
- Felon, 1189
- Felt's syndrome, 831
- Female, endocrine disorders in, 927
pseudobermaphroditism, 918
reproductive system, 921-977
diseases of, penis in, 929
- Femurization, 832
- Femur fractures, 1061
head, aseptic necrosis, 1062
in hip dislocations, 1096
fractures, 1061
intertrochanteric fractures, 1067
lower end, fractures, 1071
neck, fractures, 1062
shaft, fractures, 1069
subtrochanteric fractures, 1068
trochanteric fractures, 1068
- Penetration operation, technique, 249
- Ferguson inguinal hernioplasty, 544
- Ferguson, William, 17
- Fetal peritonitis, 499
- Fever in appendicitis, 694
- Fever in bronchogenic carcinoma, 416
of undetermined origin, antibiotic in, 504
postoperative, 121
- Fibrillation, ventricular correction, 109
in cardiopulmonary by-pass, 429
management, 468
- Fibrinogen formation by liver, 763
- Fibrosarcoma, gastric, 664
- Fibrosarcoma, breast, 367
of breast, 326, 344
- Fibroids, 942
bleeding in, 942
in pregnancy, 943
incidence, 943
intramural, 943
parasitic, of peritoneum, 512
subcutaneous, 943
subserous, 942
symptoms, 943
treatment, 943
types, 942
- Fibroma(s), cardiac, 466
chondromyxoid, 1153
gastric, 664
of abdominal wall, 482
of colon, 708
of esophagus, 601
of hand, 1236, 1246
of joints, 1172
of lips and mouth, 263
of liver, 795
of lungs, 423
of muscle, 1104
of nasopharynx, 240
of ovary, 968
of small intestine, 690
ossifying, 1157
perineural, 1459
- Fibromyoma of uterus, 942. See also Fibroids.
- Fibroplasia, in wound healing, 27
phase of wound healing, 23
- Fibrosarcoma, cardiac, 466
of bone, 1169
of colon, 708
of joints, 1172
of rectum, 729
of soft parts of extremities, 1175
- Fibrosis, nodular subepidermal, of lips and mouth, 263
- Fibrostenosis, 1103
- Fibula, fractures, 1073
- Field block, 169-175
in fracture treatment, 985
- "Field-fire" basal cell carcinoma of lip, 267
- Finger(s), amputations, congenital, 1-48
baseball, 1218
cold, hereditary, 1350
constrictions, congenital, 1248
dorsum, subcutaneous abscess, 1187
drop, 998
duplication, 1251
extensor tendon, avulsion, 998
fusion, 1250
hypertrophy, 1252
mallet, 998-1218
metacarpal bones, fractures, 1001
back fractures, 1003
transverse fractures, 1001
middle, absence, 1249
snapping, 1228
- Finger(s) spider, 1252
supernumerary, 1251
transfer in loss of thumb, 1256
webbed, 1251
- Finger tip amputations, 1213
anatomy, 1189
- Finger traps, Japanese, in Colles' fracture, 1011
in dislocation of lunate bone, 1007
in radial shaft fractures, 1022
in transverse fractures of radius and ulna, 1019
- "Finger worms", 1193
- Finney gastroduodenostomy, 639
640
- Finney procedure in peptic ulcer, 642
- Finsterer exclusion operation, 644
in peptic ulcer, 646
- Finsterer gastrojejunostomy, 639
- First aid, 81
in burns, 193
in fractures, 980, 991
in hand injuries, 1204
- Flare, anal. See Anal fissure.
- Fissure fracture, definition, 979
- Fistula(e) anal. See Anal fistula.
arteriovenous, 1358
diagnosis, 1358
symptoms, 1359
treatment, 1359
varicose veins and, 1330
associated with anal and rectal malformations, 591
biliary postoperative, 814
branchiogenic, 292. See also Neck, anomalous lateral
bronchopulmonary, in empyema, 392
carotid cavernous, 1435
cranioaural, repair, 1418
craniofacial, repair, 1418
duodenal. See Duodenum, fistulae.
gastroenterocolic, in marginal ulcer, 617-626
gastrojejunocolic, 654
treatment, 655
intestinal, postoperative, 115
lymph, 1373
pancreatic, 804
electrolyte changes in, 132
hyponatremic acidosis in, 13
postoperative, 814
recto-femoral navicularis, with imperforate anus, 591
rectoperineal, with imperforate anus, 591
rectourethral, with imperforate anus, 591
rectovaginal, with imperforate anus, 591
rectovaginal, with imperforate anus, 591
salivary duct, 285
sigmoidocolic, 705
thyroglossal. See Cysts, thyroglossal.
tracheoesophageal, 314-371
- Fistulotomy versus fistulotomy in anal fistula, 724
- Fistulotomy in anal fistula, 724
versus fistulotomy in anal fistula, 724
- Fitz, Reginald, 692
- "Flail chest", 345
- Flash, hot, in ovarian failure, 949
- Flash burns, 183

- Flatfoot 177
 hypermobile 1274
 spastic peroneal, 1275
 treatment, 1777
 Flexor pollicis longus tendon, repair 1253
 Flexor tendons of hand, rupture 1,20
 Fluorquin in trichomonads, 94
 Fluid, balance a serum, body weight in, 177
 in peritonitis, 491
 burn, 186
 cerebrospinal. See *Cerebrospinal fluid*
 compartments, 126
 boundaries, 16
 constituents, 127
 electrolytes and, management 1,3
 extracellular compartment, peritoneum and, relation, 488
 composition, 177
 sodium concentration disturbances, 133
 volume disturbances, 13
 interstitial, composition, 177
 volume sodium ion concentration and, 177
 loss during operation, replacement, 106
 peritoneal, 486
 requirements in burn shock, estimation, 189
 in surgical patients, 177
 spermatoc, examination in inferi-
 ly 918
 synovial, study of in pyarthrosis, 1132
 therapy in peritonitis, 496
 tissue, biochemical status in frac-
 ture healing, 993
 Fluorohydrocortisone in adreno-
 cor-tical surgery 83
 Fluoroscopy emergency in gastro-
 intestinal hemorrhage, 652
 Floctane, 165
 Focal seizures in head injuries, 1404
 Fold, gastrosplenic, 843
 preplemic, 843
 Folicle, *grafting, cyst, persistent, 96*
 rupture vs. appendicitis, 695
 Folliculitis of hand, 1186
 of vulva, 972, 973
 Foot, 1259-1296
 abscess, 1293
 aponeurosis, 1272
 anatomy functional, 1259
 arches, 1259
 arthritis, 1291
 axis, of balance, 1264
 of leverage, 1264
 bones, accessory 1272
 burns, infection, 1293
 diseases, 1266
 congenital, 1266
 fractures, 1080
 blood, problems, 1284
 immersion, 199
 in gout, 1291-1292
 infections, 1293
 joints, dislocations, 1099
 ligaments, 1261
 midtarsal dislocation, 1099
 motion, 1259
 muscles, 1262
 dorsiflexor paralysis, 1285
 eversion, paralysis, 1286
 intrinsic, paralysis, 1288
 Foot, muscles, inventor paralysis, 187
 plantar flexor paralysis, 1288
 osteoarthritis, 1291-1292
 osteocondritis, 1290
 osteomyelitis, 1794
 paralytic deformities, 1284
 position, 1259
 shelter 199
 subtalar dislocation, 1099
 trench, 199
 tuberculosis, 1145
 tumors, 1793
 vascular disease 1795
 Foramen, of Bochdalek, hernia through, 46
 treatment, 366
 of Winslow 486
 ovale patent pulmonary stenosis with, 437
 persistent, with incompetent valve 441
 without valve 441
 Forceps, hemostatic history 9
 Forearm, fracture first aid, 982
 Forefoot, affections, 1278
 Foreign bodies, definition, 34
 impaction of esophagus, 498
 in air passages, 315
 in breast, 337
 in joints, 1136
 in peritoneal cavity 499
 in pharynx, 235
 in stomach, 6,7
 diagnosis, 627
 symptoms, 627
 treatment, 627
 in thorax, 387
 intraocular magnetic removal, 221
 nonmagnetic, removal, 221
 pharyngeal, 235
 urinary calculus and, 877
 wound healing and, 34
 Formula, Evans, in burn shock, 189
 Fostay intradermal test in tularemia, 1203
 Fothergill's disease. See *Neuralgia trigeminal*
 Fothergill's sign, 481
 Fowler's solution, carcinoma of hand and, 1235
 Fracture(s), active function and, 991
 anesthesia in, 984
 avulsion, of extensor tendon of fingers, 998
 Bier's hyperemia in, 994
 blood, whole, injection in, 994
 bone cortex in, drilling, 995
 by direct violence, 980
 by indirect violence, 980
 by muscular action, 980
 calcium deposition in, 990
 callus formation in, 990-991
 closed, definition, 978
 Colles' 1009
 reverse, 1012
 rupture of extensor pollicis longus in, 1217
 comminuted, definition, 979
 complete, definition, 979
 complicated, definition, 979
 compound. See *Fracture open*
 compression, definition, 979
 of ankle, 1080
 depressed, definition, 979
 dislocations and, 997-1100
 double vertical of Mal-
 gaigne, 1059
 echymosis in, 990
 etiology 979
 fatigue 1087
 first aid in, 980, 991
 Bware definition, 979
 gremetick definition, 979
 healing, pit in, 992, 993
 immobilization, post-reduction, 991
 impacted, definition, 979
 in convalescence endocrine and metabolic features, 152
 incomplete definition, 979
 injury in, treatment, 991
 intertracheal of femur 1067
 local changes following, 989
 march, 1087
 marginal, of distal end of radius, 1013
 Monteggia, 1022
 naval, 227
 needling, 994
 nonunion, 992
 calcium source in, 993
 granulation tissue in, 993
 local pathologic condition in, 992
 prevention, 993
 of acetabulum, 1057
 of alveolar, upper 276
 of ankle, 1076
 of calcaneus, 1082
 of capitellum, 1029
 of cervical spine, 1041
 of clavicle, 1045
 first aid, 987
 of coccyx, 1060
 of condyle of mandible, 275
 of cuboid bone 1085
 of cubiform bones, 1085
 of distal phalanx, 997
 of dorsal spine, 1053
 of elbow 1024
 first aid in, 982
 of femur 1061
 head, 1061
 lower end, 1071
 neck, 1062
 shaft, 1069
 of fibula, 1073
 of foot, 1080
 of forearm, first aid, 982
 of hand, 997
 first aid in, 982
 of humerus. See *Humerus, frac-
 tures*
 of jaw lower first aid, 982
 osteomyelitis and, 281
 upper 276
 of larynx, 310
 of lower extremity 1056
 first aid, 983
 of lumbar vertebrae transverse processes, 1054
 of mandible, 274
 of metacarpal of fingers, 1001
 of metacarpal of thumb, 1000
 of metatarsals of foot, 1087
 of middle phalanx, 997
 of navicular bone. See *Navicular bone fracture*
 of nose, 279
 of odontoid process, 1053
 of occipital process, 1025
 of patella, 1071
 of pelvic rim, 1056

- Fracture(s) of pelvic ring, 1058
 of pelvis, 1056
 first aid, 983
 of phalanges of toes, 1089
 of proximal phalanx, 997
 of radius. See *Radius, fractures*.
 of ribs, 1048
 simple, 385
 of sacrum, 1060
 of scapula, 1048
 first aid in, 982
 of semilunar cartilages, 1089 1090
 of shoulder, 1041
 of shoulder girdle, 1045
 of skull, open, 1407
 of spine, 1051 1055
 first aid in, 982
 of talus, 1080
 of thorax, 1048
 of tibia, 1073
 anterior spine and tubercle, 1073
 of tibial plateau, 1073
 of tibial shaft, 1075
 of trachea, 314
 of ulna. See *Ulna, fractures*.
 of wrist, first aid, 982
 of wrist joint, 1003
 open, 985
 definition, 978
 hemorrhage in, treatment, 985
 immobilization, 987
 osteomyelitis in, 1146, 1127
 reduction, 987
 shock in, treatment, 985
 traction fixation in, 985 986
 wound infection in, prophylaxis
 and treatment, 986
 wound treatment in, 991
 orbital, 219
 pathologic, definition, 979
 pathology, 988-996
 physical medicine and rehabilita-
 tion in, 1316
 physical therapy in, 991
 puncture, definition, 979
 reduction, 991
 repair, 988-996
 shock in, treatment, 991
 Smith, 1012
 soft part injuries in, 991
 spontaneous, definition, 979
 sprain, definition, 979
 subtrochanteric, of femur, 1068
 transportation in, 980
 treatment, considerations, 978-988
 guides for, 991
 trochanteric, 1068
 types, 978
 union, delayed, 992
 calcium source in, 993
 granulation tissue in, 993
 local pathologic condition in,
 992
 prevention, 993
 weight bearing in, 995
 Fracture-dislocations, of distal radial
 epiphysis, 1014
 of lumbodorsal region, 1054
 of shoulder, 1044
 of thumb, Bennett's, 1000
 Fragilis osium, idiopathic, defini-
 tion, 980
 France, surgeons in, history, 3
 surgery in, history, 16
 Freezing injuries. See *Cold injuries*.
 Frei test, in inflammatory rectal stric-
 ture, 725
 Frei test, in lymphogranuloma in-
 gumale, 974
 in lymphogranuloma venereum,
 908
 Freiberg's infraction, 1290
 Fremont dog, experimental ulcer in,
 634
 Frequency urinary in urinary tract
 disease, 856
 Friedlander's organisms in perito-
 nitis, 489
 Friedlander's pneumonia, 406
 Frobenius, 11
 Frohlich's syndrome, 919
 Frontal lobe, abscess, 1431
 tumors, 1424
 Frontotemporal junction, contusions
 and lacerations, 1402
 Frostbite, 199
 Frozen shoulder, 1110
 Fugitive surgeons, 15
 Function, position of, in hand infec-
 tions, 1184
 Fungus infections, in lymphedema,
 1381
 pulmonary, 413
 Fungus, 907
 Funnel chest, 388
 Furuncle, nasal, 227
 of hand, 1186
 Furunculosis of vulva, 972, 973
 Fusion, operative, in Pott's disease,
 1142
 in tuberculosis of elbow, 1146
 in tuberculosis of hip, 1144
 in tuberculosis of knee, 1145
 in tuberculosis of shoulder, 1146
 GALEN 2, § 13, 31 1487
 Gallbladder anatomy, 760
 anomalies, congenital, 763
 calcification in chronic cholecys-
 titis, 773
 carcinoma. See *Carcinoma of gall-
 bladder*
 cholesterosis, in chronic cholecys-
 titis, 772
 disease, choledocholithiasis and,
 778
 hydrops, in chronic cholecystitis,
 773
 infection in cholecystitis, 771
 inflammation. See *Cholecystitis*
 injury, 765
 lymph vessels, 1368
 milk of calcium, chronic cholecys-
 titis and, 773
 physiology, 760
 strawberry, 772
 wounds, treatment, 510
 Gallstones, 765
 biliary tract disease and, relation-
 ship, 766
 carcinoma of bile ducts and, rela-
 tion, 786
 carcinoma of gallbladder and, 784
 choledocholithiasis and, 778
 cholesterol metabolism and, 767
 color, 768
 pain, mechanism, 768
 composition, 767
 diagnosis, 768
 formation, 767
 bile metabolism and, 767
 bile stasis and, 767
 biliary tract infection and, 767
 Gallstones, ileus, 786
 intestinal obstruction in, 757
 small intestine and, 688
 incidence, 765
 age, 765
 pregnancy and, 765
 silent, 774
 Gamma globulin, deficiency effect
 on wound healing, 38
 Ganglion of hand, 1238
 etiology, 1238
 pathology, 1239
 symptoms, 1239
 treatment, 1239
 types, 1239
 Ganglionectomy lumbar, 1465
 thoracic, 1465
 Ganglioneuroma, benign, of adrenal
 medulla, 838
 mediastinal, 378
 Gangrene, gas, 45
 serum, in open fractures, 987
 in cold injury, 201
 pulmonary, 405
 scrotal, 897
 vascular amputations for, 1179
 Gas, gangrene, 45
 serum, in open fractures, 987
 passage, in intestinal obstruction,
 739 742
 Gastrectomy distal, in chronic gas-
 tric ulcer, 635
 partial, anastomosis after, 639
 distal, in peptic ulcer, 638
 physiologic basis, 638
 gastrointestinal continuity res-
 toration following, 639
 height of dumping syndrome
 and, 639
 recurrence and, 639
 in gastric cancer, operative mor-
 tality, 675 676
 in gastroduodenal hemorrhage,
 652
 in palliation of gastric carci-
 noma, 675
 in peptic ulcer, 638
 in resecting pancreatitis, 808
 incision for, 673 674
 stomach ulcer following, 607
 versus total gastrectomy in gas-
 tric carcinoma, relative fre-
 quency, 673
 postoperative complications, 626
 subtotal. See *Gastrectomy per
 total*
 symptoms following, 626
 total, in gastric carcinoma, 671
 operative mortality, 675
 676
 survival rates, 676
 incision for, 673 674
 survival rate in relation to loca-
 tion of tumor, 674 676
 technique, 673
 versus partial gastrectomy in
 gastric carcinoma, relative fre-
 quency, 673
 Gastro. See also *Stomach*.
 Gastric acid, formation, regulation,
 612
 secretion, inhibition, 612
 stimulation, 610
 Gastric analysis in peptic ulcer diag-
 nosis, 617
 Gastric carcinoma. See *Carcinoma,
 gastric*.

- Gastric juice in peritoneal cavity 499
- Gastric phase of digestion, 612
- of gastric acid secretion, 610
- Gastric retention, peptic ulcer and 647
- Gastric ulcer. See Stomach, ulcer
- Gastrin, 610, 614 632
- Gastritis, 627
- acute 628
- antral, 628
- chronic 628
- relation to gastric carcinoma 669
- classification, 628
- corrosive, 628
- exogenous, simple 628
- following gastrectomy 626
- "hypertrophic," 628
- infectious, acute 628
- phlegmonous, 628
- postoperative, 629
- "superficial," 628
- Gastrocolic ligament, 486
- Gastrointestinal hemorrhage. See Hemorrhage gastrointestinal
- Gastrointestinal motor activity 612
- Gastrointestinal ulcer incidence, 606
- Gastrooduodenotomy after antrectomy in chronic gastric ulcer 653
- after partial gastrectomy 640
- Gastroenteritis, acute vs. appendicitis, 694
- vs. appendicitis, 693
- vs. small intestine infections, 687
- Gastroenterocolic fistula in marginal ulcer 617 626
- Gastroenterostomy in duodenal ulcer results, 625
- in palliation of gastric carcinoma, 675
- stomal ulcer following, incidence 607
- Gastrohepatic ligament, 486
- Gastrointestinal contents, aspiration, postoperative atelectasis and, 116
- Gastrointestinal continuity restoration following gastric resection, 639
- Gastrointestinal decompression in peritonitis, 496
- Gastrointestinal hemorrhage in portal hypertension, 792
- Gastrointestinal secretions, sodium, potassium and chloride losses in, 128, 129
- Gastrointestinal tract, decompression in diaphragmatic hernia, 567
- healing, 40
- in burns, pathologic changes in, 186
- perforation, in pneumoperitoneum, 510
- preoperative preparation, 105
- Gastrojejunal ulcer incidence, 660
- recurrent, 653
- hyperfunctioning islet alpha cell tumor of pancreas and, 654
- treatment, 654
- vagotomy in, 641
- Gastrojejunocolic fistula, 654
- treatment, 655
- Gastrojejunostomy after partial gastrectomy 639
- Gastrojejunostomy in gastric cancer 671
- in obstructive jaundice 813
- in peptic ulcer 625
- with vagotomy in peptic ulcer 642
- Gastrosocopy in gastric ulcer 618, 671
- Gastrosplenic fold, 843
- Gastrostomy 648
- in esophageal atresia repair 573
- Gaucher's disease splenomegaly in, 852
- Generator infrared, 1306
- radiant heat 1306
- Genitalia, external, examination in urinary tract disease 857
- female bleeding, 933
- cancer 929
- discharge from, 922
- male, external, 890
- internal, 890 908
- physiology 908
- Genitourinary pain, referral of 857
- Genitourinary surgery antibiotic prophylaxis in, 60
- Gentian violet in thrush, 928
- Germany surgery in, history 18
- Gibson, 700
- Giddiness in head injuries, 1400
- Gland(s), autolytic, physiology 63
- Bartholin's, abscess, 927
- cyst, 927
- Brunner's, physiology 632
- cardiac, physiology 632
- cervical, tuberculous, 1384
- Cowper's, 916
- lymph. See Lymph nodes
- parathyroid, 317 325
- in metabolism, 323
- removal, 325
- parotid, inflammation, postoperative, 120
- tumors, benign, removal, 287
- malignant, 288, 289
- pyloric, physiology 632
- renal, weight, 820
- salivary. See Salivary glands
- Siemens, infection, 927
- Stahr's, 1371
- Glass penis, 891
- inflammation, 894
- Glioblastoma, 1447
- Gliomas, 1420 1423 1425
- of cord, 144
- of optic chiasm, 1425
- Globe enucleation, 223
- wounds, 221
- Globulin, anaphenon, test in acquired hemolytic anemia, 850
- gamma, deficiency of effect on wound healing, 38
- Glomus, myoeurovascular, 1104
- tumor of hand, 1243
- of peripheral nerves, 1459
- Glossopharyngeal nerve, disorders, 1449
- Glossopharyngeal neuralgia, 1449
- Gloves, rubber history 7
- Glucagon 802
- Glucocorticoids, function, 124
- Glucose, in preoperative management of malnourished patient, 100
- metabolism, post-traumatic, 142
- Gluteal hernia, superior 568
- Groer endemic, 318
- multimodular 318
- Gout nodular 318
- with hyperthyroidism, 318
- without hyperthyroidism, 321
- production 317
- Gold, radioactive in adenocarcinoma of ovary 970
- Goldman, 12
- Goad(s), function, in catabolism, 141
- Gonadotropin(s) chorionic, in cryptorchidism, 899
- in male infertility 918
- Gonkometer in measurement of joint motion, 1300
- Gonorrhea, 926
- diagnosis, 977
- in female, 974
- acute, 956
- symptoms, 956
- treatment 957
- chronic 957
- postabortion recurrence, 958
- postperal recurrence 958
- treatment, 927
- symptoms, 926
- Gout, bursa in, 1109
- foot in, 1291 1292
- vs. pyarthrosis, 1133
- Graafian follicle cyst, persistent, 962
- rupture vs. appendicitis, 695
- Gradenigo's syndrome 1432
- Graft, bone. See Bone grafts
- skin. See Skin grafts
- Grand mal seizures in head injuries, 1404
- Granulation tissue, abnormal, 26
- growth in fracture healing, 993
- healthy 26, 26
- in fractures, 992
- in fracture healing, 993
- in wound healing, 25
- normal, 26
- unhealthy 26, 26
- Granulocyte count in radiation injuries, 211
- Granulocytopenia due to primary hypoparathyroidism, 848
- Granuloma, coccidioidal, pulmonary 413
- eosinophilic, 16, 1166
- foreign body of breast, 337
- hair of barbers, 1204
- Hodgkin's, 1168
- leguminal 908
- in female 974
- of mouth, 256
- lipoid, pulmonary 413
- peritoneal, 499
- pyogenic, of hand, 1243
- telangiectatic, of hand, 1243
- Granulosa cell tumor 967
- Graves' disease, 318
- in children, radiation treatment and, 319
- recurrence following thyroidectomy 319
- thyroidectomy in, 319
- Great Britain, surgery in, history 16
- Greece, early wound care by
- Greenblatt intradural skin test in chancroid 974
- Greenstick fracture, definition, 979
- Groin hernia, 524
- Growth disturbances in acute hemangioma osteomyelitis, 1120
- Grynfelt, superior lumbar triangle of, anatomy 553

- Fracture(s) of pelvic ring, 1058
 of pelvis, 1056
 first aid, 983
 of phalanges of toes, 1089
 of proximal phalanx, 997
 of radius. See *Radius, fractures*.
 of ribs, 1048
 simple, 385
 of sacrum, 1060
 of scapula, 1048
 first aid in, 982
 of semilunar cartilages, 1089-1090
 of shoulder, 1041
 of shoulder girdle, 1045
 of skull, open, 1407
 of spine, 1051-1055
 first aid in, 982
 of tibia, 1080
 of thorax, 1048
 of tibia, 1073
 anterior spine and tubercle, 1073
 of tibial plateau, 1073
 of tibial shaft, 1075
 of trachea, 314
 of ulna. See *Ulna, fractures*.
 of wrist, first aid, 982
 of wrist joint, 1003
 open, 985
 definition, 978
 hemorrhage in, treatment, 985
 immobilization, 987
 osteomyelitis in, 1126, 1127
 reduction, 987
 shock in, treatment, 985
 traction fixation in, 985-986
 wound infection in, prophylaxis
 and treatment, 986
 wound treatment in, 991
 orbital, 219
 pathologic, definition, 979
 pathology, 988-996
 physical medicine and rehabilita-
 tion in, 1316
 physical therapy in, 991
 puncture, definition, 979
 reduction, 991
 repair, 988-996
 shock in, treatment, 991
 Smith, 1012
 soft part injuries in, 991
 spontaneous, definition, 979
 sprain, definition, 979
 subtrochanteric, of femur, 1068
 transportation in, 980
 treatment, considerations, 978-988
 guides for, 991
 trochanteric, 1068
 types, 978
 union, delayed, 992
 calcium source in, 993
 granulation tissue in, 993
 local pathologic condition in,
 992
 prevention, 993
 weight bearing in, 995
 Fracture-dislocations, of distal radial
 epiphysis, 1014
 of lumbosacral region, 1054
 of shoulder, 1044
 of thumb, Bennett's, 1000
 Fragilis osseum, idiopathic, defini-
 tion, 980
 France, surgeons in, history, 3
 surgery in, history, 16
 Freezing injuries. See *Cold injuries*.
 Fret test, in inflammatory rectal stin-
 ture, 725
 Fret test, in lymphogranuloma in-
 guinale, 974
 in lymphogranuloma venereum,
 908
 Freiberg's infraction, 1790
 Fremont dog, experimental ulcer in,
 634
 Frequency urinary in urinary tract
 disease, 856
 Friedlander's organisms in perito-
 nitis, 489
 Friedlander's pneumonia, 406
 Frobenius, 11
 Frohlich's syndrome, 919
 Frontal lobe, abscess, 1431
 tumors, 14-4
 Frontotemporal junction, contusions
 and lacerations, 1407
 Frontitis, 199
 Frozen shoulder, 1110
 Fugitive surgeons, 15
 Function, position of in hand infec-
 tions, 1184
 Fungus infections, in lymphedema,
 1381
 pulmonary, 413
 Funiculus, 907
 Funnel chest, 388
 Furuncle, nasal, 227
 of hand, 1186
 Furunculosis of vulva, 972, 973
 Fusion, operative, in Pott's disease,
 1147
 in tuberculosis of elbow, 1146
 in tuberculosis of hip, 1144
 in tuberculosis of knee, 1145
 in tuberculosis of shoulder, 1146
 GALEN 2, § 13-31, 1487
 Gallbladder, anatomy, 760
 anomalies, congenital, 763
 calcification in chronic cholecys-
 titis, 773
 carcinoma. See *Carcinoma of gall-
 bladder*
 cholesterosis, in chronic cholecys-
 titis, 772
 disease, choledocholithiasis and,
 778
 hydrops, in chronic cholecystitis,
 773
 infection in cholecystitis, 771
 inflammation. See *Cholecystitis*.
 injury, 765
 lymph vessels, 1368
 milk of calcium, chronic cholecys-
 titis and, 773
 physiology, 760
 strawberry, 772
 wounds, treatment, 510
 Gallstones, 765
 biliary tract disease and, relation-
 ship, 766
 carcinoma of bile ducts and, rela-
 tion, 786
 carcinoma of gallbladder and, 784
 choledocholithiasis and, 778
 cholesterol metabolism and, 767
 color, 768
 pain, mechanism, 768
 composition, 767
 diagnosis, 768
 formation, 767
 bile metabolism and, 767
 bile acids and, 767
 biliary tract infection and, 767
 Gallstones, ileus, 786
 intestinal obstruction in, 757
 small intestine and, 683
 incidence, 765
 age, 765
 pregnancy and, 765
 silent, 774
 Gamma globulin, deficiency effect
 on wound healing, 38
 Ganglion of hand, 1238
 etiology, 1238
 pathology, 1239
 symptoms, 1239
 treatment, 1239
 types, 1239
 Ganglionectomy lumbar, 1465
 thoracic, 1465
 Ganglioneuroma, benign, of adrenal
 medulla, 838
 medistinal, 378
 Gangrene, gas, 45
 serum, in open fractures, 987
 in cold injury, 201
 pulmonary, 405
 scrotal, 897
 vascular amputations for, 1179
 Gas, gangrene, 45
 serum, in open fractures, 987
 passage, in intestinal obstruction,
 739-742
 Gastroectomy distal, in chronic gas-
 tric ulcer, 655
 partial, anastomosis after, 639
 distal, in peptic ulcer, 638
 physiologic basis, 638
 gastrointestinal continuity res-
 toration following, 639
 height of, dumping syndrome
 and, 639
 recurrence and, 639
 in gastric cancer, operative mor-
 tality, 675-676
 in gastroduodenal hemorrhage,
 652
 in palliation of gastric carci-
 noma, 675
 in peptic ulcer, 638
 in relapsing pancreatitis, 808
 incision for, 673-674
 stomal ulcer following, 607
 versus total gastrectomy in gas-
 tric carcinoma, relative fre-
 quency, 673
 postoperative complications, 646
 subtotal. See *Gastrectomy par-
 tial*
 symptoms following, 626
 total, in gastric carcinoma, 671
 operative mortality, 675
 676
 survival rates, 676
 incision for, 673-674
 survival rate in relation to loca-
 tion of tumor, 674-676
 technique, 673
 versus partial gastrectomy in
 gastric carcinoma, relative fre-
 quency, 673
 Gastric. See also *Stomach*
 Gastric acid, formation, regulation,
 612
 secretion, inhibition, 612
 stimulation, 610
 Gastric analysis in peptic ulcer diag-
 nosis, 617
 Gastric carcinoma. See *Carcinoma,
 gastric*.

- Heart-lung apparatus, extracorporeal, 477
- Heat, applicators, external, 1305
- exchangers in hypothermia, 430
- in amputations, 13
- in infections, 50
- in lower motor neuron disorders, 1320
- in orthopedic disorders, 1317
- in thoracic surgical conditions, 134
- in upper motor neuron disorders, 1318
- loss, tissue in cold injury 198
- radiant, generators, 1306
- therapy 1305
- Hedrocele 569
- Heidenhain pouch, hydrochloric acid secretion and, 633
- Hemels-Mikulicz pyloroplasty 642, 643
- Heliobacter, 8
- Hemangioblastoma, 144
- Hemangiopericytoma, cardiac, 466
- Hemangioma(s), arterial, of lips and mouth, 64
- gastric, 664
- growing, of lips and mouth, 64
- of abdominal wall, 482
- of hand, 1246
- of joint, 1177
- of lips and mouth, sclerosing, 263
- of liver 796
- of muscle, 1104
- of salivary gland, 288
- of small intestine, 690
- of spleen, 850
- Hemangiomatosis with dyschondroplasia, 1157
- Hematemesis, in gastric ulcer 619
- in peptic ulcer 616
- Hematocle 905 906
- Hematologic changes in burns, 187
- Hematomas, epidural, treatment, 1418
- extradural, in head injuries, 1402
- following breast trauma, 336
- intracerebral, 1412
- treatment, 1418
- of nose, 226
- of rectus sheath, 481
- of scalp 213 1406
- subperiosteal, 214
- of scrotum, 905
- pulmonary in aneurysm, 1355
- subdural, 1410
- acute, 1410
- chronic, 1411
- in head injuries, 1402
- pathophysiology 1411
- subacute, 1410
- treatment, 1418
- subperiosteal, 214
- wound, 112
- Hematopoiesis, extramedullary 844
- Hematuria, in bladder tumors, 879
- in renal carcinoma, 880
- in urinary calculus disease, 874
- in urinary tract disease, 856
- Hemiparesis, facial, 1449
- Hemispherectomy cerebral, in focal epilepsy 1438
- Hemiparesis, cerebral, tumors, 1423
- Hemoconcentration in burns, 187
- Hemoptysis, 510
- spontaneous, 510
- traumatic, 510
- types, 510
- Hemophilia vs. primary thrombocytopenic purpura 847
- with hemorrhage into joint, vs. pyarthrosis, 1133
- Hemoptysis in bronchogenic carcinoma, 415
- Hemorrhage, control, in wounds of large arteries, 1348
- delayed, in nonpenetrating intra-abdominal wounds, 509
- epidural, 1408
- excessive in wound healing, 24
- from bowel, in Meckel's diverticulum, 584
- gastrointestinal, 610
- general considerations, 650
- sources, 651
- surgical intervention, anesthesia in, 652
- indications, 651
- treatment, 651
- gastrointestinal, in acute gastric erosions 635
- in portal hypertension, 792
- in abortions, 940
- in cancer of cervix 933 935
- in cancer of corpus uteri, 938
- in diverticulitis, 705
- in diverticulosis, 705
- in ectopic pregnancy 944 945
- in fibroids, 94
- in hand injuries, control, 1704
- in head injuries, 1393
- in open fractures, treatment, 985
- in peptic ulcer 616, 619
- differential diagnosis, 620
- incidence 607
- medical treatment, 623
- surgical treatment, 624
- in reproductive system diseases in female, 933
- intracranial, traumatic, 1408
- vs. hypertrophic pyloric stenosis in vomiting in infants, 576
- meningeal, middle 1408
- nasal, 226
- retroperitoneal, 516
- sudden, shock prevention follow-up, 97
- treatment, history 7
- wound healing and, 37
- Hemorrhagic state in radiation injuries, 211
- Hemorrhoidectomy 719 721
- Hemorrhoids, 719
- diagnosis, 720
- etiology 719
- external, 719
- internal, 719
- strangulated, 720
- surgical treatment, indications, 721
- objectives, 721
- symptoms, 719
- treatment, 720
- Hemostasis, history 7
- Hemostasis, history 9
- Hemothorax, 386
- in rib fracture, 1050
- peel formation in, 386
- Henderson-Hasselbalch equation, 130
- Henri, 13 15
- Heparinization in extracorporeal by-pass, 428
- Hepatectomy partial, radical, in liver carcinoma, 796
- Hepatic vein thrombosis, in portal hypertension, 791
- Hepatic vein thrombosis, vs. intra-hepatic block 791
- Hepatitis, aseptic, 798
- following amebiasis, 798
- vs. pancreatic carcinoma, 811
- Hepatorenal pouch of Morison, 502
- Hereditary as factor in hernia 570
- Hermaphroditism, true 919
- Hernia 518 571
- abdominal definition, 519
- acquired, definition, 519
- adiposa definition, 520
- vs. indirect inguinal hernia, 530
- characteristics, objective, 523
- classification, 519
- complete definition, 520
- compression reduction, 523
- congenital, definition, 519
- etiology 520
- definition, 518
- diagnosis, 522
- diaphragmatic, 561
- anatomic factors, 562
- congenital, treatment, 466
- definition, 561
- diagnosis, 564
- due to suppurative necrosis, 564
- etiology 561
- incidence 561
- sliding, 541
- symptoms, 565
- traumatic, 564
- treatment, 566
- types, 561
- double definition, 519
- epigastric, 540
- anatomic factors, 550
- definition, 550
- diagnosis, 552
- etiology 550
- incidence, 550
- surgical repair 55
- esophageal, hiatus, 563
- etiology 561
- symptoms, 565
- treatment, 566
- etiology 520
- external, definition, 519
- femoral, 537
- anatomic factors, 537
- definition, 537
- diagnosis, 539
- etiology 537
- incidence, 537
- sliding, 541
- strangulated, intestinal obstruction in, 756
- surgical repair 540
- treatment, 540
- vs. indirect inguinal hernia, 530
- gangrenous, definition, 519
- gluteal, superior 568
- groin, 524
- sliding, 541
- hiatus, esophageal. See *Hernia, esophageal hiatus*.
- incarcerated, definition, 519
- strangulated hernia and, differentiation, 732
- incidence 521
- ischial, 554
- anatomic factors, 554
- definition, 519 554
- diagnosis, 559
- etiology 554
- incidence, 554
- prevention, 559

- Gulotine amputation, technique 1177
- Gulotine method of tonsillectomy and adenoidectomy 236
- Gumbell, 282
- Gynecologic and obstetric surgery
antibiotic prophylaxis, 60
- Gynecologic clinical approach to, 922
- Individualization of patient in, 921
- Gynecomastia, 339
- Hair granuloma of barbers, 1204
- Hairball, 627
- Hallux paronychia, 1281
- Hallux rigidus, 1281
- Hallux valgus, 1278
etiology, 1279
treatment, 1280
- Habited, William, 6, 9, 12, 19, 1381
- Habited I inguinal hernioplasty 544
- Habited II inguinal hernioplasty 544
- Hamman, of liver 795
- of lung, 423
- Hamilton rabbit test in diffuse osteitis fibrosa, 1161
- Hamman's sign in mediastinal emphysema, 370
- Hammner toe, 1281
- Hand, 1182-1258
abrasions, 1213
amputations, congenital, 1248
aneurysms, congenital arteriove-
nous, 1242
treatment, 1243
ape, 1454
aplasia, 1248, 1250
artificial, versus split hook, 1180
blood vessels, tumors, 1241
bone cysts, 1246
bone sarcomas, 1, 46
bone tumors, 1244
burns, 196, 1220
abrasive, 1213
capillary angioma, 1242
treatment, 1243
carcinoma, 1232
cavernous angioma, 1242
treatment, 1243
congenital deformities, 1247
classification, 1247
etiologic factors, 1247
contractions, congenital, 1248
crushing injuries, extensive, 1213
dislocation, buttonhole, 1219
enchondroma, 1244
erosions, 1, 45
cartilaginous, multiple, 1252
Ewing's tumors, 1246
feels, anatomy, 1224
fibroma, 1236, 1, 46
fibroma thymoma, tumors, 1236
flexor tendons, rupture, 1220
fractures, 997
first aid, 982
fusion of elements of, 1250
ganglion. See *Ganglion of hand*
glomus tumor, 1243
grease-gun injuries, 1220
hemangiomas, 1246
hypoplasia, 1248, 1249
immersion, 199
infections, 1182
acute, spreading, 1196
care, general principles, 1183
chronic, 1199
- Hand, infections, drainage in, 1183
incision for, 1185
types, 1186
injuries, 1204
bleeding in, control, 1204
crushing, 1213
extent, diagnosis, 1207
history, 1205
open, 1204
first aid, 1204
sensory findings, 1207
surgical repair, 1208
types, 1204
lipoma, 1237
lobster-claw, 1249
lymphangiomas, 1244
lymphatic drainage, 1197
melanoma, 1, 36
mucous cyst, 1240
myxomas, 1246
nerves, tumors, 1246
neurofibrosarcoma, 1247
neuroma, plexiform, 1247
neurinosarcoma, 1247
osteomas, 1246
osteoid, 1245
osteomyelitis, 1190
pyogenic granuloma, 1, 43
Rockingham's disease in, 1, 46
sarcomas, 1236
surgery reconstructive, 1257
synovial, 1241
telangiectatic granuloma, 1243
tendons, injuries, 1214
tumors, 1230
arising from joint capsules, 1, 38
arising from skin, 1230
arising from tendon sheaths, 1238
benign, incidence, 1230
bone, 1244
classification, 1230
giant cell, 1246
sarcomatous, 1240
malignant, incidence, 1230
metastatic, 1, 47
of fibrous tissues, 1236
of nerves, 1246
types, incidence, 1230
xanthomas, 1240
- Hand-Schuller-Christian disease, 216, 1166
- splenomegaly in, 85
- Harvey, 15
- Hashimoto's thyroiditis, 320
- Hasselbalch-Henderson equation, 130
- Haversian system, tumors, 1163
- Head, 213-290
dressing, crushing, 73
injuries, 1392. See also *Cranio-cerebral injuries*
convulsions in, 1404
high velocity, 1408
incidence, 1392
mechanism, 1392
penetrating, 1408
lymphatic supply, 1371
surgery antibiotic prophylaxis, 59
- Headache, in head injuries, 1399
- in intracranial tumors, 1421
- post-traumatic, 1399
- Healing, by first intention, 31
versus suppurative wound
healing, history
by second intention, 32
by third intention, 32
wound. See *Wound healing*
- Hearing, loss in Meniere's disease, 249
tests, 47
in Meniere's disease, 249
threshold, measurement, 247
- Heart, 426-470
arrest, 180, 467
anticipated, management, 468
by cold, 429
cardiac massage in, 468
diagnosis, 467
factors precipitating, 467
in aortic arch aneurysm repair, 464
in cardiopulmonary by-pass, 429
management, 468
prevention, 108
resuscitation tray, 469
treatment, 108
technique, 180
beat, cessation. See *Heart arrest*
restoration. See *Heart arrest*
block, complete, in ventricular septal defect closure, 445
catheterization, in aortic stenosis, 456
in atrial septal defects, 443
in mitral insufficiency, 453
in mitral stenosis, 450
in patent ductus arteriosus, 431
in pulmonary stenosis, 438
in tetralogy of Fallot, 440
in transposition of great vessels, 446
in ventricular septal defects, 445
compression, chronic, 448
disease, acquired, classification, 426
congenital, classification, 426
management, preoperative, 102
overhydration in, preoperative prevention, 103
shock prevention in, preoperative measures, 102
vs. chronic cholecystitis, 773
calibration, in aortic insufficiency, 457
in atrial septal defects, 443
great vessels, transposition, 446
lymphatic vessels, 1371
massage in cardiac arrest, 109
murmur(s), in aortic insufficiency, 457
in aortic septal defects, 433
in aortic stenosis, 433
in coarctation of aorta, 434
in patent ductus arteriosus, 431
operations, complications, 457
extracorporeal circulation in, 426
hypothermia in, 429
physiology, 426
rate in head injury, 1397
resuscitation, technique, 180
tray, 469
size, in hypertension, changes fol-
lowing adrenalectomy, 835
tamponade in pericardial effusion, 447
tumors, 466
malignant, 466
metastatic, 466
primary, 466
surgical treatment, 467
vessel, great, transposition, 446
x-ray in tetralogy of Fallot, 440

- Hydrocephalus, types, 1483
 Hydrochloric acid secretion, 608
 Hydrocortisone in adrenocortical surgery 83
 in catatonia, 140
 in peritonitis, 496
 in suspected postoperative adrenal failure 154
 Hydrocortisone in barbit, 1110
 Hydrogen ion concentration. See pH
 Hydrolysis, membrane theory of hydrochloric acid secretion, 608
 Hydromephroses, 863
 treatment, 866
 Hydrops, endolymphatic. See *Meniere's disease*
 of gallbladder in chronic cholecystitis, 773
 Hydrotherapy 1305
 Hydrothorax in fibroma of ovary 968
 17-Hydroxycorticoid excretion in hyperadrenocorticism, 879
 Hygroma coli, cystic 796
 clinical course, 797
 diagnosis, 797
 etiology 796
 pathology 297
 treatment 797
 cystic, 1385
 of neck, 266 288
 Hyperadrenocorticism, adrenalectomy in, 833
 cause, differential diagnosis, 828
 clinical patterns, 822
 17 hydroxycorticoid excretion in, 879
 in sexual precocity 919
 17-ketosteroid excretion in, 829
 management, 832
 Hyperbaric solutions, 173
 Hypercalcemia in hyperparathyroidism, 324
 Hypercalcemia, in hyperparathyroidism, 374
 urinary calculus and, 877
 Hypercapnia, prevention during operation, 107
 Hypercarbia, prevention during operation, 107
 Hyperemia, Bier's, in fractures, 994
 reactive, in testing capillary circulation, 1345
 Hyperemesis in ovarian failure, 947
 Hyperhidrosis of nervous origin, sympathectomy in, 1467
 Hypermullinism, blast cell tumors and, 816
 Hypernatremia in acute renal injury 134
 Hypernephroma, 880
 Hyperostosis, of skull, 215
 infantile, cortical, vs. acute hemogenous osteomyelitis, 1117
 Hyperparathyroidism, 323
 primary 323
 secondary 323
 symptoms and signs, 324
 types, 323
 urinary calculi and, 872
 Hyperphalangism in hand, 1251
 Hyperplasia, papillary cystophorosis of breast, 344
 Hyperplasia-involution cycle of breast, 329 332, 333
 Hyperpnea in head injury 1397
 Hypersplenism, 846
 diagnosis, 846
 primary 846
 secondary 846 849
 Hypertension, adrenalectomy in, 834
 changes in heart size following, 835
 adrenocortical relations to, 833
 essential, 1466
 in pheochromocytoma, 838
 portal, 789
 clinical picture 791
 definition, 789
 etiology 789 790
 operation in, patient selection, 794
 pathogenesis, 790
 prognosis, 795
 treatment, 792
 Hyperthyroidism, diagnosis, 318
 exophthalmos of cause 318
 following thyroidectomy 319
 treatment, 319
 types, 318
 Hypervitaminosis A vs. acute hemogenous osteomyelitis, 1117
 Hypoadrenocorticism, postoperative 154
 Hypobaric solutions, 173
 Hypodermic, history 12
 Hypogastrium, 919
 Hypoglossal nerve, disorders, 1450
 Hypoglycemia, following gastrectomy 6.6
 in islets of Langerhans tumors, 816
 spontaneous, diagnosis, 816
 Hypoparadism, male 919
 Hypokalemia, in convalescence 148
 in primary aldosteronism, 827
 Hyponatremia in convalescence, 149
 Hypopharynx, anatomy 232
 Hypophysectomy in breast carcinoma, 364
 Hypophysis cerebri, adenomas, 1420
 anatomy 32
 Hypoplasia of hand, I 48, 1249
 Hypoproteinemia, preoperative management, 101 102
 wound healing and, 38
 Hypoproteinaemia, management, preoperative 104
 vitamin K and, 39
 Hypoparadism, 892
 balance, 892
 glandular 892
 pemic, 892
 perineal, 892, 919
 Hypostension, intentional, during operation, 176
 Hypothalamic-pituitary-adrenal phase of gastric acid secretion, 611
 Hypothalamus, relay of stress to stomach from, 611
 Hypothermia, 1306
 extracorporeal circulation and, 430
 heat exchangers in, 430
 in aortic arch aneurysms, 464
 in cardiac surgery 427
 in cardiovascular surgery 429
 in cold injury 198
 in intracranial operations, 1429
 intentional, during operation, 176
 regulation, 430
 Hypotonicity serum, in convalescence 149
 Hypoventilation, postoperative atelectasis and, 115
 Hypovitaminosis, management preoperative, 103
 Hypovolemia, preoperative management, 101
 role in shock, 88
 Hypoxia, hemoglobin, in gastroduodenal hemorrhage 651
 in impending shock, damage in, prevention, 93
 prevention during operation, 107
 Hysterectomy 933
 abdominal, total, 953
 effects, 953
 in adenocarcinoma of ovary 970
 in cystadenoma of ovary 965 966
 in dermoid cyst of ovary 969
 in endocrine bleeding, 951
 in endometriosis, 964
 in fibroids, 944
 in granulosa cell tumor 968
 in pelvic tuberculosis, 959
 in thecal cell tumor 968
 radical, in carcinoma of corpus uteri, 939
 total, abdominal, 953
 in chronic endocervicitis, 926
 in preinvasive cervical cancer 937
 vaginal, 953
 Icterus, spherocytic, 846, 848
 Ileitis, regional. See *Enteritis regionalis*
 terminal. See *Enteritis regionalis*
 Ileocolostomy side-to-side, in carcinoma of colon, 713
 Ileostomy complications, 704
 double-barreled, and Mikulicz resection in meconium ileus, 588
 dysfunction, 704
 in chronic ulcerative colitis, 703
 Ileum, 685-692. See also *Intestine small*
 atresia and stenosis, 577
 duplex. See *Alimentary tract duplications*
 lymph vessels, 1368
 Ileus, 732, 752
 adynamic. See *Ileus paralytic*
 causes, classification, 752
 definition, 731
 dynamic, 731 754
 gastroic, 786
 intestinal obstruction in, 757
 small intestine and, 688
 inhibition. See *Ileus paralytic*
 meconium, 588
 paralytic, 731 75
 diagnosis, 753
 in bacterial peritonitis, 753
 peritonitis and, 490
 prevention, 753
 treatment, 754
 vs. intestinal obstruction, 745
 postoperative, 118
 prolonged, peritonitis and, 491
 spastic, 731 754
 Immersion foot, 199
 Immersion band, 199
 Immersion leg, 199
 Immobilization, of fractures, post-reduction, 991
 of open fractures, 987

- Hernia, incisional, symptoms, 559**
 treatment, 559
- Incomplete, definition, 570**
- Inguinal, direct, 534**
 anatomic factors in, 535
 definition, 534
 diagnosis, 536
 etiology, 534
 incidence, 535
 treatment, 536
 vs. femoral hernia, 539
 vs. indirect inguinal hernia, 530
- Indirect, 524**
 anatomic factors in, 525
 definition, 524
 diagnosis, 528
 differential diagnosis, 530
 etiology, 524
 incidence, 524
 large, 526
 treatment, 532
 medium, 526
 treatment, 532
 alkling, 541
 small, 526
 treatment, 530
 surgical principles of repair, 53
 treatment, 530
 types, 526
 vs. femoral hernia, 539
- Internal, definition, 519**
- Intestinal, definition, 520**
- Irreducible, definition, 519**
- Lavater's, definition, 520**
- Littre's, definition, 570**
 in femoral hernia, 540
- Lumbar, 553**
 anatomic factors, 553
- Obturator, 568**
 types, 553
- Of inguino-femoral region, 524**
 of linea alba, 545
 of linea semilunaris, 545
 of lung, 549
 of Morgagni, 562
 treatment, 566
 of pelvic diaphragm, 567
 of pelvis, 567
- Pantaloon, definition, 519**
- Parasternal, 562**
 treatment, 566
- Perineal, 568**
- Pleuropertoneal, 562**
- Potential, 568**
- Recurrent, definition, 519**
- Reducible, definition, 519**
- Reduction, compression, 523**
- Richter's, 568**
 definition, 520
 strangulated, intestinal obstruction in, 756
- Scrotic, 568**
- Semilunar, 55**
 anatomic factors, 552
 definition, 520
- Striking, 541**
 definition, 520
 mechanism, 541
- Spiegel's, 552**
 anatomic factors, 552
 definition, 520
 strangulated, definition, 519
 incarcerated hernia and, differential diagnosis, 732
 intestinal obstruction in, 756
- Hernia, superior gluteal, 568**
 suprapubic, 568
 surgical repair, 523
 symptoms, 522
 through foramen of Bochdalek, 562
 treatment, 566
- Topographic, definition, 519**
 treatment, 523
- Injection, 523**
- Triple, definition, 519**
- Truss treatment, 523**
- Umbilical, 479-545**
 adult, 545
 anatomic factors in, 548
 surgical repair, 550
 symptoms, 548
 anatomic factors in, 547
 definition, 545
 diagnosis, 548
 etiology, 545
 fetal, 545
 anatomic factors in, 547
 surgical treatment, 549
 symptoms, 548
 infantile, 545
 anatomic factors in, 547
 symptoms, 548
 treatment, 549
 vaginal, posterior. See *Enterocela*
- Ventral, treatment, 952**
- Uterine prolapse and, anatomic similarity, 952**
- Hernioplasty(ies), consecutive, incidence of common hernias in, 521-522**
 femoral, 540
 inguinal, methods of, comparison, 542
 surgical principles, 532
- Herniorrhaphy, surgical principles, 532**
- Herophilus, 2**
- Herpes progenitalis, 894**
- Herpes vulvae, 973**
- Hesitancy urinary in urinary tract disease, 856**
- Hesselbach's triangle, 535**
- Hexobarbital, 168**
- Hidradenitis of vulva, 972, 973**
- Hilar, pulmonary enlargement, in bronchogenic carcinoma, 416**
- Hind foot problems, 1284**
- Hindgut, primitive, 472**
- Hindu surgeons, 2**
- Hip joint, dislocations, 1094**
 sprained, 1108
 tuberculous. See *Tuberculosis of hip*
- Hippocrates, 2, 13**
- Hirschsprung's disease, 589**
- Histamines in Meniere's disease, 450**
- Histiocytosis, nonlipoid, 1166**
- Histioplasmosis, pulmonary, 413**
- Hodgkin's disease, 1386**
 differential diagnosis, 1387
 of retroperitoneal space, 516
 of stomach, 666
 supervoltage ray therapy in, 1388
 treatment, 1388
 vs. lymphosarcoma, 1385
- Hodgkin's granuloma, 1168**
- Hofmeister gastric resection, in gastric carcinoma, 672, 673**
 in peptic ulcer, 645
- Hofmeister gastrojejunostomy, 639-640**
- Hofmeister operation, in chronic gastric ulcer, 656**
 in gastric cancer, 671
- Hopner maneuver in hernia repair, 537**
- Hollander insulin test in recurrent ulcer, 654**
- Holmes, Oliver Wendell, 11**
- Homans sign in venous thrombosis, 1337**
- Homografts in burns, 195**
- Hooks, split, versus artificial hand, 1180**
- Hormonal pathway in relay of stress from hypothalamus to stomach, 611**
- Hormonal phase of gastric acid secretion, 610**
- Hormone(s), adrenal medulla, 140**
 adrenocortical, 139
 in adrenocortical surgery, 837
 postoperative increase in, 144
 anikretic excretion, postoperative, 126
 in breast carcinoma, 363
 in prostatic carcinoma, 914
 sex, in peptic ulcer, 614
 thyroid, 317
 thyroid-stimulating, 317
 increase in, 317
- Horn, cutaneous, of hand, 1231**
- Hospitalitis, 1324**
- Hospital, infections acquired in, prevention and control, 58**
- Host-drug relationships, 55**
- Hot flash in ovarian failure, 949**
- Hosehead's knee, 1109**
- Hugo de Lucos, 3, 10, 13, 15**
- Human bites, care of, 66**
- Humerus, fractures, 1034**
 first aid, 982
 of capitulum, 1029
 of distal end, in adults, 1034
 of head, 1041
 of lateral condyle, in children, 1030
 of medial epicondyle, 1031
 of shaft, 1036
 of surgical neck, 1040
 supracondylar in children, 1032
 head, aseptic necrosis, in fracture dislocation of shoulder, 1045
- Hunter John, 4, 6, 16, 18**
- Hunter Whitlam, 16**
- "Hunting" in cold injuries, 200**
- Hutchinson's melanotic whiflow of hand, 1236**
- Hydronephrosis, production, 34**
- Hydral crystals, of mesenteric and omentum, 514**
 of spleen, 870
 pulmonary, 41
- Hydration, maintenance during operation, 106**
- Hydrocele, acute, 905**
 idiopathic, 906
 of cord, 906
 vs. indirect inguinal hernia, 530
 traumatic, 905
- Hydrocephalus, 215-1482**
 diagnosis, 1484
 in open bottle, 1477
 signs, 1484
 surgical treatment, 1484
 symptoms, 1484

- Intestine obstruction, parenteral replacement therapy in, 746
- partial, 732
- peritonitis and, 489
- postoperative early 730
- roentgenographic studies in, 74
- simple 732, 737
- treatment, 749
- strangulated hernia and, 736
- strangulation, 732, 737 740
- treatment and, 748
- immediate, 749
- toemia theory in, 738
- treatment, 745
- nonoperative, 747
- operative, 750
- vascular 732, 754
- volvulus and, 757
- rotation, incomplete. See *Intestinal malrotation*
- rupture peritonitis and, 489
- small, decompression in intestinal obstruction, 746
- infections, nonspecific, 687
- specific, 687
- infectious diseases involving, 687
- injuries, 685
- diagnosis, 686
- postoperative treatment, 687
- treatment, 686
- obstruction, clinical picture, 739
- diagnosis, 744
- strangulation in, 740
- treatment, 748
- systemic diseases and, relation, 688
- tumors, 690
- benign, 690
- malignant, 690
- prognosis, 692
- symptoms, 691
- treatment, 692
- stenosis, 577
- definition, 577
- diagnosis, 578
- volvulus. See *Intestine malrotation*
- Intoxication, water 133
- Intra-abdominal abscess as cause of postoperative fever 122
- Intra-abdominal pressure, increased, as factor in hernia, 521
- Intra-abdominal trauma. See *Trauma, intra-abdominal*
- Intracellular fluid, composition, 127
- volume, sodium ion concentration and, 177
- Intracerebral hematoma, treatment, 1418
- Intracranial abscesses, 1413 1430
- diagnosis, 1432
- pathology 1430
- signs, 1431
- sites, 1431
- symptoms, 1431
- treatment, 1432
- Intracranial aneurysms, 1433
- demonstration, 1434
- rupture, 1433
- Intracranial hemorrhage, traumatic, 1408
- vs. hypertrophic pyloric stenosis in vomiting in infants, 576
- Intracranial injury ulceration of upper gastrointestinal tract following, 614
- Intracranial pressure increased, in head injuries, pathology 1394
- in intracranial injuries, 1393
- Intracranial tumors, 1419
- classification, pathologic, 1420
- clinical syndromes, 1421
- congenital, 1420
- diagnosis, 1426
- differential diagnosis, 1428
- manifestations, 1421
- metastatic 1426
- pathologic classification, 1420
- radiation therapy 1429
- syndromes, 1423
- treatment, 1428
- surgical, 1428
- types, incidence 1422
- vs. intracranial abscess, 1432
- Intratumoral tension, increased, in intestinal obstruction, 736
- Intramedullary nail, Kuntscher in femoral shaft fractures, 1069 1071
- Intramedullary rod, fixation, infection following, 1125
- in Monteggia fractures, 1024
- in oblique or comminuted fractures of radius and ulna, 1021
- in ulnar shaft fractures, 1022
- Rush, in femoral shaft fractures, 1069 1071
- Intramedullary stem prosthesis in femoral neck fractures, 1064
- Intrapertoneal abscesses, 500
- in peritonitis, 493
- Intravenous feeding, prolonged, in convalescence 150
- Istibation, endotracheal, nasal, open, 166
- oral, 166
- closed, 166
- open, 166
- nasal, blind, 166
- Istmusception, idiopathic, 755
- in infancy 755
- in Meckel's diverticulum, 584
- intestinal obstruction in, 755
- Invertor muscles of foot, paralysis, 1287
- Involucrum formation in osteomyelitis, 1121
- Iodine, protein-bound, in hyperthyroidism, 319
- radioactive, in Graves disease, 319
- malignant disease following, 319 320
- in thyroid papillary carcinoma, 323
- in undifferentiated cancer of thyroid, 322
- uptake, in hyperthyroidism, 319
- Ion transfer in orthopedic disorders, 1317
- Irradiation. See *Radiation*
- Irritation, chemical, chronic cholecystitis and, 773
- in carcinoma of hand, 1235
- in carcinoma of hand, 1235
- Islets of Langerhans, tumors, 816
- diagnosis, 817
- functioning, 816
- hyperinsulinism and, 816
- nonfunctioning, 816
- peptic ulcer and, 818
- symptoms, 816
- treatment, 817
- Isobaric solutions, 173
- Isoniazid in pulmonary tuberculosis, 405
- Isonicotinic acid in pulmonary tuberculosis, 405
- Isotopes, radioactive in intracranial tumors, 1427
- JACKIT Milner, in cervical vertebral fractures, 1051
- Jackson, Charles T. 11
- Jacksonian seizures in head injuries, 1404
- Jagrikite 419
- Japanese finger traps. See *Finger traps Japanese*
- Jaundice, acholic, 846, 848
- hemolytic, congenital 846 848
- obstructive, 787
- in newborn, 764
- in pancreatic carcinoma, 811
- operation for vitamin K in, 782
- vs. hepatitis, 811
- Jaw(s) 274
- abscess, 255
- Actinomyces infection, 283
- ankylosis, 283
- false 283
- bone grafts to, 284
- carcinoma, 270 284
- metastatic 284
- chondrosarcoma, 284
- dislocation, anterior 276
- anterosuperior 276
- posterior 276
- fracture osteomyelitis and, 281
- infections, 279
- lower fracture, first aid 982
- osteofibroma, 284
- sarcoma, osteogenic, 284
- syphilis, 283
- tuberculosis, 282
- tumors, 283
- giant cell, 284
- upper fractures, 276
- transverse 276
- Jejunum, 685-692. See also *Intestine small*
- atresia and stenosis, 577
- duplex. See *Alimentary tract duplications*
- lymph vessels, 1368
- Jelly belly 513 698
- Jenner 17
- Joker(s) 978-1181
- acromioclavicular dislocations, 1046
- bones and, 978-1181
- capsules, hand tumors arising from, 1238
- effusion, traumatic, vs. pyarthrosis, 1133
- elbow dislocation, 1027
- foot, dislocations, 1099
- foreign bodies in, 1136
- hemophilic, vs. pyarthrosis, 1133
- hip, dislocations, 1094
- infections, acute and chronic, 1111-1147
- Progenic. See *Pyarthrosis*
- knee. See *Knee joint*
- motion, measurement, goniometer in, 1300
- mouse, 1093
- of lower extremity dislocations, 1093

- Immunologic response, alterations by antibiotics, 35
- Impotence, sexual, 916
causes, 917
in urinary tract disease, 857
incidence, 917
treatment, 917
- Inched wounds of neck, 707
- Incision, abdominal, ideal, 555
in infection, 50
inflammation, as cause of postoperative fever, 121
type, incisional hernia and, 554
- Inclusion cysts, dermoid, submental, 296
- Incontinence, in urinary tract disease, 856
in vaginal relaxations, 952
- Incubation period of infection, 48
- Infant(s) acute hematogenous osteomyelitis in, 1113
anal stenosis in, 774
burns in, treatment, 196
intestinal obstruction in, 755
intussusception in, 755
pyarthrosis in, 1138
- Infantism, primary, 919
sexual, 919
- Infection of omentum, 515
- Infection(s) 42-62
acquired, in hospitals, prevention and control, 58
Bacillus anthracis, 44
carotid sheath, 306
cervical, 300
chloramphenicol in, 58
chronic, in carcinoma of hand, 1235
clostridial, 44
course, factors in, local, 48
systemic, 48
influences on, 48
definition, 42
development, requisites for, 46
diagnosis, early accurate, 48
errors in, 49
etiologic, 48
effects, systemic, 47
foci of, extirpation, 50
fungal, 46
gram-negative bacilli and, 45
gram-positive bacteria and, 43
in burns, 187-192
in convalescence, 152
incubation period, 48
kanamycin in, 57
lag phase, 48
local effects, 47
Mycobacterium tuberculosis, 44
neomycin in, 57
of abdominal wall, 479
of bones and joints, 1111-1147
of jaw, 279
of larynx, 311
of mastoid space, 302
of neck, 300
of peritid space, 303
of pharyngomaxillary space, 303
of retropharyngeal space, 303
of salivary system, 286
of submandibular space, 304
of trachea, 314
of umbilicus, 479
of visceral space of neck, 306
penicillin in, 56
pneumoperitoneum and, 511
- Infection(s) polymyxin B in, 58
Proteus, 46
protozoal, 46
Pseudomonas, 46
pyogenic, of breast, 337
rectus sheath hematoma and, 481
resistance to, 47
nonspecific, natural mechanisms of, 43
Salmonella, 46
secondary protection against, 50
spirochetal, 44
staphylococcal, 43
streptococcal, 44
streptomycin in, 57
suppurative, following breast trauma, 336
surgical, antibiotics in, importance, 56
tetracyclines in, 57
therapeutic measures in, local, 49
specific, 51
supportive, 51
treatment, principles, 48
types of importance to surgeon, 42
virus, 46
wound, incisional hernia and, 554
postoperative, 111
prevention, history, 2
wound healing and, 33
- Infertility male, 917
causes, 917
treatment, 918
- Infiltration anesthesia, 169-175
- Inflammation, as reaction to infection, 47
definition, 23
treatment, principles of, 48
- Infracture, Freiberg's, 1290
- Infrared generator, 1306
- Infrared photography in breast lesions, 335
- Inguinal adenitis, 908
- Inguinal ligament in hernia repair, 54
- Inguinal lymph nodes, 908, 1366
anatomy, 908
tumors, metastatic, 908
- Inguinal wall, posterior reconstruction in hernia, 532
- Inguinofemoral region, anatomy, 525-535-537
hernia, 524
- Injuries See also *Wounds* and *Trauma*.
accidental, classification, 79
definitive care in, 82
craniovertebral See *Craniovertebral injuries*.
crushing, open, 81
in fractures, treatment, 991
intra-abdominal See *Trauma*.
reaction to, 23
severe care of, 67
soft tissue, from mechanical forces, 79-86
stress after early, 139
thermal and irradiation, 182-212
- Innominate artery aberrant, esophageal constriction by, 574
left common carotid artery originating from, 436
- Instrumentation, endoscopic, pneumoperitoneum and, 411
- Insufficiency circulatory bacterial factor in, 90
- Insufficiency circulatory balance between vasoconstrictor material and, 90
irreversible shock following, 81
mitral. See *Mitral insufficiency*.
renal, acute, management, 134
- Insults, 801-802
- Intensive care unit, 110
- Intensin states, 918
- Interstitial cells of Leydig, 897
- Interstitial fluid, composition, 127
- Intertrigo, vulval, 972
- Interventricular septal defects. See *Ventricular septal defects*.
- Intervertebral disk, 1444
cervical, "rigid", 1445
rupture, 1445
lumbar rupture, 1444
rupture, 1444
treatment, 1445
- Intestinal contents, aspiration, postoperative atelectasis and, 116
- Intestinal phase, of digestion, 632
of gastric acid secretion, 610
- Intestine, atresia, 577
definition, 577
diagnosis, 578
decompression, in intestinal obstruction, 746
embryology, 580
large decompression, 747
obstruction, 741
treatment, 748
- Malrotation, 580
diagnosis, 580
embryologic factors, 580
postoperative cure, 583
symptoms, 580
treatment, 580
- Nonrotation. See *Intestine malrotation*.
- Obstruction, 731-758
acute, 732
by adhesions, 756
by bands, 756
cause, specific lesions, 755
changing character of, 733
chronic, 732
classification, 732
clinical, 733
pathologic, 733
closed loop, 732
complete, 732
considerations, general, 731
decompression, 746
diagnosis, 739
differential diagnosis, 744
distention in, treatment, 749
diverticulum and, 757
fecal impaction and, 757
from malrotation, 580
gallstone ileus and, 757
gallstones in, 786
in colon cancer, 709
in infancy, 755
in Meckel's diverticulum, 583
intussusception and, 755
level of, treatment and, 748
mechanical, 732
physiopathology, 734
mechanisms, 733-734-735
Meckel's diverticulum and, 756
neurogenic. See *Neurogenic*.
nonstrangulating, 732
immediate dangers, 735
treatment, 749
onset, treatment and, 748

- Leukemia, 1385
 Leverage axis of foot, 1264
 Levon tube in distention prevention, 747
 padding, technique 76
 Leydig, interstitial cells of 897
 Lhermitte sign in intraspinal tumors, 1443
 Lidocaine 171
 Liebig, 5
 Ligament(s) 1101 1106
 calcaneonavicular 1261
 gastrocolic, 486
 gastroepiploic, 486
 inguinal, in hernia repair 54
 long plantar 1261
 of foot, 1261
 of knee collateral, tears of 1089
 cruciate anterior tears of 1091
 posterior tears of 1092
 sprain, 1108
 tears of 1089 1091
 treatment, 1092
 splenoportal, 843
 sprain, definition, 1106
 strain, definition, 1106
 Ligamentum arteriosum with right aortic arch, 436
 Ligature, history 8
 Limbs. See also *Arms Legs and Extremities*.
 artificial, 1177 1181
 Lines alba, 475
 anatomy 550
 hernia, 545
 Lines semicircular, anatomy 552
 of Douglas, 475
 Lines semilunars, hernia, 545
 of Spiegel, 475
 Lines, tension, of Langer 475
 Listeria plastica, 667 668
 Lip(s) carcinoma. See *Carcinoma of lip*.
 cleft, 257
 double embryologic cause 260
 repair 258 259 260
 residual deformities, repair 262
 single repair 257 258 259
 embryology 258
 keratoses, 266
 port-wine stains, 265
 sarcoma, 770
 tumors, benign, 265
 malignant, 267
 mixed, 267
 Lipase, pancreatic, 802
 Lipocalic, 803
 Lipoma, arborescent, of hand, 1238
 vs tuberculous synovitis, 1701
 articular 1172
 cardiac, 466
 gastroic, 664
 incidence, 663
 of abdominal wall, 482
 of breast, 346
 of colon, 708
 of esophagus, 601
 of hand, 1237
 of liver 795
 of muscle, 1104
 of salivary glands, 287 288
 of small intestine, 690
 pulmonary 423
 theory of Cloquet, 518, 551
 Liposarcoma, of bone, 1171
 of colon, 708
 of retroperitoneal space, 516
 Liposarcoma, of soft parts of extremities, 1173
 Listerine 700
 Lister Joseph, 5 6 15 16 1488
 Liston, Robert, 4 9 11 17
 Luteolipase 873
 Luteal 700
 Luteal hernia, definition, 520
 in femoral hernia, 540
 Luteal reticularis, 1351
 Liver 759 799
 abscess, amebic 798
 pathogenesis 798
 symptoms, 798
 treatment, 799
 pyogenic 797
 pathogenesis, 797
 symptoms, 797
 treatment, 797
 anatomy 760
 bile formation by 762
 carbohydrate metabolism and, 762
 carcinoma, 796
 metastatic, 796
 primary 796
 cutaneous, wound healing and, 39
 damage gallstones and, 766
 detoxification by 763
 diseases, 789-799
 erythrocyte production by 763
 fat metabolism and, 762
 fibrogen formation by 763
 hamartoma, 795
 in irreversible shock, 91
 injury 764
 treatment, 509
 insufficiency deaths due to in cholecystectomy 774
 lymph vessels, 1368
 physiology 762
 protein metabolism and, 764
 prothrombin formation by 763
 trauma to, 764
 tumors, 795
 benign, 795
 malignant, 796
 vasodepressor substance elaboration by 763
 vitamin metabolism and, 762
 Lobe, atypic, 409
 frontal, tumors, 1424
 middle, syndrome, 406
 occipital, tumors, 1424
 parietal, tumors, 1424
 pulmonary accessory 409
 temporal, abscess, 1431
 herniation in head injuries, 1402
 tumors 1424
 Lobectomy in bronchiectasis, 412
 in bronchogenic carcinoma, 471
 Lobotomy frontal, 1436, 1471
 bilateral, 1471
 prefrontal, 1436
 Lobster-claw hand, 1249
 Long, Crawford W 11
 Loach, 798
 Lottes nail in tibial shaft fractures, 1076
 Low sodium syndrome, 133
 in convalescence, 149
 Lowenberg's test in venous thrombosis, 1337
 Luckhardt, 12
 Ludwig's angina 256, 304
 Lumbar cord, injuries, 1439
 Lumbar pelvic traction, 1315
 Lumbar puncture in craniocerebral injuries, 1414 1418
 in head injuries, 1405
 in intracranial tumors, 1426
 in intraspinal tumors, 1443
 technique, 75
 Lumbar vertebrae transverse processes, fractures, 1054
 Lumbodorsal region, fracture-dislocations, 1054
 Lumbodorsal spine fractures, 1053
 Lunate bone aseptic necrosis, spontaneous, 1008
 dislocation, 1006
 Lung(s) See also *Pulmonary*.
 abscess, 405
 in bronchogenic carcinoma, 417
 postoperative atelectasis and 116
 actinomycosis, 413
 anatomy 398
 anomalies, congenital, 409
 blastomycosis, 413
 Boeck's sarcoid, 414
 bronchopulmonary segments, 399
 capacity reduction, 383
 coccioidomycosis, 413
 collapse massive 386
 cysts, congenital, 409
 echinococcus, 412
 hydatid, 412
 diseases, 398-415
 management, preoperative 103
 edema in head injury 1397
 function, 382, 400
 tests, 383, 401
 fungus infections, 413
 gangrene, 405
 hamartoma, 423
 hernia, 389
 histoplasmosis, 413
 injury blast, 388
 lipoid granuloma, 413
 lymphatic vessels, 1371
 lymphosarcoma, 422
 resection in pulmonary tuberculosis, 403
 sarcoma, 422
 stones, 408, 414
 suppuration, postoperative atelectasis and, 116
 syphilis, 414
 thromboembolism following phlebotrombosis, 1338
 tuberculosis. See *Tuberculosis, pulmonary*.
 tumors, 414
 benign, 423
 malignant, lymphatic, 422
 reticuloendothelial, 422
 metastatic, 422
 vanishing, phenomenon, 409
 ventilation, tests of, 383
 Lymph, anatomy 1362
 collections, congenital, 1375
 fistula, 1373
 nodes, anatomy 1364
 anterior tibial, 1366
 axillary 1365
 cervical, 1371
 deep, 1372
 superficial, 1371
 deep, 1365
 gastroic, 1366
 ileocolic, 1367
 in sarcoma, 1388
 inguinal, 908 1366

- Joint(s) sacrochac, tuberculous, 1143
sternoclavicular dislocation, 1048
temporomandibular disorders, 275
tuberculosis. See *Tuberculosis of bones and joints*.
tumors, 1148 1171
classification, 1171
wrist, fractures, 1003
- Jugular compression, test, 1432
- Junctions, myosaponrotic, of anterolateral abdominal muscles, 474
- KANAMYCIN in surgical infections, 57
toxic reactions, 56
- Kanavel, 1182
- Kaposi's sarcomas of soft parts of extremities, 1175
- Keller bonelectomy in osteoarthritis of foot, 1292
- Keller resection in arthritis of foot, 1791
- Keller Blake splints in lower extremity fractures, 983
- Keloid(s) formation in wounds, 29 29
secondary in lymphedema, 1381
tendency incision in, 112
- Keratosis of abdominal wall, 482
of hand, 1231
of lips, 266
- Kernig sign in meningitis, 1412
- Kerr 700
- 17 Ketosteroid excretion in hyperadrenocorticism, 829
- Kidney agenesis, 869 870
anomalies, 869
double, 869 870
failure, in convalescence, 155
in urinary calculus disease, 874
function tests, in prostatism, 912
in urinary tract disease, 859
horseshoe, 870
infection in pregnancy 868
injuries, 870
acute, hypernatremia in, 134
insufficiency acute, management, 134
lymph vessels, 1369
obstruction, adjustments to, 865
cancer, 864
pain, chronic, relief 1473
in urinary tract disease, 855
parenchymal tumors, classification, 880
pelvis, tumors, 880
clinical signs, 880
pathologic changes, 880
treatment, 880
penetrating injuries, 507
rupture, 870
tumors, 880
etiology 880
pathology 880
- Kienbock's disease, 1008
- Klimfelter's syndrome, 919
- Klimpke's palsy 1456
- Knee, housemaid's, 1109
joint, collateral ligaments, tears of, 1089
cruciate ligaments, tears of, 1089
dislocations, 1096
internal derangements, 1089
sprain, 1107
tuberculosis, 1144
- Knife wounds of neck, 307
- Knott technique, 1313
- Knuckle pads in Dupuytren's contracture, 1226
- Knyveton, John, 10 17
- Koch, Robert, 5 1134
- Kocher Theodore, 6
- Kocher maneuver in islet of Langerhans tumor operation, 817
- Kocher method in shoulder dislocation, 1044
- Koeberle, Eugene, 9
- Kohler's disease, 1290
- Koller 12
- Kondoleon operation in lymphedema, 1379
- Kraske, 700
- Kraurosis vulvae 975
- Kronlein operation in gastric cancer 671
- Krukenberg tumors, 972
- Kummel, 780
- Kunischer nail, in tibial shaft fractures, 1076
intramedullary in femoral shaft fractures, 1069 1071
- Kypsois in Pott's disease, 1140, 1141
- LARIA, lymphedema, 1382
- Laboratory clinical, procedures, in breast carcinoma, 357
studies in infection, 49
- Labyrinthotomy in Meniere's disease, 250
- Laceration(s) 81
of brain, 1394
in head injuries, 1393
of face, 253
of frontotemporal junction, 1402
of neck, 307
superficial, 307
of scalp, findings, 1406
of tongue, 54
- Lactation, breast carcinoma in, treatment, 362
- Lactula, 1373
- Ladd technique, in intestinal malrotation, 582
in small bowel stenosis or stricture, 579
- Lag phase, of infections, 48
of wound healing, 43, 77
- Lahry procedure in peptic ulcer 642
- Lamina dura, disappearance in hyperparathyroidism, 325
- Lamnectomy compression, in spinal injuries, 1440
- Landa-Gibbon test in peripheral vascular disorders, 1323
- Landfranch, 8, 15
- Langenbeck, Conrad, 18
- Langer tension lines of, 475
- Langerhans, islets of. See *Islets of Langerhans*.
- Laparotomy exploratory in gastric carcinoma, 672
- Larey 16
- Laryngopharynx, anatomy 37
- Larynx, 310-316
bullet wounds, 308
carcinoma, 313
disease, 310
edema, acute inflammatory 311
noninflammatory 31
foreign body in, 315
fractures, 310
- Larynx, infectious, 311
injuries, 310
paralysis, bilateral, 311
stenosis, 311
chronic, 312
cicatricial, 31
tumors, benign, 31
malignant, 313
wounds, 311
- Lauege sign in acute hematogenous osteomyelitis, 1115
- Lavater's hernia, definition, 520
- Lawrence test in venous thrombosis, 1337
- Lealett, coronary deformity in aortic insufficiency prostheses for 459
- Ledderhose's disease, 1293
- Leg(s) See also *Extremities*.
amputation, levels, 1177 1178
fracture, first aid, 983
function, prosthetic replacement and, 1180
immersion, 199
lymphangiography 1389
milk, 1338, 1375
postphlebotic, 1338 1375
spastic paralysis, foot deformities in, 1, 89
vena, 1328
- Leiomyoma, gastric, 664
incidence, 663
of colon, 708
of epididymis, 904
of esophagus, 601
of peritoneum, 512
of small intestine, 690
of uterus, 94... See also *Fibroids*.
vs. adrenogenital syndrome, 827
- Leiomyosarcoma, cardiac, 466
duodenal, 680
esophageal, 601
gastric, 664 666
clinical features, 667
treatment, 667
rectal, 729
- Lembert, 16
- Lens, myopia to, 222
- Leontine's eyes, 215
- Leptomenigitis vs. intracranial abscess, 1432
- Leriche's syndrome, 1353
- Lerone, 169
- Lesions, surface, evaluation, 1316
- Letterer-Siwe's disease, 16, 1166
- Leukemia, 1165
lymphatic, 1388
myeloid, bone changes in, 1165
vs. acute hematogenous osteomyelitis, 1117
vs. Hodgkin's disease, 1387
- Leukocyte count, in peritonitis, 493
in radiation myeloma, 210
- Leukocytosis in appendicitis, 694
- Leukopenia in radiation myeloma, 210
- Leukoplakia, of mouth, 266
of vulva, 975
carcinoma of vulva and, relation, 976
- Leukorrhea, 922
causes, differential diagnosis, 923
in chronic endocervicitis, 925
in endocervicitis, 974
in genital cancer 929
in gonorrhea, 926
in thrush, 928
in trichomoniasis, 923
in vaginitis, 928

- Mastoiditis, acute in otitis media 4
surgical treatment, indications, 43
Mata, 12
Mata's test of collateral blood flow 1345
Maxilla, osteomyelitis, 87
Mayo procedure in peptic ulcer 642
MBC 401
McDowell, Ephraim, 18 489 965
McKernick exclusion operation, 644
McKay inguinal hernioplasty 445
McWhorter technique in breast carcinoma, 358
Mechanical equipment, care of 69
Mechanical obstruction. See *Intestinal obstruction*
Meckel's diverticulum. See *Diverticulum Meckel's*
Meconium ileus, 488
Meconium peritonitis, 499
Median bar 91
Median nerve. See *Nerve median*
Mediastinitis, 371
diagnosis, 373
roentgenography in, 375
treatment, 373
Mediastinotomy cervical, 372
in mediastinal tumors, 380
Mediastinum, 369-381
abscess, 377
anatomy 369
anomalous vessels in, esophageal constriction by 374
chondromyxosarcoma, 378 380
diverticula, 376
emphysema, 369 387
gangliocarcinoma, 378
neuroblastoma, 378
neurofibroma, 378, 379
osteofibrosarcoma, 378
tumors, 376
location, 370
symptoms, 379
treatment, 380
Medical ethics, 1490
Medication, internal, in carcinoma of hand, 1, 35
Medicine, physical. See *Physical medicine*
separation from surgery 3
Mediterranean anemia, 852
Medulla, adrenal. See *Adrenal medulla*
herniation, in head injuries, 1403
Medulloblastoma, 1424
Meeserophagus, 594
dilation of esophagus in, 596
etiology 595
treatment, 596
types, 594
Megalocephaly 215
Meigs syndrome 968
Melanoma, of abdominal wall, 483
malignant, 482
of colon, malignant, 708
of hand, 1236
of hip, malignant, 268
Melanosarcoma, rectal, 729
Melena, in gastroduodenal hemorrhage, 650
in peptic ulcer 616, 619
Meisner Frank L, 7
Meltzer 12
Membrane, formation in diphtheritic tonsillopharyngitis, 234
Membrane mucosa, of gastrointestinal tract, healing of 40
peridental, disappearance in hyperparathyroidism, 345
pseudotumor, formation in infections of pharynx and tonsils, 233 234
nerve, healing, 40
synovial, 1128
Membrane hydrops theory of by diuretic acid secretion, 608
Menarche 933
Meniere's disease 49 1449
labyrinthotomy in, 50
medical management, 250
proliferators in, 250
Menigeal hemorrhage middle 1408
Meningioma, 1470, 1423 1425 144
incidence, 1441
of tuberculum sellae 1425
Meningitis, traumatic 1412
Meningocele with spina bifida, 1475 1476, 1477
treatment, 1478
Meningeal fractures, 1089 1090
mucoid cysts, 1089
temporomandibular syndrome 776
Menopause, 951
artificial, induction, in breast carcinoma, 363
Menorrhagia, 950
Menstruation, amount of flow 933
character of flow 933
cycle breast changes in, 379 332, 333
disorders, in chronic gonorrheal salpingitis, 958
in endometriosis, 964
in granulosa cell tumor 967
in ovarian failure 949
in thecal cell tumor 968
duration, 933
interval, 933
onset, 933
painful. See *Dysmenorrhea*
symptoms, associated, 933
Meperidine 169
Menses (ies) 513
adenitis, 687
vs. appendicitis, 695
anatomy 513
cysts, 514 See also *Alimentary tract duplications*
congenital, 517
embryology 484
physiology 513
Mesh, wire, in incisional hernia repair 560
Mesotendon, 1191
Mesothelioma, 512
pleural, 397
Mesothelium, tumor 512
Metabolic acidosis, postoperative, 132
Metabolic balance in management of extrarenal losses, 147
Metabolism, abnormal, skull diseases resulting from, 216
basal, raw, in hyperthyroidism, 319
calcium and phosphorus, parathyroid glands in, 323
cholesterol, chronic cholecystitis and, 772
gallstones and, 772
diabetes, effect on wound healing, 39
Metabolism endocrinology and in surgical care 138-156
fat liver and 762
pancreas and 803
in late convalescence 146
postoperative alterations in, 123
post-traumatic, 141
steroid, alterations, in breast carcinoma, 363
Metabolism, influence in irreversible shock, 91
Metal(s) as cause of osteomyelitis of jaw 281
cold injury and 198
cups in femoral neck fractures, 1064
use in surgery 34
Metatarsal, of foot, fractures, 1087
heads, prominent, 1282
Metatarsophalangeal dislocation, 1100
Metatarsus adductor, congenital, 1269
Metatarsus laterus, 1279
Metatarsus varus, 1279
congenital, 1269
Meteorism postoperative, 118
Methadone 169
Methanbelline in peptic ulcer 622
Methyl morphine, 169
Methyltestosterone in dysmenorrhea, 932
Metrorrhagia, 950
Metycaine, 171
Meyer Willy 19
Microbial flora, alteration by antibiotics, 55
Microbial invasion. See *Infection*
Microcephaly vs. craniosynostosis, 1480
Micrognathia in cleft lip and palate, repair of 262
Microorganisms of surgical importance, 43
Microtia, 251
Middle lobe syndrome, 406
Mildgut, embryology 590
Mildred dislocation of foot, 1099
Mikulicz, 700
Mikulicz procedure, in Meckel's diverticulum, 583
in peptic ulcer 644
Mikulicz resection with double-barreled ileostomy in meconium ileus, 583
Mikulicz Heineke pyloroplasty 642, 643
Milk leg, 1338, 1375
Milk of calcium gallbladder in chronic cholecystitis, 773
Milkers nodules, 1204
Miller Abbott tube, in decompression of small intestine, 746
passing, technique, 76
Miles's disease 1376
Mineralocorticoids, function, 124
Miner's elbow 1109
Minerva jacket in cervical vertebral fractures, 1051
Mitchell, Weir 1452, 1466
Mitral insufficiency 453
mitral stenosis and, 453
surgical correction, 452
surgical correction, 453
Mitral stenosis, 449
mechanism, 450

- Lymph, nodes, inguinal, anatomy 908
 tumors, metastatic, 908
 involvement in cervical cancer 934
mesenteric, superior 1367
mesocolic, 1368
 metastasis of mammary carcinoma to 350
parietal, 1366 1369
pectoral, 1365
popliteal, 1366
 regional, involvement, in bronchogenic carcinoma, 420
scalene, biopsy in bronchogenic carcinoma, 419
subclavicular, 1366
sublingual, 1366
 enlarged, vs. femoral hernia, 539
submaxillary 1371
submental, 1371
subscapular 1365
superficial, 1365
suprahyoid, 1371
supraclavicular, 1368
 venographic demonstration, 1390
visceral, 1366 1369 1370
 obstruction. See *Lymphadenoma*
 stasis. See *Lymphedema*
 Lymphadenectomy pelvic, bilateral, in carcinoma of corpus uteri, 939
 Lymphadenitis, 1383
 inguinal, secondary in chancre, 974
mesenteric, 513
 acute, nonspecific, 513
 tuberculous, 514
 retroperitoneal, involving hip joint, vs. pyarthrosis, 1133
 syphilitic, 1383
 tuberculous, 1383
 vs. Hodgkin's disease, 1387
 vs. metastatic lymph node tumors, 908
 Lymphadenoma, malignant. See *Hodgkin's disease*
 Lymphangiectasia, congenital, 1385
 cystic, of mesenteries and omentum, 514
 Lymphangiography 1388
 of leg and thigh, 1389
 Lymphangoma (*) 1384
 bone involvement in, 1170
 cavernous, 1385
 cavernous, of mesenteries and omentum, 514
 cystic, 1385
 of hand, 1244
 of joints, 1172
 of lips and mouth, 66
 of salivary glands, 287 288
 simplex, 1384
 Lymphangitis, acute, 1382
 chronic, 1383
 of hand, 1196
 tuberculous, 1383
 Lymphatic drainage, of abdomen and pelvis, 1366
 of abdominal wall, 476
 of thorax, 1369
 Lymphatic duct, injuries, 1373
 right, 1364
 Lymphatic system, anatomy 1362
 carcinoma, 1388
 Lymphatic system, carcinoma, vs. Hodgkin's disease, 1387 1388
 definition, 1361
 embryology 1362
 injuries, 1373
 physiology 1372
 tumors, 1384
 Lymphatics, 1361-1391
 anatomy 1364
 anorectal, 716
 cutaneous, of breast, 329
 infectious, 1382
 intramammary of breast, 329
 of abdominal and pelvic viscera, 1368
 of head, 1371
 of lower extremity 1366
 of neck, 1371
 of upper extremity 1365
 small, injuries, 1373
 Lymphedema, 1374
 allergic, 1375
 congenital, 1376
 of upper extremity 1382
 essential, 1376
 following thrombophlebitis, 1338
 infectious, 1375
 labia, 1382
 lymphangiography in, 1390
 malignant, 1376
 of lower extremity 1374
 etiology 1374
 of upper extremity 1381
 pathology 1378
 post-thrombotic, 1375
 scrotum, 897 1382
 specific, 1374
 symptoms, 1376
 traumatic, 1375
 treatment, 1378
 wound healing and, 37
 Lymphoblastoma, 1385
 Lymphocyte count in radiation injuries, 211
 Lymphocytoma, 1385
 gastric, 666
 Lymphogranuloma inguinale, 908, 1384
 in female, 974
 Lymphogranuloma venereum, 908
 in female, 974
 virus in rectal structure, 725
 Lymphoma, macrofollicular of stomach, 666
 malignant, gastric, 666
 diagnosis, 666
 gastrointestinal, 666
 of colon, 708
 of retroperitoneal space, 516
 Lymphopathia venereum, 908
 Lymphopenia in radiation injuries, 211
 Lymphorrhagia, 1373
 Lymphorrhea, 1373
 Lymphosarcoma, 1385
 cardiac, 466
 gastric, 666
 of bone, 1169
 of esophagus, 601
 of lung, 422
 of rectum, 729
 of retroperitoneal space, 516
 of small intestine, 691
 vs. Hodgkin's disease, 1387
 Lymphosarcomatous, 1385
 Lynch frontoethmoid operation in frontal sinusitis, 231
 Macrocheilia, 1385
 Macrocheilia, 1385
 Macroducty in foot, 1272
 Macrogloma, 1385
 Macules, Singer's, 297
 Maffucci's syndrome, 1152
 Magnesium, balance, 129
 compounds in peptic ulcer 622
 Malaria, splenic involvement in, 851
 Males, breast carcinoma in, 366
 hypogonadism in, 919
 pseudobermaphrodism in, 919
 reproductive system, 890-920
 Malformation, Arnold-Chiari, 1477 1484
 Malignancy, 16
 Malignancy, double vertical fracture of, 1059
 "Malleolus, posterior" 1078
 fractures, 1079
 Mallet finger 998 1218
 Mallet tapping in fractures, 994
 Malnutrition in cold injury 194
 operative risk and, 98
 vitamin therapy in, preoperative, 103
 Mainstem artery implantation into myocardium in coronary artery disease, 461
 internal, ligation in coronary artery disease, 461
 Mammary duct ectasia, 338
 Mandible, condyle, fractures, 775
 fractures, 274
 Maneuver Hoguet, in hernia repair 537
 Mann-Whitman dog, experimental ulcer and, 634
 Manual arts therapy definition, 1298
 in orthopedic disorders, 1317
 in surgical conditions, 1325
 March fractures, 1087
 Marcy H. O., 544
 Marginal ulcer See *Stomach ulcer*
 Marrow tumors, 1163
 Marrow transplantation of pancreatic cyst, 810
 Massage, 1308
 cardiac, in cardiac arrest, 109 468
 compression, 1309
 deep stroking, 1309
 friction, 1309
 in lower motor neuron disorders, 1320
 in orthopedic disorders, 1317
 kneading, 1309
 percussion, 1309
 superficial stroking, 1309
 Mastectomy lymphedema following, 1381
 radical, 360
 chemotherapy in, 361
 in breast carcinoma, radiation therapy following, 358, 361
 postoperative care, 361
 simple, and massive postoperative roentgenotherapy in breast carcinoma, 358
 Mastectomy space, 302
 infection, 302
 Mastitis, chronic cystic, 340
 plasma cell, 338
 Mastectomy radical, in chronic otitis media, 246
 modified, in chronic otitis media, 246

- Neck, wounds, superficial, 307
treatment, 308
- Necrosis, coagulation, of muscle in
cold injury 201
- Nat. of breast following trauma,
337
pressure prevention during oper-
ation, 106
slow of muscle in cold injury 201
thermal, of jaw 281
- Needle biopsy in breast carcinoma
356
technique 356
- Needling of fracture site 994
- Neihen method of artificial respi-
ration, 179
- Nelherian infection. See *Conorheia*.
- Nelson, 16
- Neomycin, in surgical infections, 57
toxic reactions, 56
- Neoplasms. See *Tumors*
- Nephrectomy in renal carcinoma,
831
- Nerve(s) acoustic, disorders, 1449
- axillary injuries, 1456
block, 169 175
in fracture treatment, 985
- cranial, 1446-1450
injury in head injuries, 1403
sensory section of 1470
damage in hand injuries, 1707
excision, in wound cleansing, 84
- facial, disorders, 1448
preservation in removal of saliv-
ary gland tumors, 287
trauma, 1449
- glossopharyngeal, disorders, 1449
healing, 40
- hypoglossal, disorders, 1450
in reconstructive surgery of hand,
1253
- infraorbital, avulsion, in trigeminal
neuralgia, 1447
- injuries, in fractures, 991
in Volkmann's contracture 1221
- median, compression in carpal
tunnel, 1458
- injuries, 1453
paralysis, surgery in, 1-54
ulnar nerve and, combined in-
juries, 1455
- of abdominal wall, 475
of anorectum, 716
of hand, tumors, 1246
- paralysis, in bronchogenic carci-
noma, 420
- peripheral, 1451-1460
injuries, 1451
after-care, 1457
diagnosis, 1452
distribution, 1451
surgical treatment, 1456
sweating pattern tests in, 1304
- symptoms, 1451
- palsy tardy 1457
- pathology 1451
- tumors, 1458
metastatic, 1459
- peroneal, injuries, 1456
- radial, injuries, 1452
paralysis, surgery in, 1254
repair in hand injuries, 1210
- root, symptoms in intraspinal tu-
mors, 1442
- scatic, injuries, 1456
in hip dislocations, 1096
- sheath, tumors, 1442
- Nerve(s) spinal accessory disorders,
145
- supraorbital avulsion, in trigem-
inal neuralgia, 1447
- tibial, injuries, 1456
those healing of 40
in cold injury 201
- trigeminal, 1446
- ulnar injuries, 1454
median nerve and, combined in-
juries, 1455
paralysis surgery in, 1254
tardy 1458
- vagus, sensory fibers, disorders,
1449
- Nervous factors in inhibition of gas-
tric acid secretion 612
- Nervous symptoms of ovarian failure,
948
- Nervous system, 139-1485
- autonomic, 1461-1474
blood pressure regulation and
1466
central, 1419-1460
anomalies, congenital, 1475-
1485
craniosacral, 1461
depression, progressive in anes-
thesia, 159
parasympathetic, 1461
peripheral, 1419-1460
sympathetic, 1461
thoracolumbar 1461
- Nervus intermedius of Wrisberg,
1448
- Nesacaine 171
- Neutrian, preservation of Greek
medicine by 14
- Neuralgia, glossopharyngeal, 1449
- segmental, pelvic pain in, 931
- segmental, 1446
alcohol injection in, 1446
avulsion of supraorbital or in-
fraorbital nerves in, 1447
pain in, 1446
- Neurectomy presacral, in dysenor-
rhea, 932
in endometriosis, 964
in pelvic pain, 930
- Neurogenic cyst, 1480
- Neurinoma, 1459
malignant, bone involvement in,
1170
of soft parts of extremities, 1175
- Neurinoma(s) 1420, 1459
acoustic, 1425
- Neuritis, optic, as complication of
sinusitis, 429
- retrobulbar as complication of
sinusitis, 229
- Neuroblastoma, mediastinal, 378
sympatheticum, 839
- Neurofibrosarcoma, 1459
- Neurofibroma, gastric, 664
of abdominal wall, 482
of mediastinum, 378 379
of peripheral nerves, 1459
- Neurofibromatosis, 1459
- Neurofibrosarcoma, 1459
mediastinal, 378
- Neurogenic pathway in relay of stress
from hypothalamus to stomach,
611
- Neurogenic phase, of digestion, 632
of gastric acid secretion, 610
- Neurogenic symptoms of ovarian
failure, 949
- Neurologic disorders, physical medi-
cine and rehabilitation in, 1317
- Neurologic manifestations in head
injuries, 1401
- Neurologic syndromes in head injur-
ies 1402
- Neuroma(s) fusiform, of hand, 1246
interdigital, 1282
of peripheral nerves, 1459
plexiform, of hand 1247
- Neuromuscular disorders, chronic
in 1302
direct current impulse stimuli in
1301
direct current tetanus ratio in
1307
electrical skin resistance meas-
urements in, 1304
electrodiagnostic procedures in,
1301
electromyography in, 1307
repetitive stimulation in, 1302
rheobase in, 1302
ratio 1302
strength-duration curve in, 1307
tetanizing current in, 1301
- Neuromuscular re-education, 1308
and relaxation in upper motor
neuron disorders, 1318
in lower motor neuron disorders,
1320
- Neuron, motor lower disorders,
1320
dynamic splints in, 1320
electrical stimulation in,
1320
electrodiagnostic proce-
dures in, 1301
beat in, 1320
massage in, 1320
neuromuscular re-education
in, 1320
physical medicine and re-
habilitation in, 1320
objectives, 1320
procedures, 1320
therapeutic exercise in, 1320
traction in, 1320
upper disorders, heat in, 1318
neuromuscular re-education
and relaxation in, 1318
physical medicine and re-
habilitation in, 1317
proper positioning in, 1317
self-care activities in, 131.
therapeutic exercises in,
1318
- Neurosurgery of hand, 1 47
- Neurosurgery in relief of pain, 1469
- Neutropenia, splenic, primary 846
848
- Neutrophils in radiation injuries, 211
- Nevus(i) lymphaticus, 1385
of abdominal wall, 482
of lips and mouth, blue, 263
combined, 263
intralesional, 63
junctional, 263
types, 263
- Newborn, diaphragmatic hernia in,
treatment, 566
intestinal stenosis and atresia in,
symptoms, 577
obstructive jaundice in, 764
ophthalmia, 479
- Nicoladoni-Brannhan phenomenon in
arteriovenous fistulae, 1358

- Mitral stenosis, mitral insufficiency and, 453
 surgical correction, 452
 postoperative complications, 452
 surgical correction, 451
 contraindications, 451
 indications, 450
 results, 452
 symptoms, 450
 valvuloplasty in, low sodium syndrome following, 133
- Moles, pigmented, on hand, 1231
- Mongolism, congenital duodenal obstruction and, 579
- Monteggia fracture 1022
- Morcel, 9
- Morgagni, hernia of, 562
 treatment, 566
- Morrison, hepatorenal pouch of 502
- Morphine, 169
- Morton, William T. 11
- Morton's toe, 1282
- Motor activity gastroduodenal, 61.
- Motor neurons. See *Neurons motor*
- Mott, Valentine, 18
- Moore, joint, 1093
- Mouth, 252-290
 congenital deformities, 257
 effect of vitamin deficiencies, 256
 floor carcinoma, 270
 lacerations inside, 254
 leukoplakia, 266
 phlegmon, septic, 256
 sarcoma, 270
 soft tissue, injury 252
 infection, 255
 syphilis, 256
 trench, 234
 tuberculous, 256
 tumors, benign, 263
 malignant, 267
 mixed, 267
 ulcer biopsy 271
- Mouth-to-mouth breathing in artificial respiration, 179
- Movement of patients, shock and, 91
- Moynihan, 1468
- Moynihan gastrojejunostomy 640
- Mucocoele of appendix, 698
- Mucoid cysts of semilunar cartilages, 1049
- Mucosa, gastric, 610
 prolapse, 629
- Mucoviscidosis, meconium ileus and, 588
- Mucus, gastric, secretion, 609
- Multiple suture technique in cavernous angioma of mouth, 265
- Mumps orchitis, 900
 vs. acute epididymitis, 900
- Munro, Alexander 16
- Munro, John, 16
- Murmur cardiac. See *Heart murmur*
- Murphy 700
- Muscle(s) 1101
 abdominal, anterolateral, myoaponeurotic junctions, 474
 abductor of larynx, paralysis, bilateral, 311
 atrophy in cold injury 201
 calf, paralysis, 1288
 contraction, isometric, 1307
 denervated, electrical stimulation, 1312
 dorsiflexors, of foot, paralysis, 1285
- Muscle(s) evertors, of foot, paralysis, 1286
 fibers, herniation, 1104
 gastric, tone changes in, 613
 healing, 40
 in cold injury 201
 intrinsic of foot, paralysis, 1288
 inverter of foot, paralysis, 1287
 of abdominal wall, 474
 abnormalities, congenital 476
 agenesis, 476
 aponeurosis, 480
 of toes, extensors, paralysis, 1285
 plantar flexor of foot, paralysis, 1288
 relaxants, 169
 setting, 1307
 skeletal, absence, 1102
 attrition, 1104
 degeneration, 1104
 hematogenous infection, 1103
 repair 1102
 rupture, 1102
 trauma, 1102
 Trichinella spiralis infection, 1103
 tumors, 1104
 spasm, in appendicitis, 694
 in spinal injuries, 1440
 strength, measurement, dynamometer in, 1300
 tumors malignant, 1104
 metastatic, 1104
 viability determination, 84
 voluntary atrophy 1102
 hypertrophy 1102
- Musculosponurotic deficiencies as factor in hernia, 521
- Mycobacterium tuberculosis infection, 44
- Mycostatin, in superinfection, 58
 in thrush, 928
- Mycotic aneurysms of aortic arch, 463
- Myelitis, advanced, in intraspinal tumors, 1443
- Myelography in intraspinal tumors, 1443
- Myeloma, endothelial, 1163
 multiple, 1164
 of skull, 216
- Myelomeningocele with spine bifida, 1475 1476 1477
 treatment, 1478
- Myoaponeurotic junctions of anterolateral abdominal muscles, 474
- Myoma(s) of epididymis, 904
 of lips and mouth, 263
 of liver 796
 of small intestine 690
 of uterus, 942. See also *Fibroids*
- Myomectomy in fibroids 944
- Myometer in measurement of muscle strength, 1300
- Myosarcoma, bone involvement in, 1171
 rectal, 729
- Myositis, 1103
 clostridial, 45
 osseous, 1103, 1158
 circumscribed, 1159
 progressive, 1159
- Myringoplasty in chronic otitis media, 245
- Myringotomy in ear drum inflammation, 43
- Myxedema, following thyroidectomy 319
 postoperative, 154
- Myxoma, cardiac, 466 467
 of hand, 1246
- Myxosarcoma, cardiac, 466
- NABOTHIAN cysts of cervix, 925
- Nail, intramedullary in open fractures, 987
 Kitchner in femoral shaft fractures, 1069 1071
 Lottes, in tibial shaft fractures, 1076
 Smith-Petersen, in femoral neck fracture, 1065
 Street, in femoral shaft fractures, 1071
- Nail-plate fixation, of intertrochanteric fractures, 1068
 of subtrochanteric fractures, 1069
- Narcotics, 169
 basal, 167
 in anesthesia, 158
 in burns, 193
- Nares, stenosis, 226
- Nasogastric suction in peptic ulcer perforation, 650
- Nasopharynx, anatomy 232
 epidermoid carcinoma of, 241
 fibroma, 240
 tumors, 237
 benign, 240
 malignant, 240
- Nasoes, in peptic ulcer 616
 in stoma ulcer 617
- Navicular bone, fractures, 1003 1045
 nonunion, 1004
 radial styloidectomy in, 1006
 types, 1003
- Neck, 291-316
 anatomy applied, 301
 anomalies, developmental, 291
 lateral, 291
 lateral, clinical course, 297
 diagnosis, 293
 embryology and pathology correlation, 293
 pathology 292
 treatment, 294
 trachea, 294
 dissection, in carcinoma of lips, 269
 in carcinoma of tongue, 273
 embryology 291
 fascial spaces, 302
 hygroma, 266 288
 infections, deep, 300
 etiology 300
 lymphatic supply 1371
 rigidity in craniocerebral injuries, 1401
 sprained, 1108
 surgery and head surgery antibiotic prophylaxis in, 60
 teratomas, 296
 tumors, primary 298
 vascular injuries, 308
 visceral injuries, 308
 visceral space infections, 306
 wounds, 306
 deep 307
 effects, delayed or secondary 308

- Mitral stenosis, mitral insufficiency and, 453
 surgical correction, 452
 postoperative complications, 452
 surgical correction, 451
 contraindications, 451
 indications, 450
 results, 452
 symptoms, 450
 valvuloplasty in, low sodium syndrome following, 133
- Moles, pigmented, on hand, 1231
- Mongolian, congenital duodenal obstruction and, 579
- Mongrel fracture 1022
- Morel, 9
- Morgagni, hernia of 562
 treatment, 566
- Morison, hepatorenal pouch of 502
- Morphine, 169
- Morton, William T. 11
- Morton's toe, 1282
- Motor activity gastroduodenal, 612
- Motor neurons. See *Neurons motor*
- Mott, Valentine, 18
- Mouse, joint, 1093
- Mouth, 252-290
 congenital deformities, 257
 effect of vitamin deficiencies, 256
 floor carcinoma, 270
 lacerations inside, 254
 leukoplakia, 266
 phlegmon, septic, 256
 sarcoma, 270
 soft tissue, injury 252
 infection, 255
 syphilis, 256
 trench, 234
 tuberculosis, 256
 tumors, benign, 263
 malignant, 267
 mixed, 267
 ulcer biopsy 271
- Mouth-to-mouth breathing in artificial respiration, 179
- Movement of patients, shock and, 91
- Moynihan, 1488
- Moynihan gastroyjunostomy 640
- Mucocele of appendix, 698
- Mucoid cysts of semilunar cartilages, 1089
- Mucosa, gastric, 610
 prolapse, 629
- Mucovaccinosis, meconium ileus and, 588
- Mucus, gastric, secretion, 609
- Multiple suture technique in cavernous angoma of mouth, 65
- Mumps orchitis, 900
 vs. acute epididymitis, 900
- Munro, Alexander 16
- Munro John, 16
- Murmur cardiac. See *Heart murmur*
- Murphy 700
- Muscle(s) 1101
 abdominal, anterolateral, myoaponeurotic junctions, 474
 abductor of larynx, paralysis, bilateral, 311
 atrophy in cold injury 201
 calf paralysis, 1288
 contraction, isometric, 1307
 denervated, electrical stimulation, 1312
 dorsiflexors, of foot, paralysis, 1285
- Muscle(s) evertors, of foot, paralysis, 1286
 fibers herniation, 1104
 gastric, tone changes in, 613
 healing, 40
 in cold injury 201
 intrinsic, of foot, paralysis, 1288
 inverter of foot, paralysis, 1287
 of abdominal wall, 474
 abnormalities, congenital, 476
 atrophy, 476
 spasm, 480
 of toes, extensors, paralysis, 1285
 plantar flexor of foot, paralysis, 1288
 relaxants, 169
 setting, 1307
 skeletal, absence 1102
 attrition, 1104
 degeneration, 1104
 hematogenous infection, 1103
 repair 1102
 rupture, 1102
 trauma, 1102
 Trichinella spiralis infection, 1103
 tumors, 1104
 spasm, in appendicitis, 694
 in spinal injuries, 1440
 strength, measurement, dynamometer in, 1300
 tumors, malignant, 1104
 metastatic, 1104
 viability determination, 84
 voluntary atrophy 1102
 hypertrophy 1102
- Musculoponeurotic deficiencies as factor in hernia, 521
- Mycobacterium tuberculosis infection, 44
- Mycostatin, in superinfection, 58
 in thrush, 928
- Mycotic aneurysms of aortic arch, 463
- Myelitis, advanced, in intraspinal tumors, 1443
- Myelography in intraspinal tumors, 1443
- Myeloma, endothelial, 1163
 multiple, 1164
 of skull, 216
- Myelomeningocele with spina bifida, 1475 1476 1477
 treatment, 1478
- Myoaponeurotic junctions of anterolateral abdominal muscles, 474
- Myoma(s) of epididymis, 904
 of lips and mouth, 263
 of liver 796
 of small intestine, 690
 of uterus, 942. See also *Fibroids*
- Myomectomy in fibroids, 944
- Myometer in measurement of muscle strength, 1300
- Myosarcoma, bone involvement in, 1171
 rectal, 729
- Myotum, 1103
 clonidial, 45
 ossificans, 1103 1158
 circumscribed, 1159
 progressive, 1159
- Myrtoplasty in chronic otitis media, 245
- Myringotomy in ear drum inflammation, 43
- Myxedema, following thyroidectomy 319
 postoperative 154
- Myxoma, card ac, 466, 467
 of hand, 1 46
- Myxosarcoma, cardiac, 466
- NABOTHIAN cysts of cervix, 925
- Nail, intramedullary in open fractures, 987
 Kuntzner in femoral shaft fractures, 1069 1071
 Lottes, in tibial shaft fractures, 1076
 Smith-Petersen, in femoral neck fracture, 1065
 Street, in femoral shaft fractures, 1071
- Nail-plate fixation, of intertrochanteric fractures, 1068
 of subtrochanteric fractures, 1069
- Narcotics, 169
 basal, 167
 in anesthesia, 158
 in burns, 193
- Nares, atresia, 26
- Nasogastric suction in peptic ulcer perforation, 650
- Nasopharynx, anatomy 32
 epidermoid carcinoma of 41
 fibroma, 40
 tumors, 237
 benign, 240
 malignant, 40
- Nausea, in peptic ulcer 616
 in stoma ulcer 617
- Navicular bone, fractures, 1003, 1085
 nonunion, 1004
 radial styloidectomy in, 1006
 types, 1003
- Neck, 291-316
 anatomy applied, 301
 anomalies, developmental, 291
 lateral, 291
 lateral, clinical course, 292
 diagnosis, 293
 embryology and pathology correlation, 293
 pathology 297
 treatment, 294
 midline, 294
 dissection, in carcinoma of lips, 269
 in carcinoma of tongue, 273
 embryology 291
 fascial spaces, 302
 hygroma, 266, 288
 infections, deep, 300
 etiology 300
 lymphatic supply 1371
 rigidity in craniocerebral injuries, 1401
 sprained, 1104
 surgery and head surgery antiseptic prophylaxis in, 60
 teratomas, 296
 tumors, primary 298
 vascular injuries, 308
 visceral injuries, 308
 visceral space infections, 306
 wounds, 306
 deep 307
 effects, delayed or secondary 308

- Otitis(s)** 1157
 of auditory canal, 251
 of hand, 1 46
 osteoid, 1 45
 of skull, 216
 osteoid, 1158
 parotid, 1158
- Otitis media, alveolar upper 287**
 definition, 1111
 due to direct contamination, 1112, 1124
 etiology 11 4
 pathologic anatomy 11 3
 treatment, 11 6
- Otitis media, acute 111**
 differential diagnosis, 1117
 drainage in, 1118
 etiology 1112
 in adults, 1115
 in children, 1114
 in infants, 1113
 laboratory findings, 1116
 mortality 11 70
 of vertebral column, 1115
 prognosis, 1119
 prophylaxis, 1117
 signs, 1113
 symptoms, 1113
 treatment, 1117
 vs. pyarthrosis, 1132
- Otitis media, chronic, 1121**
 prognosis, 11 4
 treatment, 1122
- Otitis media, 282**
 of foot, 1294
 of hand, 1190
 of jaw 279
 of rib, acute, 390
 typhoid, 390
 of skull, 216, 1413
 as complication of stenoma, 229
- Osteophytes, loose bodies in knee joint and, 1092**
- Osteotomy in femoral neck fractures, 1064**
 supracondylar transverse, in supracondylar humeral fractures in children, 1033
- Oxium primum, permanent, 442**
- Oxium secundum, permanent, 442**
 surgical treatment, 444
- Otitis media, acute, 742**
 chronic 245
 cholesteatoma in, 246
 myringoplasty in, 245
 radical mastoidectomy in, 246
 modified, 246
 tympanoplasty in, 246
 types, 245
 mucous, chronic, 244
 recurrent, in tonsil and adenoid infections, 235
 serous, 243
- Otitis media, 248**
 fenestration operation in, 249
 stapes mobilization in, 248
- Ovary(ies) adenocarcinoma, primary solid, 969**
 carcinoma. See *Carcinoma of ovary*
 cyst, dermoid, 969
 twisted, vs. appendicitis, 695
 cystadenocarcinoma, pseudomucinous, 971
 sarcoma, 970
 cystadenoma, 965
- Ovary(ies) cystadenoma incidence 965**
 pseudomucinous, 965
 serous, 965
 types, 965
- embryology 960**
 failure causes, 947
 endocrine disorders due to, 947
 extrinsic 947
 intrinsic 947
 primary 947
 diagnosis, 949
 secondary 947
 symptoms, 947
 treatment, 950
- fibromas, 968**
 granulosa cell tumor 967
 lesions, vs. adrenogenital syndrome 827
 lymph vessels, 1369
 palpation, 955
 polycystic, 962
 removal in breast carcinoma, 363
 364
 unilateral, in dysgerminoma, 966
 in granulosa cell tumor 968
- sarcoma, 971**
 teratoma, 971
 thecal cell, 968
 tumors 959
 classification, clinical, 961
 histologic, 960
 masculinizing, 966
 nonproliferative, 962
 symptoms, 961
- Overatropinization, postoperative atelectasis and, 116**
- Overhydration, in heart disease pre-operative prevention, 103**
 in peritonitis, 496
- Ovulation, suppression, in dysmenorrhea, 932**
- Oxford modification of DeLorme exercises 1307**
- Oxygen, consumption by organ systems, regional blood flow and, 89**
 in shock treatment, 93
 tension, determination, 401
- Oxygenator(s) bubble type, 428**
 disposable, 428
 membrane type, 428
 pump, in cardiac surgery 427
 types, 428
 rotating disk type, 428
 stationary screen type, 428
 types, 428
- Oxytetracycline in surgical infections, 57** See also *Tetracyclines*
- PACEMAKER, artificial, in complete heart block, 446**
 external, in cardiac arrest, 448
 Pachymeningitis, externa, 1432
 arteria. See *Abacera, subdural*
 vs. intracranial abscess, 1432
- Pads, knuckle, in Dupuytren's contracture, 1226**
- Paget type of breast carcinoma, 347**
- Paget's disease of skull, 216**
- Pain(s) abdominal, cramping, in intestinal obstruction, 739**
 in appendicitis, 693
 central misinterpretation, 488
 cerebral, pelvic, 929
 conduction, from internal organs, 1471
- Pain(s) genitourinary referral of 857**
 in bronchogenic carcinoma 415
 in cervical cancer 936
 in diverticulitis, 704
 in duodenal ulcer 617
 in endometriosis, 964
 in esophageal carcinoma, 602
 in esophageal lesions, 594
 in extremities, relief 1466
 in female reproductive system diseases, 929
 in fibroids, 943
 in ovarian tumors, 962
 in peptic ulcer 615
 in peritonitis 491
 in relapsing pancreatitis, 806
 operation to relieve 808
 in stoma ulcer 617
 in subphrenic abscess, 504
 in trigeminal neuralgia, 1446
 in urinary calculous disease, 874
 in urinary tract disease 855
 menstrual. See *Dysmenorrhea*
 organic, pelvic, 929
 pelvic, in female 929
 causes, 929
 cerebral, 929
 organic 929
 peritoneal, 930
 psychogenic, 929
 somatic, 930
 types, 929
 peritoneal, pelvic, 930
 psychogenic, pelvic, 929
 relief in malignant disease of pharynx, 1470
 neurosurgical, 1469
 somatic, pelvic, 930
- Palate cleft, 257**
 dental requirements in, 262
 double embryologic cause, 260
 repair 258, 260, 261
 residual deformities, repair 262
 types, 260
 embryology 258
- Palsy See also Paralysis**
 Bell's, 1448
 drummer's, 1216
 Erb's, 1456
 Klumpke's, 1456
 nerve, tardy 1457
 pressure of ulnar nerve, prevention during operation, 106
- Pancoast syndrome, 1459**
 in bronchogenic carcinoma, 420
- Pancoast tumor 397**
- Pancreas, 800-819**
 aberrant, in small intestine, 690
 incidence, 663
 anatomy 800
 anular 587 803
 anomalies, 803
 body carcinoma, 815
 calculi, 808
 carcinoma. See *Carcinoma of pancreas*
 cystadenocarcinoma 809
 cystadenoma, 809
 cystic lesions, 808
 cysts, diagnosis, 809
 retention, 809
 symptoms, 809
 treatment, 810
 embryology 485
 fibrocystic disease, meconium ileus and, 588

- Niemann-Pick disease, splenomegaly in, 852
- Night cries, 1139-1141
- Niles, rule of, in burn estimation, 184-185
- Nipple, discharge, 335
breast dysplasia treatment in, 344
supernumerary, 336
- Nissen, 169
- Nissen exclusion operation, 644
in peptic ulcer, 646
- Nitrogen, balance, control in malnourished patient, 100
in postoperative patient, 124
metabolism, in anabolic phase of convalescence, 144
post-traumatic, 141
- Nitrous oxide, 16.
history, 10
- Nitrous oxide-oxygen, history, 12
- Nocardia infection, 46
- Nocturia in urinary tract disease, 856
- Nodes, lymph. See *Lymph nodes*.
- Stable's, 1371
- Virchow's, in gastric carcinoma, 671
- Nodule(s) in milkers, 1204
solitary in bronchogenic carcinoma, 416
thyroid, 318
with hyperthyroidism, 318
without hyperthyroidism, 321
- Noma, 256
- Norepinephrine, 1461
function, 821
in shock treatment, technique, 94
- Norkutin, in menstrual disturbances, 950
in threatened abortion, 942
- Nose, 224-241
bleeding, 226
fracture, 227-279
fungus, 227
hematoma, 226
mucous polyp, 237
surgical removal, 237
obstruction, 225
in paranasal sinus infection, 228
physiology, 224
septum, abscess, 226
deviation, 225
submucous resection in, 225
sinuses, accessory. See *Sinuses*.
- paranasal tumors, 237
benign, 237
malignant, 238
- Notes, operative, 111
progress, 111
- Novobiocin, toxic reactions, 56
- Novocain in fracture treatment, 984
- Nuclear radiation injuries, 208-212
classification, 208
- Nucleus pulposus, herniations, 1444
- Nupercal, 171
- Nusbaum, 5
- Nutrition, following peptic ulcer surgery, 661
in burns, 191
management, preoperative, 97
in obese patient, 102
oral preparation, 97
parenteral preparation, 100
postoperative requirements, 111
wound healing and, 37
- Nystatin in dermatophytosis, 1295
- Oesophagus as factor in hernia, 521
preoperative nutritional management in, 102
- Obstetric and gynecologic surgery
antibiotic prophylaxis in, 60
- Obstruction, arterial, organic, 1351
duodenal, surgical treatment, 578
gastrointestinal, preoperative preparation, 103
intestinal. See *Intestine obstruction*.
- lymph. See *Lymphedema*.
- mechanical. See *Intestine obstruction*.
- peptic ulcer and, 647
pyloric, in peptic ulcer medical treatment, 623
surgical treatment, 624
salivary system, 285
small bowel, in peritonitis, 493
stomach, in peptic ulcer, 620
urinary tract. See *Urinary tract obstruction*.
- Obturator hernia, 568
- Occipital lobe tumors, 1424
- Occlusion, arterial, acute, prophylactic sympathectomy in, 1465
coronary, 460
of radial artery Allen's test in, 1346
of ulnar artery Allen's test in, 1346
vascular intestinal obstruction in, 754
venous, massive, treatment, 1340
- Occupational therapy, definition, 1298
in orthopedic disorders, 1317
in surgical conditions, 1325
- Ocular proptosis, 219
- Oddi, sphincter of, section of, in relapsing pancreatitis, 807
- Odonoid process, fractures, 1053
- Odontomas, 283
- Ointment, Sebreech's, for skin protection in duodenal fistula, 684
- Oleandomycin, in osteomyelitis, acute hematogenous, 1118
toxic reactions, 56
- Olecranon process, fractures, 1025
- Oleoma formation, 1220
- Oligodendroglioma, 1423
- Oliguria in urinary tract disease, 856
- Omentum, 513
anatomy, 513
cysts, 514
congenital, 517
greater, 513
embryology, 485
infarction, 515
infarction, 515
lower embryology, 485
lymph vessels, 1369
physiology, 513
torsion, 515
primary, 515
secondary, 515
- Omphalitis, in adult, 479
in newborn, 479
- Omphalocele. See *Hernia, umbilical*.
- Omphalomesenteric duct, 472
anomalies, 477
persistent, 583. See also *Diverticulum, Meckel*.
- Omphalomesenteric vessels, anomalies, 479
- Oophorectomy in breast carcinoma, 363-364
- Oophorectomy unilateral, in dysgerminoma, 966
in granulosa cell tumor, 968
- Operating room, fatalities due to anesthesia, 176
- Operation, care of patient during, 106
contamination, peritoneal and, 489
preoperative discussion with patient, 106
- Operative care, 96-123
- Operative fields, "clean," surgical procedures in, 60
contaminated, surgical procedures in, 60
- Operative notes, 111
- Ophthalmia, sympathetic, 222
- Opiates, shock and, 92
- Optic nerves, injury in craniocerebral trauma, 1403
- Optic neuritis as complication of sinusitis, 229
- Orbit, abscess, as complication of sinusitis, 229
surgical treatment, 231
cellulitis, as complication of sinusitis, 229
fractures, 19
wounds, 219
- Orchectomy in cryptorchidism, 900
in male pseudohermaphrodite, 919
in testicular tumors, 902
in torsion of spermatic cord, 907
- Orchiopexy in cryptorchidism, 900
- Orchitis, bacterial, 900
mumps, 900
vs. acute epididymitis, 900
- Order sheet, postoperative, 110
- Organs, intraperitoneal, 486
- Oribasius, 8, 13
- Oropharynx, anatomy, 237
- Orthopedic disorders, cold in, 1317
educational therapy in, 1317
heat in, 1317
ion transfer in, 1317
manual arts therapy in, 1317
massage in, 1317
occupational therapy in, 1317
therapeutic exercise in, 1317
- Oscillometric tests in peripheral vascular disorders, 1322
- Osmolality urine and plasma, postoperative, 126
- Osteitis deformans of skull, 216
- Osteitis fibrosa cystica generalisata of von Recklinghausen, 324
diffuse, 1161
polycystic, 1160
- Osteoarthritis, hypertrophic, 1446
loose bodies in knee joint and, 1092
of foot, 1291-1292
- Osteocartilaginous bodies, loose, in knee joint, 1089-1092
- Osteochondritis, dissecans, loose bodies in knee joint and, 1092
of foot, 1290
- Osteochondroma(s) 1149
hereditary, 1151
multiple, 1151
of chest wall, 396
of skull, 216
- Osteochondromatosis, loose bodies in knee joint due to, 1093
- Osteofibroma of jaw, 283
- Osteogenesis imperfecta, definition, 980

- Peptic ulcer antrectomy in**, 638
 chronic acute erosion and relationship, 636
 location, 636
 pathology, 613
 coffee restriction in, 644
 complicated, medical treatment, 643
 complications, 618
 of previous surgery surgical treatment, 625
 constitutional factors in, 614
 course, 626
 diagnosis, 617
 diet in, 644
 differential diagnosis, 618
 endoscopic examination in, 617
 eroding factors in, 613
 esophageal, 638
 surgical treatment, 638
 etiology, 613-634
 gastric resection in, 638
 gastroduodenal hemorrhage in, 650
 hemorrhage, 619
 differential diagnosis, 620
 incidence, 607
 medical treatment, 643
 surgical treatment, 644
 incidence, 606
 intractability surgical treatment, 644
 islets of Langerhans tumors and, 818
 laboratory examination in, 617
 local defense factors in, 614
 local tissue resistance in, 614
 location, 606
 symptoms related to, 617
 obstruction and, 647
 pain in, 615
 pathogenesis, 615
 pathology, 615
 perforation, 619
 free, 648
 primary gastric resection in, 649
 simple closure, 649
 treatment, 648
 in pneumoperitoneum, 510
 incidence, 607
 medical treatment, 623
 peritonitis in, 489
 treatment, 494
 recurrent acute, incidence, 607
 surgical treatment, 644
 walled-off, 619
 medical treatment, 623
 physical examination in, 617
 postsurgical management, 625
 production, experimental, 633
 prognosis, 626
 psychotherapy in, 622
 pyloric obstruction in, medical treatment, 623
 surgical treatment, 624
 recurrence, 626
 surgical treatment, 625
 rest in, 622
 roentgenography in, 618
 sedation in, 644
 signs, 615
 surgery, 624-631-663
 choice of operation, 625
 dumping syndrome following, 660
 mortality, 659
- Peptic ulcer surgery nutrition following**, 661
 recurrent ulcer following, 660
 results, 625
 nutritional, 659
 selection of patient in, 637
 selection of procedure in, 638
 suspected malignancy in, surgical treatment, 625
 symptoms, 615
 systemic factors in, 614
 tobacco restriction in, 622
 treatment, 671
 philosophy, 644
 results, 646
 uncomplicated, medical treatment, 643
 vagotomy in, 638-641
 vs. gastric carcinoma, 617
- Perforation in diverticulosis**, 705
Periapical abscess, 281
Periapical abscess. See *Abscess periapical*
Pericardial cysts, 447
Pericardial effusion, acute serous, 447
 fibrous, 447
 in bronchogenic carcinoma, 420
Pericardial poudrage in coronary artery disease, 461
Pericardectomy in pericarditis, 449
Pericarditis, acute idiopathic, 449
 chronic, 448
 pericardectomy in, 449
 purulent, treatment, 449
 suppurative, 449
 tuberculous, 449
Pericardium, decompression, in cardiac tamponade, 447
 diseases, 447
 classification, 447
 diverticulum, 447
Pericoronitis, 281
 acute, 255
Peridental membrane disappearance in hyperparathyroidism, 325
Peridural anesthesia, 174
 disadvantages, 174
Peridural block, 169
Perineum, burns, 197
 hemias, 568
Perineural fibrosis, 1459
Periostitis, definition, 1111
Peripheral nerves. See *Nerves peripheral*
Peristalsis, gastric, 613
 increase in intestinal obstruction, 735
 sounds, increase in intestinal obstruction, 739
Peritoneal cavity bile in, 499
 blood in, 499
 foreign bodies in, 499
 gastric juice in, 499
 masses, vs. abdominal wall masses, 481
 pancreatic juice in, 499
 third space, 491
 urine in, 499
Peritoneal fluid, 486
Peritoneal sac, greater, 486
 lesser, 486
Peritoneum, 484-517
 adhesions, 505
 anatomy, 486
 gross, 486
 microscopic, 486
- Peritoneum, area of**, 486
 surface, 488
 attachments to spleen, 842
 bands, 505
 embryology, 484
 extracellular fluid compartment and, relation, 488
 granulomatous lesions, 499
 inflammation, 488. See also *Peritonitis*
 innervation, sensory, 488
 parietal, 486
 sensibility, 488
 physiology, 488
 tumors, 512
 primary, 512
 secondary, 512
 visceral, 486
 sensibility, 488
- Peritonitis, 488**
 acute, 488
 aseptic, 488-498
 bacterial, acute, 489
 due to hematogenous spread, 498
 paralytic ileus in, 753
 chronic, 488
 septic, 493
 clinical manifestations, 491
 complications, 491
 diagnosis, 492, 493
 differential diagnosis, 493
 drainage prophylactic, 493
 etiology, 489
 fatal, 499
 gastrointestinal decompression in, 496
 generalized, in acute appendicitis, treatment, 494-696, 697
 pathology, 490
 historical, 489-491
 idiopathic, 489-497
 vs. secondary peritonitis, 497
 in abdominal wall rigidity, 480
 in appendicitis, 695
 in peptic ulcer perforation, 649
 treatment, 650
 incidence, 489
 laboratory aids, 492
 localization, 495
 localized, in acute appendicitis, treatment, 494
 pathology, 490
 meconium, 499
 observation of patient in, continued, 496
 paralytic ileus and, 490
 pathogenesis, 489
 pathology, 490
 pathophysiology, 490
 physical findings, 492
 primary, 489-497
 vs. secondary peritonitis, 497
 prognosis, 493
 prolonged postoperative ileus and, 491
 prophylaxis, 493
 puerperal, 958
 secondary, 489
 vs. primary peritonitis, 497
 secondary to contamination, 489
 septic, 488
 shock in, 491
 surgical treatment, contraindications, 494
 indications, 494
 symptoms, 491

- Pancreas, fistulae.** See *Fistula, pancreatic*
- head, carcinoma, 811
- heterotopic, 803
- hormone secretion by 802
- inflammation, 804
- injuries, 803
- treatment, 510
- islet alpha cell tumor of, hyperfunctioning, gastrojejunal ulcer and, 654
- islet cell adenoma in peptic ulceration, 614
- lymph vessels, 1368
- operations, in relapsing pancreatitis, 808
- physiology 801
- pseudocyst, 808
- manipulation, 810
- sarcoma, 811
- secretion, decrease, operations for 808
- tail, carcinoma, 816
- trauma, 803 811
- tumor aberrant, 665
- benign, 811
- ulcerogenic, in peptic ulcer 614
- Pancreatotomy partial, in islets of Langerhans tumors, 817**
- radical, in islets of Langerhans tumors, 818
- Pancreatic juice, 801**
- drainage into duodenum, in relapsing pancreatitis, operation to improve, 807
- intrapertitoneal, 499
- sodium, potassium and chloride losses in, 129
- Pancreatic phase of gastric acid secretion, 612**
- Pancreatopancreostomy in obstructive jaundice, 813**
- Pancreatolith, acute, 804**
- types, 804
- edematous, acute, 805
- treatment, 805
- hemorrhage, acute, 806
- recurrent See *Pancreatitis elapsus*
- relapsing, 806
- operations on biliary tract in, 807
- operations on pancreas in, 808
- pain in, operation to relieve, 808
- pancreatic juice drainage into duodenum in, operation to improve, 807
- symptoms, 806
- treatment, 807
- vs. perforated peptic ulcer 619
- Pancreatoduodenectomy radical, in obstructive jaundice, 814**
- Pancreozymin, 802**
- Panendoscopy in neurogenic bladder 813**
- Panhematopenia, splenic, primary 848, 849**
- Pannus, 1129**
- Panophthalmitis, 223**
- Papavicolau technique in cervical cancer 936**
- Papavarine in thromboembolism, 1342**
- Papillae, circumvallata, hypertrophied, 270**
- Papillary cystadenoma lymphomatous of salivary glands, 287 288**
- Papillary cystophorous hyperplasia of breast, 344**
- Papillomas of abdominal wall, 482**
- of biliary tract, 784
- of bladder 879
- of breast, intraductal, 345
- of choroid plexus, 1424
- of hand, squamous, 1231
- of lips and mouth, 263
- Papillomatous, ductal, 342**
- Papyrus Ebers, 2, 1487**
- Papyrus, Edwin Smith, 2**
- Para-aminoalkyl acid and streptomycin in pulmonary tuberculosis, 405**
- Paracekma, 1193**
- Paracentesis in serous otitis media, 244**
- Paraffin baths, 1305**
- Paraffinoma of breast, 337**
- Parapanglioma of carotid body See Carotid body tumors**
- Paraldehyde, 167**
- Paralysis. See also Palsy**
- abductor bilateral, 311
- foot deformities in, 1284
- in head injuries, 1401
- median nerve, 1453
- nerve in bronchogenic carcinoma, 420
- peroneal nerve, 1456
- pseudobulbar in head injuries, 1402
- radial nerve, 1452
- recurrent, bilateral, 311
- sciatic nerve, 1456
- spastic of lower extremity foot deformities in, 1289
- tibial nerve, 1456
- ulnar nerve, 1454
- tardy 1457
- Paranasal sinuses. See Sinus, paranasal**
- Paraparesis in cauda equina injuries, 1439**
- Paraphimosis, 893**
- Paraplegia in cauda equina injuries, 1439**
- Parasite-drug relationships, 55**
- Parasitic diseases, small intestine and, 688**
- Parasternal hernia, 562**
- Parathyroid glands, 317-325**
- in metabolism, 323
- tumor removal, 325
- Paraurthral crypts, infection, 927**
- Paré, Ambroise, 4 9 15 16, 1487 1488**
- Parietal cells, 608**
- physiology 631
- Parietal lobe tumors, 1424**
- Parker 700**
- Paronychia, 1168**
- of toe 1293
- Parotid gland, inflammation, postoperative, 120**
- tumors, benign, removal, 287
- malignant, 288, 289
- Parotid space, 303**
- infection, 303
- Parotitis, postoperative 120**
- PAS and streptomycin in pulmonary tuberculosis, 405**
- Pasteur 4, 1488**
- Pasteurella tularensis infection, 46**
- Patella, chondromalacia, 1089**
- dislocations, 1097
- fractures, 1071
- Patellectomy primary in patellar fractures, 1073**
- Patient, postoperative status, incisional hernia and, 554**
- preoperative discussion of operation with, 106
- preoperative status, incisional hernia and, 554
- preparation, shock prevention and 92
- Paul, 700**
- Paul of Aegina, 8, 13**
- Pavex boot in peripheral vascular disease, 1323**
- Pavlov 632**
- Pavy procedure in peptic ulcer 644**
- Peau, Jules, 9**
- Pectus excavatum, 388**
- Pedicle flaps in wound closure, 85**
- Peel formation in hemothorax, 386**
- Pelvic-lumbar traction, 1315**
- Pelvis, abscess. See Abscess, pelvic**
- diaphragm, hernia, 567
- fascia, anatomy 952
- female, pain in, 929
- fractures, 1056
- first aid 983
- hernias, 167
- infections, 956
- vs. appendicitis, 695
- inflammation, peritonitis and, 489
- lymphatic drainage, 1366
- masses, 953
- differentiation 953 955
- in chronic gonorrheal salpingitis, 958
- pain in, in chronic gonorrheal salpingitis, 958
- relaxations, 951
- ring, fractures, 1056
- rim, fractures, 1056, 1058
- viscera, lymph vessels of 1364
- Penetration wounds of neck, 307**
- Pencellon, in acute gonorrhea in female, 957**
- in gonorrhea, 927
- in hand infections, acute spreading, 1198
- in meningitis prophylaxis, 141
- in surgical infections, 56
- in traumatic wounds, 59
- resistance of organisms to, 53
- Penicillin G 56**
- toxic reactions, 56
- Penis, anatomy 891**
- anomalies, 892
- carcinoma, 895
- diseases, idiopathic, 895
- dislocation, 894
- examination in urinary tract disease 857
- infections, 894
- neoplasms, 895
- phallic induration, 895
- trauma, 893
- Pentothal, 168**
- Peptic formation, 609**
- Peptidogen conversion, 609**
- Peptic ulcer 606-627**
- acute, 635
- pathology 615
- alcohol restriction in, 622
- antacids in, 622
- anticholinergics in, 622

- Polyp(s)** adenomatous, cancer and, relation, 706
 diagnosis, 707
 of colon, 706
 classification, 706
 operation, 707
 symptoms, 707
 gastric 664
 incidence, 663
 malignant degeneration, 664
 relation to gastric carcinoma, 669
 mucosa, of nasal cavity 237
 surgical removal, 237
 of colon, 706
 of rectum, 706
 of small intestine, 690
 pulmonary inflammatory 423
Polypectomy and colectomy in adenomatous polyps, 707
Polyps, multiple, of colon, 706, 708
 scattered, of colon, 706
Polyps en masse, 708
Postcane, 171
Port-wine stain, of hand, 1242
 treatment, 1243
 of hps, 265
Portacaval shunt, in portal hypertension, 792, 793
 surgical procedure, 794
Portal hypertension. See *Hypertension, portal*.
Position, of function in hand infections, 1184
 of patient in shock treatment, 93
 of postoperative patient, 107
 proper in amputations, 1320
 in upper motor neuron disorders, 1317
Postabortion infections, 958
Postanesthetic care, 107 110
Posthile, 894
Postoperative care 96-123
 locomotive, unit, 110
 principles, 96-137
Postoperative order sheet, 110
Postoperative patient, physiologic characteristics, 123
 rectus sheath hematoma and, 481
 status, incisional hernia and, 554
Postphlebotic syndrome, prognosis, 1337
 treatment, 1341
Posture, bed, of thoracic surgical patient, 1234
Potassium, balance in anabolic phase of convalescence, 145
 in postoperative patient, 124
 deficiency metabolic alkalosis due to, 130
 in gastrointestinal discharges, estimation, 147
 losses, in bile, 129
 in convalescence, 148
 in gastrointestinal fluids, 128, 129
 in pancreatic juice, 129
 metabolism, post-traumatic, 141
Potency in prostatic, 911
Pott, Percival, 16, 1140
Pott's disease. See *Tuberculosis of spine*.
Pouch, hepatorenal, of Morrison, 502
Poodrags, pericardial, in coronary artery disease, 461
Proctocyt sexual, 919
Predhione in adrenocortical suppression, 838
Pregnancy abdominal, 946
 breast carcinoma in, treatment, 362
 ectopic, 944
 atypical type 945
 causes, 944
 chemical type, 945
 clinical picture 945
 diagnosis, 945
 hemorrhage in, 944
 in hemoperitoneum, 510
 incidence 945
 mortality rate 947
 rupture 945
 symptoms following, 946
 symptoms, 945
 treatment, 947
 vs. acute gonorrheal salpingitis, 946
 extruterine. See *Pregnancy ectopic*.
 fibroids in, 943
 gallstones and, 765
 kidney infection in, 868
 ovarian, 946
Premam in amenorrhea, 950
Premedication in anesthetics, 177
Preoperative care 96-123
 emergency 97
 final steps, 105
 principles, 96-137
 Preoperative order sheet, 105
 Preoperative patient, status, incisional hernia and, 554
 Preoperative routines, 97
 Preparation of patients, shock prevention and, 92
 Prepuce inflammation, 894
 Presbycusis, 248
 Presplenic fold, 843
 Pressure, intra-abdominal, increased, as factor in hernia, 521
 Intracranial, increased, in head injuries, pathology 1394
 in intracranial injuries, 1393
 necrosis, prevention during operation, 106
 Pretracheal fascia, 301
 Prevertebral fascia, 302
 Priapism, 896
 Procaine, 170
 Procaine hydrochloride, history 12
 Process, odontoid, fractures, 1053
 olecranon, fractures, 1025
 Proctectomy in chronic ulcerative colitis, 703
 Proctoscopy in rectal carcinoma, 730
 indications, 717
 Proetz technique for displacing ethmoid sinus secretions, 230
 Progesterone, in endometriosis, 964
 in menstrual disturbances, 950
 in threatened abortion, 942
 Prognathism in cleft lip and palate, repair of, 262
 Progress notes, 111
 Proptosis, classification, 220
 ocular 219
Prostate, 908
 abscess, 910
 anatomy 908
 anomalies, 909
 calculus, 915
 carcinoma. See *Carcinoma of prostate*.
Prostate enlargement, 911
 examination in urinary tract disease 858
 hyperplasia, benign, 912
 infections, 909
 nonspecific, 909
 lymph vessels, 1369
 obstruction, 911
 pain, in urinary tract disease, 855
 transurethral resection, 913
 trauma to, 909
 tumors, secondary 915
Prostatectomy conservative, 912
 methods, 914
 perineal, 914
 radical, in carcinoma of prostate, 914 915
 retropubic, 914
 suprapubic, 914
Prostatitis, 911
 carcinoma of prostate and, 915
 obstructive symptoms, 911
 sexual symptoms, 911
 superimposed infection in, symptoms, 911
 uremic symptoms, 911
Prostatitis, acute, 910
 chronic, 910
 tuberculous, 910
Prostatotomy in prostatic abscess, 910
Prostatovesiculectomy in prostatic carcinoma, 914
Prosthesis, arm function and, 1180
 ball-valve, in aortic insufficiency 459
 for coronary leaflet deformities in aortic insufficiency 459
 in amputations of extremities, 1321
 training in use of 1371
 in aortic arch aneurysms, 463
 in incisional hernia repair 560
 intraosseous stem, in femoral neck fracture, 1064
 leg function and, 1180
Protein, deficiency preoperative management, 101 102
 wound healing and, 38
 hydrolytates in malnourishment, 101
 in burn diet, 191
 metabolism, liver and, 762
 post-traumatic, 141
 serum, metabolism, post-traumatic, 143
 translocation in peritonitis, 491
Protein-bound iodine in hyperthyroidism, 319
Proteinases, production of, 34
Proteus infections, 46
Prothrombin formation by liver 763
Protozoa infection, 46
Pruritus ani, 727
Pseudocyst of pancreas, 808
 marsupialization, 810
Pseudohermaphrodite, female, 918
 male, 919
Pseudomembrane formation in infections of pharynx and tonsils, 233 234
Pseudomonas infection, 46
Pseudomyoma peritonei, 513 698
Pseudopolyps of colon, 708
Pseudotumor 1429
PSP test in urinary tract disease, 859

- Peritonitis, terminal phase, 492
treatment, 493
 definitive, 494
 prophylactic, 493
 supportive, 496
tuberculosis, 498, 959
 dry form, 498
 moist form, 498
 types, 498
- Peritonsillar abscess, 234
- Peroneal nerve, injuries, 1456
- Peroneus brevis, paralysis, foot de-
formities in, 1287
- Peroneus longus, paralysis, foot de-
formities in, 1287
- Personality factors in peptic ulcer
615
- Pertussis test in varicosis, 1331
- Pes cavus, 1277
- Pes planus, 1272
- Petit, 9
- Peck, inferior lumbar triangle anat-
omy 553
- Petrissage, 1309
- Peyronie's disease, 895
 Dupuytren's contracture and,
 1226
- pH, disturbances, 129
 in healing of fractures, 992, 993
 of tissues in wound healing, 24
- Phagocytes in inflammation, 47
- Phalangitis, drop, 1218
- Phalanx (phalanges) distal, frac-
tures, 997
 middle, fractures, 997
 of toes, fractures, 1089
 proximal, fractures, 997
- Pharyngeal bursa, 32
- Pharyngitis, bacterial, acute, 233
- Pharyngomaxillary space, 303
 infection, 303
- Pharynx, 232
 abscess, lateral, 234
 anatomy 32
 foreign bodies in, 235
 function, 233
 malignant disease, pain relief in,
 1470
 streptococcal infection, acute, 233
 tumors, 235
- Phenothiazine test in urinary
tract disease, 839
- Phenomenon, Raynaud's 1350, 1463
- Phenochromocytoma, 838
 diagnosis, 839
 hypertension in, 838
 surgical treatment, 839
 symptoms, 839
- Phimosis, 892
- Phlebosclerosis in varicose veins,
1330
- Phlebothrombosis, 1332
 as cause of postoperative fever 122
 clinical picture, 1336
 deep veins, active treatment, 1340
 etiology, 1333
 incidence, 1335
 pathogenesis, 1333
 thrombophlebitis and, distinction,
 1334
 treatment, 1339
- Phlegmasia alba dolens, 1336
- Phlegmon, of submandibular space,
304
 septic, of mouth, 256
 subperiosteal, 390
 V 1193
- Phosphatase, acid, blood, in pros-
tatic carcinoma, 913
 alkaline, serum, level in breast
 carcinoma, 357
- Phosphorus, metabolism, parathyroid
hormone in, 323
 radioactive, in breast lesions, 335
- Photography infrared, in breast le-
sions, 335
- Physiatrist, definition, 1298
- Physic, 17 18
- Physical medicine and rehabilitation,
1297-1326
 definition, 1298
 electrodiagnostic procedures,
 1301
 in amputations, 1320
 objectives, 1320
 procedures, 1320
 in dislocations, 1316
 in fractures, 991 1316
 in lower motor neuron dis-
 orders, objectives, 1320
 procedures, 1320
 in neurologic disorders, 1317
 in orthopedic disorders, ob-
 jectives, 1316
 procedures, 1317
 in peripheral vascular dis-
 orders, 1322
 objectives, 1322
 procedures, 1322
 in sprains, 1316
 in strains, 1316
 in surgical conditions, 1324
 objectives, 1324
 procedures, 1324
 in thoracic surgical condi-
 tions, 1323
 objectives, 1323
 procedures, 1323
 in upper motor neuron disor-
 ders, objectives, 1317
 procedures, 1317
 objectives, fundamental, 1299
 principles, basic and general,
 1299
 scope, 1298
 stages, 1298
- Physiology of wound healing, 22-41
- Phytoheizer 627 671
- Pigmentation of new epithelium, 30
- Pillows, 700
- Pilonidal cyst. See *Cyst pilonidal*.
- Pm, fixation, of metatarsal fractures,
1088
 Stammann, in calcaneal fractures,
 1085
 in rib fractures, 1049
 in ulnar shaft fractures, 1022
- Pinealoma, 1425
- Piperocaine, 171
- Pirogoff, 11
- Pituitary basophilism. See *Cushing's
syndrome*.
- Pituitary body adenomas, 1425
 adrenal relations and, in Cush-
 ing's syndrome, 822
 diseases, postoperative, 154
 tumors, 1425
- Pituitary infundibulum, 919
- Plantar aponeurosis, 1261
- Plantar callosities, 1282
- Plantar flexor muscles of foot, paral-
ysis, 1288
- Plantar ligament, long, 1261
- Plantar warts, 1282
- Plasma, bicarbonate, changes in, 130
 blood, composition, 127
 loss in intestinal obstruction, 737
 osmolality postoperative, 126
- Plasma cell mastitis, 338
- Plate fixation in tibial shaft fractures,
1075
- Platybasia, 15
- Plant Vincent's angina, 234
- Plethysmography in peripheral vascu-
lar disorders, 1322
- Pleura, 382-398
 empyema, acute, 391
 chronic, 395
 inflammations, 390, 391
 lymphatic vessels, 1371
 tumors, 396
 metastatic, 397
 primary, 397
- Pleural effusion in bronchogenic car-
cinoma, 420
- Pleuroperitoneal hernia, 562
- Pleura, brachial, block, 175
 injuries, 1456
 choroid, papillomas, 1424
- Pliny 10
- Plummer Vinson syndrome, 600
- Pneumatocele, 409
- Pneumocephalus in head injuries,
1407
- Pneumectomy in bronchiectasis,
412
 in bronchogenic carcinoma, 421
- Pneumonia, in bronchogenic carci-
noma, 417
 Friedlander's, 406
 staphylococcal, 408
- Pneumonia, suppurative, chronic,
407
 vs. bronchogenic carcinoma,
 408
- Pneumoperitoneum, 510
 artificial, 512
 causes, classification, 510
 diagnosis, 511
 diagnostic, 512
 endoscopic instrumentation and,
 511
 idiopathic, 512
 in pulmonary tuberculosis, 402
 infections and, 511
 postanesthetic, 511
 postoperative, 511
 therapeutic, 512
 traumatic, 511
- Pneumothorax, artificial, in pulmo-
nary tuberculosis, 402
 closed, 388
 diagnostic, in empyema, 392
 in rib fracture, 1050
 spontaneous, 410
 tension, 384
 wounds, open or sucking, 384
- Polio-myelitis, foot deformities fol-
lowing, 1284
 susceptibility following tonsillec-
 tomy and adenoidectomy 236
- Polya gastric resection in peptic ulcer
625
- Polya gastrectomy 639 640,
641
- Polya operation for gastric cancer
671
- Polydactylism, in foot, 1271
 in hand, 1251
- Polymyx, toxic reactions, 56
- Polymyx B in surgical infections, 58

- Rectocele symptoms, 912
treatment, 91
vs. enterocele, 468
- Recto-fossa navicularis fistula with imperforate anus, 491
- Rectoperineal fistula with imperforate anus, 491
- Rectosigmoid, anatomy, 715
- Rectourethral fistula with imperforate anus, 491
- Rectovaginal fistula with imperforate anus, 491
- Rectovesical fistula with imperforate anus, 491
- Rectum, 714-731 See also *Anorectum*
anatomy, 715
gross, 715
cancer. See *Carcinoma of rectum*
examination in urinary tract disease, 818
injuries in pelvic ring fractures, 1046
lymph vessels, 1368
malformations. See *Anorectum malformations*
prolapse, 725
acute reduction, 777
classification, 725
diagnosis, 725
etiology, 725
partial, vs. hemorrhoidal prolapse, 725
recurrent, reduction, 726
treatment, 776
stricture, benign, 725
types, 725
tumors, 706 See also *Colon tumors*
- Rectus sheath, 475
hematoma, 481
etiology, classification, 481
idiopathic, 481
treatment, 487
- Red degeneration of subcutaneous fibroids, 943
- Redox cycle in hydrochloric acid secretion, 609
- Reduction, of fractures, 991
of open fractures, 987
- Reflex, bulbocavernosus, in urinary tract disease, 858
tubal, theory, 963
- Refrigeration anesthesia, 175
local, in amputations, 1347
- Regurgitation, aortic, 457 See also *Aorta, insufficiency*
- Rehabilitation. See also *Physical medicine and rehabilitation*
assistive devices in, 1309
definitive, 1798
in spinal injuries, 1440
maintenance, 1298
of blind, therapy, deflation, 1298
preventive, 1298
self-help devices in, 1309
- Relatives of patient, discussion of operation with, 106
- Relaxants, muscle, 169
- Relaxations, pelvic, 951
- Remanence surgery, 15
- Renal glands, weight, 820
- Reproductive organs, male, 890
- Reproductive symptoms of ovarian failure, 948
- Reproductive system, female, 921
977
- Reproductive system, female diseases,
bleeding in, 933
pain in, 979
male, 890-970
physiology, 890
- Resection, segmental, in bronchies
laws, 41
subcutaneous, in deviation of nasal septum, 225
- Resistance, bacterial, to antibiotics, 43
nonspecific to infection, natural mechanism of, 43
skin, electrical measurements in neuromuscular disorders, 1304
- Respiration(s), artificial, Sylvester method, 179
mouth-to-mouth breathing in, 179
Nissen method, 179
awaked, 167
Cheyne-Stokes, in head injuries, 1198
function, 400
in head injury, 1397
- Respiratory acidosis, 132
- Respiratory diaphragm, hernia. See *Hernia diaphragmatic*
- Respiratory system complications of anuria, 229
- Respiratory tract in burns, pathologic changes in, 186
- Rest effect on wound healing, 39
of part in hand infections, 1183
position of function, 1184
- Resuscitation, cardiac. See *Heart arrest*
in anesthesia, 179
ventilatory, 179
- Reticulocyte count in radiation injuries, 211
- Reticuloendothelial storage diseases, splenomegaly in, 852
- Reticuloendothelial system, spleen and, 844
- Reticuloendothelium, 216
- Reticulum cell sarcoma, 1385
of retroperitoneal space, 516
of stomach, 666
- Retina, injuries to, 222
- Retrolabial neuritis as complication of anuria, 229
- Retrorotax space, 1192-1193
- Retroperitoneal space, 515
anatomy, 515
hemorrhage into, 516
tumors, 516
primary, 516
- Retropharyngeal abscess, 234-303
- Retropharyngeal space, 303
infection, 303
- Reyherd, 700
- Rhabdomyoma, 1104
cardiac, 466
- Rhabdomyosarcoma, cardiac, 466
of abdominal wall, 483
of esophagus, 601
of soft parts of extremities, 1174
- Rheobase, in neuromuscular disorders, 1302
ratio in neuromuscular disorders, 1302
- Rheumatic aortic valvulitis, 455
- Rheumatic fever vs. acute hematogenous osteomyelitis, 1117
vs. pyarthrosis, 1133
- Rheumatism, muscular, 1103
- Rheumatoid arthritis, of foot, 1791
therapy pyarthrosis and, 1130-1137
vs. pyarthrosis, 1133
- Rhinith, hyperplastic chronic, 226
- Rhiphorhea, cerebrospinal fluid, 1407
- Rhizotomy in trigeminal neuralgia, 1447
posterior, 1470
- Rib cervical, 389-1458
fractures, 1048
simple, 385
osteomyelitis, acute, 390
typical, 390
- Rice bodies in tuberculous tenosynovitis, 1700
- Richter's hernia, 568
deflation, 570
strangulated intestinal obstruction in, 746
- Riedel's struma, 370
- Rigidity abdominal wall causes, classification, 480
decerbrate in head injuries, 1404
mucal, in craniocerebral injuries, 1401
- Ring(s), vascular, 435
symptoms, 436
treatment, 437
Walden's, 232
- Rinne hearing test, 47
- Ritocetin, toxic reactions, 56
- "Rock" aortic, 457
- Rod, intramedullary. See *Intramedullary rod*
- Rodent ulcer, 267
- Röntgenotherapy. See also *Radium and Radiation*
palliative in breast carcinoma, 363
postoperative and radical mastectomy in breast carcinoma, 358
and simple mastectomy in breast carcinoma, 358
preoperative in breast carcinoma, 362
- Root cysts, 283
- Rubin test pneumoperitoneum and, 512
- Rule of nines in burn estimation, 184-185
- Rumpel-Leede phenomenon in primary thrombocytopenic purpura, 847
- Run-around, 1188
- Ruptures of soft tissues, 80
- Rush intramedullary rod in femoral shaft fractures, 1069-1071
- Rush rod, in tibial shaft fractures, 1076
in ulnar shaft fractures, 1022
- Russell traction, in acetabular fractures, 1058
in intertrochanteric fractures, 1068
- Rynd, Francis, 12
- SAC, peroneal, greater, 486
lower, 486
- Sacral anesthesia, 174
- Sacroiliac joint, tuberculous, 1143
- Sacrum, fractures, 1044-1046

- Psychic phase of gastric acid secretion, 610
- Psychogenic symptoms of ovarian failure, 948
- Psychosomatic symptoms of ovarian failure 948
- Psychotherapy 1436
- results, 1436
- Psychotherapy in peptic ulcer 622
- Puberty precocious, in granulosa cell tumor 967
- Pudendal hernia, 568
- Puerperium, infections in, 958
- Pulley portable, apparatus, 1308
- Pulmonary. See also Lungs.
- Pulmonary artery anatomy 399
- Pulmonary diffusion, tests of 383
- Pulmonary stenosis. See *Pulmonic stenosis*.
- Pulmonary sulcus tumor 397
- Pulmonary tuberculosis. See *Tuberculosis pulmonary*.
- Pulmonary vein, anatomy 400
- Pulmonic stenosis, 437
- cardiac x-ray in, 440
- diagnosis, 438
- in tetralogy of Fallot, 439
- infundibular 437
- surgical treatment, 439
- isolated, 437
- surgical treatment, 438
- patent foramen ovale with, 437
- pulmonary arterial, 437
- pure, 437
- symptoms, 438
- treatment, 438
- types, 437
- valvular type, 437
- operative treatment, 438
- Pulse rate in head injury 1397
- Pulson diverticulum, 235
- Pump, finger type, 428
- roller type, 428
- vesticule type, 428
- Pump oxygenator in cardiac surgery 427
- types, 428
- Puncture, fracture, deflation, 979
- lumbar. See *Lumbar puncture*
- spinal. See *Lumbar puncture*
- wounds of neck, 307
- Pupils, inequality in craniocerebral injuries, 1401
- Purpura, hemorrhagic vs. primary thrombocytopenic purpura, 847
- thrombocytopenic, primary 846, 847
- hematologic findings, 847
- types, 847
- Pw, "handable," 4 1488
- Pustules, (acne), 355
- Pyarthrosis, 1127
- by direct contamination, 1128, 1137
- by direct extension, 1128, 1137
- diagnosis, 1131
- differential diagnosis, 1137
- etiology 1128
- hematopneumia, 1127
- of foot, 1294
- rheumatoid arthritis therapy and, 1130, 1137
- sympoms, 1129
- treatment, 1134
- vs. acute hematogenous osteomyelitis, 1117
- Pyelitis vs. appendicitis, 694
- Pyelography intravenous, in urinary calculous disease 874
- Pyelonephritis, acute, 866
- treatment, 867
- chronic, 868
- in urinary calculous disease, 874
- vs. acute prostatitis, 910
- Pyeloplebitis, in appendicitis, 696
- Intestinal obstruction in, 754
- Pyloric glands, physiology 632
- Pyloroplasty Hemeke-Mikulicz, 642, 643
- Ramstedt-Fredet, in hypertrophic pyloric stenosis, 679
- with vagotomy in peptic ulcer 642
- Pylorospasm in congenital hypertrophic pyloric stenosis, 576
- Pylorus, sphincter function, 613
- stenosis, hypertrophic 629
- acquired, 629
- adult, 629
- congenital, 575
- postoperative feeding in, 577
- surgical repair 576
- symptoms, 575
- infantile 629
- Pyogenic infections of breast, 337
- Pyosalpinx, 957
- Pyothorax, 391
- QUECKENSTEDT test, in intersegmental tumors, 1443
- in spinal injuries, 1440
- de Quervain's disease 1228
- Quinzani dye test in sweating pattern alterations, 1305
- Quincy sore throat, 234
- RACHNICHTIS with spina bifida, 1475
- 1476
- Radial nerve, injuries, 1452
- Radiant heat generators, 1306
- Radiation. See also Radium and Roentgenotherapy.
- as factor in carcinoma of hand, 1232
- as factor in osteomyelitis of jaw 281
- carcinoma, treatment, 1233
- dermatitis, 1232
- prevention, 1233
- injuries, 182-211
- acute, 205
- treatment, 206
- causality, classification, 209
- causes, 205
- chronic, 205
- treatment, 207
- clinical course, 206
- internal, 208
- nuclear 206-712
- classification, 208
- pathology 205
- severity according to dose, 209
- treatment, 212
- surface, 208
- whole body 208
- diagnosis, 210
- symptoms, 209
- treatment, 212
- lymph vessel injuries from, 1373
- sickness, 208
- Radiation, syndrome, acute, 208
- therapy in adenocarcinoma of ovary 970
- in bladder tumors, 830
- in carcinoma of tongue, 773
- in carcinoma of vulva, 975
- in cervical cancer 937
- in dysgermatoma, 966
- in endocrine bleeding, 951
- in fibroids, 944
- in gastric carcinoma palliation, 675
- in gastric sarcoma, 666
- in intracranial tumors, 1429
- in renal carcinoma, 881
- in testicular tumors, 907
- radical mastectomy and, in breast carcinoma, 361
- ultraviolet, 1312
- Radicular cysts, 283
- Radioactive isotopes in intracranial tumors, 1427
- Radioactive phosphorus in breast lesions, 335
- Radioresistance in testing prognosis in breast carcinoma, 359
- Radionecrosis, 206
- Radioactivity in testing prognosis in breast carcinoma, 359
- Radix. See also Radon and Roentgenotherapy.
- as factor in carcinoma of hand, 1237
- in carcinoma of corpus uteri, 979
- in cervical cancer 937
- Radix, absence, congenital, 1748
- distal end, fracture, marginal, 1013
- epiphysis, distal, fracture-dislocation, 1014
- fractures, 1016
- comminuted, 1019
- in children, 1017
- oblique, 1019
- transverse in adults, 1019
- in children, 1018
- head, dislocation, with ulnar shaft fracture 1022
- head and neck, fractures, 1074
- shaft, fractures, 1021
- Radon seeds in carcinoma of tongue, 271
- Ramstedt-Fredet pyloroplasty in hypertrophic pyloric stenosis, 629
- Ramstedt operation in congenital hypertrophic pyloric stenosis, 576
- Ramstedt procedure in peptic ulcer 642
- Rankin, 700
- Ramie, 264
- Ratio direct current tetanus, in neuromuscular disorders, 1307
- rheobase in neuromuscular disorders, 1302
- Raynaud, Maurice, 1463
- Raynaud's disease, 1349 1463
- Raynaud's phenomena, 1340, 1463
- Reaction of degeneration, 1501
- Recklinghausen's abdominal wall neurofibromas, 482
- Recklinghausen's disease, 1459
- in hand, 1246
- Recklinghausen's osseous fibroma cystic generalized, 374
- Recovery ward, 107
- Rectocele, 951
- etiology factors, 951

- Rectocele symptoms, 952
treatment, 95
vs. enterocele 968
- Recto-fossa navicularis fistula with imperforate anus, 591
- Rectoperineal fistula with imperforate anus, 591
- Rectosigmoid, anatomy 715
- Rectourethral fistula with imperforate anus, 591
- Rectovaginal fistula with imperforate anus, 591
- Rectovesical fistula with imperforate anus, 591
- Rectum, 714-731 See also *Anorectum*
anatomy 715
gross, 715
cancer See *Carcinoma of rectum*
examination in urinary tract disease 858
injuries in pelvic ring fractures, 1046
lymph vessels, 1368
malformations. See *Anorectum, malformations*
prolapse 725
acute reduction, 727
classification, 725
diagnosis, 725
etiology 725
partial, vs. hemorrhoidal prolapse 725
recurrent, reduction, 726
treatment, 726
structure benign, 725
types, 725
tumors, 706 See also *Colorectal tumors*
- Rectus sheath, 475
hematoma, 481
etiology classification, 481
idiopathic, 481
treatment, 482
- Red degeneration of subcutaneous fibroids, 943
- Redox cycle in hydrochloric acid secretion, 609
- Reduction of fractures, 991
of open fractures, 987
- Reflex, bulbocavernosus, in urinary tract disease, 858
tibial, theory 963
- Refrigeration anesthesia, 175
local, in amputations, 1347
- Regurgitation, aortic, 457 See also *Aortic insufficiency*
- Rehabilitation. See also *Physical medicine and rehabilitation*
assistive devices in, 1309
deformity 1298
in spinal injuries, 1440
maintenance, 1298
of blind, therapy definition, 1298
preventive, 1298
self-help devices in, 1309
- Relatives of patient, discussion of operation with, 106
- Relaxants, muscle 169
- Relaxations, pelvic, 951
- Remanence surgery 15
- Renal glands, weight, 820
- Reproductive organs, male, 890
- Reproductive symptoms of ovarian failure, 948
- Reproductive system, female, 921
977
- Reproductive system, female diseases, bleeding in, 933
pain in, 979
male 890 970
physiology 890
- Resection, segmental in bronchec-tases, 41
subcutaneous, in deviation of nasal septum, 225
- Resistance bacterial, to antibiotics, 53
non-specific to infection natural mechanisms of 43
skin, electrical measurements in neuromuscular disorders, 1304
- Respiration(s) artificial, Sylvester method, 179
mouth-to-mouth breathing in, 179
Neilsen method, 179
assisted, 167
Cheyne Stokes, in head injuries, 1398
function, 400
in head injury 1397
- Respiratory acidosis, 132
- Respiratory diaphragm, hernia. See *Hernia, diaphragmatic*
- Respiratory system complications of sinusitis, 229
- Respiratory tract in burns, pathological changes in, 186
- Rest, effect on wound healing, 39
of part in hand infections, 1183
position of function, 1184
- Resuscitation, cardiac. See *Heart arrest*
in anesthesia 179
ventilatory 179
- Reticulocyte count in radiation injuries, 211
- Reticuloendothelial storage diseases, splenomegaly in, 852
- Reticuloendothelial system, spleen and, 844
- Reticuloendotheliosis, 216
- Reticulum cell sarcoma, 1385
of retroperitoneal space, 516
of stomach, 666
- Retina, injuries to, 222
- Retrolubular neuritis as complication of sinusitis, 229
- Retroflexor space 1192, 1193
- Retroperitoneal space, 515
anatomy 515
hemorrhage into, 516
tumors, 516
primary 516
- Retropharyngeal abscess, 334 303
- Retropharyngeal space 303
infection, 303
- Reynard, 700
- Rhabdomyoma, 1104
cardiac, 466
- Rhabdomyosarcoma, cardiac, 466
of abdominal wall, 483
of esophagus, 601
of soft parts of extremities, 1174
- Rheobase, in neuromuscular disorders, 1302
ratio in neuromuscular disorders, 1307
- Rheumatic aortic valvulitis, 455
- Rheumatic fever vs. acute hematogenous osteomyelitis, 1117
vs. pyarthrosis, 1133
- Rheumatism, muscular 1103
- Rheumatoid arthritis, of foot, 1791
therapy pyarthrosis and, 1130
1137
vs. pyarthrosis, 1133
- Rhinitis, hyperplastic, chronic 226
- Rhinorrhea, cerebrospinal fluid, 1407
- Rhinotomy in trigeminal neuralgia, 1447
posterior 1470
- RRs, cervical, 389 1458
fractures, 1048
simple, 385
osteomyelitis, acute 390
typhoidal, 390
- Rice bodies in tuberculous tenosynovium, 1200
- Richter's hernia, 568
deflation, 520
strangulated, intestinal obstruction in, 756
- Riedel's struma, 370
- Rigidity abdominal wall, causes, classification, 480
decrebrate in head injuries, 1404
nuchal, in cranio-cerebral injuries, 1401
- Ring(s) vascular 435
symptoms, 436
treatment, 437
Waldeyer's, 23
- Rinne hearing test 47
- Rintocetan, toxic reactions, 56
- Rock aortic, 457
- Rod, intramedullary. See *Intramedullary rod*
- Rodent ulcer 67
- Röntgenotherapy. See also *Radium and Radiation*
palpative in breast carcinoma, 363
postoperative and radical mastectomy in breast carcinoma, 358
and simple mastectomy in breast carcinoma, 358
preoperative, in breast carcinoma, 362
- Root cysts, 283
- Rubin test, pneumoperitoneum and, 51
- Rule of nines in burn estimation, 184 185
- Rumpel-Leede phenomenon in primary thrombocytopenic purpura, 847
- "Run-around," 1188
- Ruptures of soft tissues, 80
- Rush intramedullary rod in femoral shaft fractures, 1069 1071
- Rush rod in tibial shaft fractures, 1076
in ulnar shaft fractures, 1022
- Russell traction, in acetabular fractures, 1058
in intertrochanteric fractures, 1068
- Rynd, Francis, 12
- Sac, peritoneal, greater 486
lesser 486
- Sacral anesthesia, 174
- Sacroiliac joint, tuberculosis, 1143
- Sacrum, fractures, 1060
- Saddle block anesthesia, 177

- Shock, electric in defibrillation, 109
in ventricular fibrillation, 468
etiology 88
hemorrhagic, 87
impeding, 87
hypoxic damage in prevention, 93
in fractures, treatment, 981-991
in open fractures, treatment, 985
in peritonitis, 491
in wound evisceration, 113
irreversible 87
adrenal cortex in, role 91
autonomic blocking agents in 90
bacterial factor in, 90
balance between vasoconstrictor material and vasodilator material and, 90
etiology 88
liver in, role of 91
metabolics in, 91
neurogenic, 87
patient, management during operation, 94
readiness for operation, 94
prevention, 91
in heart disease preoperative measures, 107
qualifications of 87
spinal, treatment, 883
surgical, 87
treatment, 91
principles, 93
wound, 87
- Shoulder dislocation, 1106
recurrent, 1044
fractures, 1041
fracture-dislocation, 1044
frozen, 1110
function, portable pulley apparatus in, 1308
girdle, fractures, 1045
joint, dislocations, 1043
tuberculous, 1145
- Shunt, portacaval, in portal hypertension, 792, 793
surgical procedure 794
- Sigmoid, perforation, 700
- Sigmoidoscopy in chronic ulcerative colitis, 703
perforation of sigmoid during, 700
- Sigmoidostomy historical aspects, 700
- Sigmoidovesical fistulae, 705
- Sign, Babinski in head injuries, 1404
Ballancette, in splenic rupture, 845
Battle's, in head injuries, 1406
Bouchard's, 493
Cullen's, in ectopic pregnancy 946
Hamman's, in mediastinal emphysema, 370
Homans's, in venous thrombosis, 1337
Kernig's, in meningitis, 1412
Laszlo's, in acute hemogenous osteomyelitis, 1115
Lhermitte's, in intraspinal tumors, 1443
Sister Joseph's, 484
string, in regional enteritis, 689
Tinel's, in peripheral nerve injuries, 1457
in tardy median nerve palsy 1458
- Silk sutures, history 6
- Simpson, Sir James Y., 11
- Snare, anal, definition, 722
etiology 722
branchiogenic 792. See also Neck anomalies, lateral
coronary ligation in coronary artery disease, 461
dermal congenital, 1479
ethmoid, displacement of secretions in, 230
infection, surgical treatment, 231
frontal, infection maxillary sinusitis and, treatment, 230
Lynch frontotomoid operation in, 231
surgical treatment 231
maxillary infection, osteotomy in, 230
Caldwell-Luc procedure in, 231
frontal sinusitis and treatment, 230
nasal accessory. See Sinuses, paranasal
of Vahlva, aneurysm, congenital, 462
surgical repair 459
paranasal, 227
carcinoma, 238
surgical treatment, contraindications, 240
infection, 227
complications, 229
diagnosis, 228
mucous membrane changes in, types, 228
treatment, 229
surgical, 30
tumors, 237
benign, 237
malignant, 238
electrosurgery in, 240
surgical treatment, 40
- Sphenoid, infection, surgical treatment, 232
irrigation, 230
superior longitudinal, syndrome, in head injuries, 1402
- Thyroglossal. See Cysts thyroglossal.
- Umbilical, 478
urachal, umbilical, 478
venous, lateral, thrombosis, vs. intracranial abscess, 1432
- Ununited. See Sinuses, paranasal, infection.
- Slater Joseph's sign, 484
- Skeleton, metastasis of mammary carcinoma to, 350
- Stone's ducts, gonorrheal infection of 974
- Sterne's gland, infection of, 927
- Skin, closure, materials for 11
grafts, in burns, 195
in hand injuries, 1212
in wound closure, 84
pedicle in wound closure, 85
types, 84
hand tumors arising from, 1230
in cold injury 201
preoperative preparation, 104
resistance, electrical, measurements in neuromuscular disorders, 1304
tension lines, 475
test, intradermal, in chancroid, 974
viability determination, 84
- Skull, 213-217
changes in injuries, 1393
compression 1393
deformation, 1393
depression, 1407
diseases, acquired, of unknown origin, 215
congenital, 215
of abnormal metabolism, 216
eosinophilic granuloma and, 216
fracture open, 1407
Hand-Schüller-Christian disease and, 216
hyperostosis, 215
Letterer-Siwe disease and, 216
osteitis deformans, 216
osteomyelitis, 216-217
as complication of sinusitis, 229
Paget's disease of 216
structural characteristics, 1392
tumors, benign, 216
malignant, 216
metastatic, 217
- Smith, Edwin, Papyrus, 2
- Smith fracture 1012
- Smith-Petersen nail in femoral neck fracture 1063
- Smoking, cigarette and lung cancer relationship, 415
postoperative atelectasis and, 116
restriction in peptic ulcer 622
- Snapping finger 1228
- Sodium, balance postoperative 1-4
1-6
concentration, effect on intracellular fluid volume, 127
of extracellular fluid, disturbances, 133
extrarenal losses, as cause of low serum sodium, 149
in gastrointestinal discharges, estimation, 147
losses, in bile, 179
in gastrointestinal fluids, 128, 129
in pancreatic juice 179
low syndrome 133
in convalescence 149
metabolism, post-traumatic, 142
- Soft tissue. See Tissue soft.
- Solution(s) Fowler's carcinoma of hand and, 1235
hyperbaric, 173
hypobaric, 173
isobaric, 173
- Soreness, 13
- Sore throat, quincy 234
septic, 233
- Specie's, dead, in infection, elimination of, 51
facial, of hand, anatomy 1191
infections, 1191
of neck, 302
mastocyst 302
infection, 307
palmar middle 1192, 1193
parotid, 303
infection, 303
retroflexor 1192, 1193
retroperitoneal. See Retroperitoneal space
retropharyngeal, 303
infection, 303
subhepatic, 302
submandibular. See Submandibular space
submaxillary, 302
subphrenic, 302

- Sagittal suture premature closure, 1481
- St. Martin, Alex, emotional states and, 632
- Salivary ducts, 285
- fistula , 284
- Salivary glands, 285
- and duct, calculus, 286
- deformity in obstruction, 286
- infection, 286
- lacerations, 285
- scars in obstruction, 286
- trauma, 285
- carcinoma, jaw invasion by 284
- tumors, benign, 286
- malignant, 288
- mixed, of anlage origin, 287
- Salivary system, obstruction, 285
- Salmonella infection, 46
- Salpingitis, acute vs. appendicitis, 695
- gonorrheal, 956
- acute differential diagnosis, 956
- chronic, 957
- Salpingo-oophorectomy bilateral, in
- adenocarcinoma of ovary 970
- in carcinoma of corpus uteri, 939
- in cystadenoma of ovary 965
- 966
- in granulosa cell tumor 968
- in pelvic tuberculosis, 959
- in thecal cell tumor 968
- in dermoid cyst of ovary 969
- Salt content of gastrointestinal tract discharges, 147
- Samitis, Charaka, 2
- Scars, 2
- Sange s muscles, 927
- Sarcoid, Boeck's, pulmonary 414
- splenomegaly in, 851
- Sarcoma, cardiac, 466
- Ewing's, 1163
- vs. primary reticulum sarcoma of bone 1164
- gastric, 665
- incidence, 663 665
- pathologic classification, 666
- treatment, 666
- results, 666
- types, 665
- Kapow's, of soft parts of extremities, 1175
- lymph gland involvement in, 1388
- neurogenic, bone involvement in, 1170
- of abdominal wall, 483
- of soft part of extremities, 1175
- of abdominal wall, 483
- of bladder 878
- of bone, of hand, 1246
- reticulum, primary 1163
- of breast, 367
- of chest wall, 397
- of corpus uteri, 940
- of duodenum, 680
- of ear 251
- of endometrial stroma, 940
- of epiglottitis, 904
- of esophagus, 601
- of hand, 1236
- of lung, 422
- of mouth and lips, 270
- of nose, 239
- of ovary 971
- of pancreas, 811
- of paranasal sinuses, 239
- of pleura, 397
- Sarcoma, of prostate, 915
- of rectum, 779
- of small intestine, 691
- of soft parts of extremities of undetermined origin, 1173
- of spleen, 850
- of thyroid 371
- osteogenic 1154
- of jaw 284
- osteoblastic 1155 1156
- osteolytic, 1155 1156
- sclerosing, 1155
- telangiectatic, 1155
- reticulum, primary of bone, 1163
- reticulum cell, 1385
- of retroperitoneal space, 516
- spindle cell, of abdominal wall, 483
- synovial, 1172
- of soft parts of extremities, 1173
- Scab formation in wound healing, 30
- Scalene node biopsy in bronchogenic carcinoma, 419
- Scalene syndrome, 389 1458
- Scalp, 13-217
- contusions, findings in, 1406
- cylindromas 214
- epithelial cysts, 214
- hematomas, 85 1406
- lacerations, findings, 1406
- layers of 213
- surgical dressings for 73
- tumors, benign, 214
- malignant, 215
- wounds, 214
- Scapula, fractures, 1048
- first aid, 987
- Scar(s) burn, in carcinoma of hand, 1235
- contraction, 27 28
- phase of wound healing, 23
- time, effect of trauma on, 79
- formation, 27
- Scarpa's fascia, 475
- Schwannoma, 1459
- Schwartz's sign in otosclerosis, 48
- Scrotic hernia, 568
- Scrotic nerve, injuries, 1456
- in hip dislocations, 1096
- Scleroderma, esophagitis and stricture formation following, 601
- Scotland, surgery in, history 17
- Scrotum, anatomy 896
- anomalies, 896
- diseases, 896
- edema, 896
- elephantiasis, 897
- examination in urinary tract disease, 857
- gynecomastia, 897
- infections, 896
- lymphedema, 1387
- periplasma, 897
- physiology 896
- trauma to, 896
- Scurvy joint effusion in, vs. pyarthrosis, 1133
- vs. acute hematogenous osteomyelitis, 1117
- Sebrecht's ointment for skin protection in duodenal fistula, 684
- Secretin, 802
- Secretion(s) duodenal, 612
- gastric. See *Stomach secretion*
- gastrointestinal, sodium, potassium and chloride losses in, 128, 129
- postoperative removal, 107
- Sedimentation rate in breast carcinoma 357
- Seibers, Jacksonian in head injuries, 1404
- Sella turcica, tumors, 1425
- Sellar region, tumors, 1425
- Semilunar cartilages, fractures, 1089 1090
- mucoid cysts, 1089
- Semilunar hernia, 55
- anatomic factors, 552
- definition, 520
- Seminal vesicles, 916
- anatomy 916
- anomalies, 916
- infection, 916
- physiology 916
- trauma to, 916
- tumors, 916
- Seminal vesiculectomy in chronic seminal vesiculitis, 916
- Seminal vesiculitis, 916
- Seminoles, 901
- Semmelweis, 4
- Sensation, hand injuries affecting, 1207
- in healed wounds, 31
- Septic sore throat, 233
- Septicemia in peritonitis, 493
- Septum, aortic. See *Aorta, aortic*.
- nasal, abscess, 226
- deviation, 225
- submucous resection in, 225
- Sequestrectomy in acute hematogenous osteomyelitis, 1120
- Sequestrum, formation, 1113
- in osteomyelitis, 1111
- Seroma, wound, 112
- Serum, alkaline phosphatase, level in breast carcinoma, 357
- amylase, determination in acute edematous pancreatitis, 805
- electrolytes, metabolism, post-traumatic, 142
- potassium, level in peritonitis, 496
- protein, level in peritonitis, 496
- metabolism, post-traumatic, 143
- sodium, level in peritonitis, 496
- Setting, muscle, 1307
- Sex, as factor in hernia, 521
- hormones in peptic ulcer 614
- incidence of congenital hypertrophic pyloric stenosis, 575
- Sexual infantilism, 919
- Sexual precocity 919
- Sexual symptoms of ovarian failure, 948
- Shay rat, experimental ulcer in, 634
- Sheath, carotid, 306
- infection, 306
- rectus, 475
- testes, band tumors arising from, 1238
- of hand, anatomy 1191
- infections, 1191
- Shelter foot, 199
- Shock, 87-95
- burn. See *Burn shock*.
- "chronic," 102

- Stoma ulcer incidence 607
surgical treatment, 625
results, 625
symptoms, 617
- Stomach, 486, 606-677
aberrant pancreatic tumor of 665
anatomy 607
atrophy 629
carcinoma. See *Carcinoma gastric*
dilatation, acute, postoperative 117
foreign bodies in, 627
functions in digestion, 613
inflammation, specific 629
lesions, benign, 627-630
lymph vessels, 1368
mucosa, 610
prolapse 629
muscle, tone changes in, 613
obstruction in peptic ulcer 620
medical treatment, 623
surgical treatment, 624
peristalsis, 613
physiology 608, 631
precancerous lesions, 669
resection. See also *Gastrectomy*
dumping syndrome following, 626
duodenostomy following, 647
in duodenal ulcer results, 625
in peptic ulcer 625-638. See also *Gastrectomy partial*
in sarcoma of stomach, 666
palliative in gastric carcinoma, 672
primary in peptic ulcer with free perforation, 649
radical, partial, in gastric carcinoma, 671
subtotal. See *Gastrectomy partial*
secretion, 608. See also *Gastric acid secretion*
components, 608
mechanism, 608
phases, 63
small, syndrome, following peptic ulcer surgery 660
following subtotal gastric resection, 639
suction and laparotomy in perforated peptic ulcer 630
syphilis, 629
tube, passing, technique, 76
tuberculosis, 629
tumors, 663-677
benign, 663
incidence, 663
classification, 663
incidence, 663
malignant, 665
mortality 667
pathologic classification, 666
ulcer benign and malignant, differential, 621
resection, anastomosis following, 640
chronic, 655
pathogenesis, 655
surgical treatment, 655
diagnosis, 620
duodenal ulcer and, 655
differences, 620
gastric carcinoma and, relation, 620-669
occurrence, 606
location, 615
malignancy in, incidence, 656
- Stomach, ulcer malignancy in, operation, indications, 656
perforated treatment 649
roentgenography in, 618
surgical treatment immediate 64
results, 625
symptoms, 617
treatment results, 626
ulcer-cancer problem and, 620
vs. cancer 656
wounds, treatment, 510
- Stomachitis, 256
ulcerative 256
- Stones, lung, 408, 414
urinary tract, vs. appendicitis, 694
- Strains, of ligaments, definition, 1106
physical medicine and rehabilitation in, 1316
- Strangulation, in intestinal obstruction, 735-737, 740
treatment and, 748, 751
immediate 749
intestinal, 732
peritonitis and, 489
- Strangury in urinary tract disease 856
- Stratum germinativum, importance in epithelial regeneration, 30
- Straw technique in gastroduodenal hemorrhage, 653
- Strawberry gallbladder 772
- Strawberry mark of hand, 1242
treatment, 143
- Strawberry vagina, 923
- Street nail in femoral shaft fractures, 1071
- Strength-duration curve in neuromuscular disorders, 1302
- Streptococcus, aerobic, in infection, 44
effect on wound healing, 33
hemolytic, in infection, 44
infection and, 44
nonhemolytic, in infection, 44
- Streptococcus fecalis in infection, 44
- Streptococcus viridans in infection, 44
- Streptodornase, production, 34
- Streptokinase, production, 34
- Streptomycin, in pulmonary tuberculosis, 405
in surgical infections, 57
toxic reactions, 56
- Stream, circulatory response to, in etiology of shock, 88
relay from hypothalamus to stomach, 611
ulcer 635
acute, reaction to, 636
vagotomy in, 636
- String sign in regional enteritis, 689
- Stromeyer 18
- Struma lymphomatosa, 320
- Struma, Riedel's, 320
- Stryker bed in Pott's disease, 1141
- Stump, amputation, bandaging, 1321
- Stumpure arterial, 1351
- Sturmudorf operation in chronic endocervicitis, 926
- Styloidectomy radial, in nonunion of navicular bone fracture, 1006
- Subaortic stenosis, 455
surgical correction, 456
- Subclavian artery right, origin from left of aortic arch, 436
- Subdiaphragmatic abscess, suppurative necrosis of diaphragmatic hernia due to, 364
- Subdural abscess. See *Abscess subdural*
- Subdural empyema. See *Abscess subdural*
- Subdural hematoma. See *Hematoma, subdural*
- Subhepatic space, 502
- Sublingual lymph node enlarged, vs. femoral hernia, 539
- Submandibular space 304
infection, 304
treatment, 305
phlegmon, 304
- Submaxillary space 302
- Submaxillary tumors, malignant, 288, 289
- Submucous resection in nasal septal deviation, 225
- Subpectoral abscess, 390
- Subphrenic abscess. See *Abscess subphrenic*
- Subphrenic region, anatomy 502
- Subphrenic spaces, 502
- Subrecovery unit, 110
- Subscapular abscess, 390
- Subtalar dislocation of foot, 1099
- Subungual abscess, 1189
- Soby's solution C in urinary calculi, 877
- Succinylcholine 169
- Suction, gastric, and laparotomy in perforated peptic ulcer 650
- Suction aspiration, direct, in intestinal obstruction, 750
- Suction biopsy in fibroids, 944
- Suicide, attempted, incised wounds of neck in, 501
- Sulcus, pulmonary tumor 397
superior syndrome, 1459
in bronchogenic carcinoma, 420
- Sulf derivatives in preoperative bowel preparation in colonic cancer 711
- Sulfadiazine, in chancroid, 974
in endocervicitis, 925
in gonorrhea, 927
- Sulfonamides in meningitis prophylaxis, 1412
- Sulkowitch test, in urinary calculi, 872
in urinary tract disease, 858
- Sump drainage as replacement for heavy dressings, 74
- Swan in hand carcinoma, 1232
- Supercooling of tissues, 200
- Superinfection, 47-55
Mycostatin in, 58
protection against, 50
- Superior sulcus syndrome, 1459
in bronchogenic carcinoma, 420
- Supervoltage ray therapy in Hodgkin's disease, 1348
- Suppuration, following breast trauma, 336
pulmonary postoperative atelectasis and, 116
versus primary wound healing, history 3
- Suprahepatic space 502
- Suprathyroid dissection in carcinoma of lip 270
- Suprathymic hernia, 568

- Space(s) suprabepatic, 502
surgical, of anorectum, 717
thelar 1192, 1193
third, of peritoneal cavity 491
visceral, of neck, infections, 306
- Spade-handle deformity in Smith fracture, 1012
- Sperm, arterial, segmental, 1351
muscular in appendicitis, 694
- Speech, audiograms, 247
audiometry in Meniere's disease, 250
disturbances, in head injuries, 1401
in intracranial tumors, 1422
therapy definition 1298
in surgical conditions, 1325
- Spermatic cord, 906
anatomy 906
anomalies, 906
infection, 907
torsion, 906
vs. acute epididymitis, 903
907
trauma to 906
tumors, 907
- Spermatic fluid examination in infertility 918
- Spermatocoele, 894
- Spermatogenesis, restoration of 918
- Sphenoid sinus, irrigation, 230
- Spherocytosis in primary hemolytic anemia, 848
- Sphincter of Oddi, section of in relapsing pancreatitis, 807
pyloric, function, 613
- Sphincterotomy in relapsing pancreatitis, 807
- Sphygmomanometer cuff pain test in venous thrombosis, 1337
- Spider fingers, 1252
- Spider web varicosities, 1330
- Spiegel, Luca semilunaris of 475
- Spiegel's hernia, 552
- anatomic factors, 552
definition, 520
- Splina bulbia, 1475
etiology 1475
occult, 1475 1476
diagnosis, 1477
surgical treatment, 1477
treatment, 1478
with meningocele, 1475 1476
1477
treatment, 1478
with myelomeningocele, 1475
1476, 1477
treatment, 1478
with rachischisis, 1475 1476
- Spinal accessory nerve, disorders, 1450
- Spinal anesthesia. See *Anesthesia, spinal*.
- Spinal cord, 1439-1446
cervical, injuries, 1439
compression in intraspinal tumors, 1442
injuries, 1439
care of patient in, 1439
rehabilitation in, 1440
treatment, 1440
lumbar injuries, 1439
thoracic, injuries, 1439
tumors, 1441
classification, 1441
diagnosis, 1443
extradural, 1442
intramedullary 1442
- Spinal cord, tumors, incidence, 1441
intradural, 1442
intramedullary 1442
metastatic, incidence, 1441
symptoms, 1442
treatment, 1444
- Spinal puncture. See *Lumbar puncture*.
- Spinal shock, treatment, 883
- Spine, cervical, dislocations, 1051
fractures, 1051
dorsal, fractures, 1053
fractures 1051 1055
first aid 982
lumbodorsal, fractures, 1053
tuberculous. See *Tuberculosis of spine*.
- Sphenoidal tractotomy 1469
- Spirillum minus infection, 44
- Spirochetes in infection, 44
- Splanchinectomy in relapsing pancreatitis, 808
- Spleen, 842-853
abscess, 851
accessory 844
anatomy internal, 843
surgical, 842
anomalies, 844
contusion, 845
cysts, 850
hydatid, 850
nonparasitic, 850
parasitic, 850
diseases, 852
ectopic, 845
embryology 485
in Banti's disease, 852
infections, 851
lymph vessels, 1368
peritoneal attachments, 842
physiology 844
rupture, 845
in hemoperitoneum, 510
secondary 845
spontaneous, 845
treatment, 509
trauma to, 845
tumors, 850
- Splenectomy in acquired hemolytic anemia, 850
in cysts of spleen, 850
in infections of spleen, 851
in primary hemolytic anemia, 848
in primary splenic neutropenia, 848
in primary splenic panhematopoiesis, 849
in primary thrombocytopenic purpura, 848
in secondary hypersplenism, 850
in splenic rupture, 845
in tumors of spleen, 850
- Splenic artery aneurysm, 851
- Spleen, 844
- Splenorenal ligament, 843
- Splenorenal shunt in portal hypertension, 792
- Splitting, dynamic in lower motor neuron disorders, 1320
in hand infections, 1183
position of function, 1184
in lower extremity fractures, 983
- "Sponge, dry technique of estimating blood loss in surgery 92
- Spongia sponifera, 10
- Spongioleptostoma polare, 1425
- Sporotrichosis, hand involvement in, 1203
- Spots, coal, in irradiation carcinomas, 1234
- Sprains, 80
fracture, definition, 979
of ligaments, dislocation, 1106
of wrist, treatment, 1004
physical medicine and rehabilitation in, 1316
- Spring water cyst of mediastinum, 377 378
- Squamous carcinoma of lung, 418
- Squamous cell carcinoma of lip, 267
268
treatment, 268
- Stub wounds of neck, 307
- Stuhr node of, 1371
- Stains, port-wine, of lips, 265
- Standing, 1264
- Stapes mobilization, technique, 248
- Staphylococcus, effect on wound healing, 33
infection and, 43
- Staphylococcus albus in infection, 43
- Staphylococcus aureus, bacteriophage typing of, 44
in infection, 43
- Starch-iodine test in sweating pattern alterations, 1304
- Starvation, bodily derangements produced by 98
- Stasis, bile, gallstone formation and, 767
blood, in cold injury 200
lymph. See *Lymphedema*.
- Stenotria, postoperative, in obstructive jaundice, 814
- Stein-Leventhal syndrome vs. uterine leiomyoma, 967
- Steinmann pin(s), in calcaneal fractures, 1085
in rib fractures, 1049
in ulnar shaft fractures, 1022
- Stem prosthesis, intramedullary in femoral neck fracture, 1064
- Stenosis, aortic. See *Aorta, stenosis*.
cricoid, of larynx, 312
laryngeal, 311
chronic, 312
mitral. See *Mitral stenosis*.
pulmonic. See *Pulmonic stenosis*.
subaortic, 455
surgical correction, 456
- Sterility endometriosis and, 963
in chronic gonorrheal salpingitis, 957
in urinary tract disease, 857
male, 917
- Sternoclavicular joint, dislocation, 1048
- Steroid metabolism, alterations, in breast carcinoma, 363
- Stilbestrol, in dysmenorrhea, 932
in endocervicitis, 925
in threatened abortion, 942
- Silman method in hip dislocation, 1096
- Stimulation, electrical, 1312
in lower motor neuron disorders, 1320
repetitive, in neuromuscular disorders, 1302
- Stockholm technique in cervical cancer 937
- Stoma ulcer gastroenterocolic fistula in, 626

- Tenosynovitis, of hand tuberculous, 1700
stenosing, 1228
- Tension, intraluminal, increased in intestinal obstruction, 736
- Tension lines of Langer, 475
- Tension pneumothorax, 384
- Tetaloema(), of neck, 796
- of ovary 971
- of testis, 901
- Tetramycin, 57. See also *Tetracycline*
- in gonorrhea, 927
- Test, Allen's, for occlusion of radial or ulnar artery 1346
- balloon motility in recurrent ulcer 654
- caloric, in Meniere's disease 250
- cobalt chloride in sweating pattern alterations, 1305
- Coombs in acquired hemolytic anemia, 840
- cough-pain, in venous thrombosis, 1337
- dorsiflexion, in venous thrombosis, 1337
- Foxhay intradermal, in tularemia, 1203
- Friei, in inflammatory rectal stricture, 725
- in lymphogranuloma inguinale 974
- in lymphogranuloma venereum, 908
- Hamilton rabbit, in diffuse osteitis fibrosa, 1161
- Landis-Gibson, in peripheral vascular disorders, 1323
- Lawrence in venous thrombosis, 1337
- Lowenberg's, in venous thrombosis, 1337
- Matis, of collateral blood flow 1345
- occulometric, in peripheral vascular disorders, 1322
- Perthes in varicoceles, 1331
- phenolsulfonphthalein, in urinary tract disease, 859
- Queckenstedt in intraspinal tumors, 1443
- in spinal injuries, 1440
- quinazarin dye, in sweating pattern alterations, 1305
- Rinne hearing, 247
- Rubin, periumphreum and, 512
- Schaller's, in cervical cancer 936
- Schwabach hearing, 47
- spinymanometer cuff pain, in venous thrombosis, 1337
- starch-iodine, in sweating pattern alterations, 1304
- Sulkowitch, in urinary calculi, 872
- in urinary tract disease, 858
- sweating pattern, in peripheral nerve injuries, 1304
- three-glass, in urinary tract disease, 858
- tilt, in gastroduodenal hemorrhage, 651
- Tobey Auer of jugular compression, 143
- tomiquet, in primary thrombocytopenic purpura, 847
- Trendelenburg, in varicoceles, 1330
- Test, tuberculin, in tuberculous of bones and joints 1139
- in tuberculous of spine 1149
- Weber hearing, 47
- Testes, anatomy 897
- anomalies, 898
- appendix torsion of 900
- ectopic 900
- incompletely descended, indirect inguinal hernia and 530
- infections, 900
- lymph vessels, 1369
- pain in urinary tract disease 856
- physiology 897
- removal. See *Orchiectomy*
- trauma to 900
- tumors, 901
- diagnosis, 901
- interstitial cell, 901
- metastatic 901
- prognosis, 902
- radioactivity 902
- types, 901
- undescended. See *Cryptorchism*
- Testosterone in adenocarcinoma of ovary 970
- in male infertility 918
- prophylaxis, in breast carcinoma treatment, 364
- in cryptorchism, 899
- in menorrhagia and metrorrhagia 950
- Tetanzizing current in neuromuscular disorders, 1301
- Tetanus, 44
- antitoxin, 66
- in open fractures, 986
- in burns, 197
- prophylaxis in open injuries, 85
- protection against, 66
- toxoid, 66
- in open fractures, 987
- Tetanus direct current ratio in neuromuscular disorders, 1302
- Tetracaine 171
- Tetracycline, in osteomyelitis, acute hematogenous, 1118
- in surgical infections, 57
- toxic reactions, 56
- Tetralogy of Fallot. See *Fallot tetralogy*
- Thalamotomy 1436
- Thecal cell tumor 968
- Theca luteum cyst, 96
- Theodoris, 3 10 13 15
- Therapeutic exercise, 1306
- in amputations, 13 1
- in lower motor neuron disorders, 1320
- in orthopedic disorders, 1317
- in surgical conditions, 1324
- in thoracic surgical conditions, 1324
- in upper motor neuron disorders, 1318
- Therapy blind rehabilitation, definition, 1298
- cold, 1306
- corrective, definition, 1298
- educational, definition, 1298
- heat, 1305
- in orthopedic disorders, 1317
- manual arts, definition, 1298
- in orthopedic disorders, 1317
- occupational, definition, 1298
- in orthopedic disorders, 1317
- in surgical conditions, 1325
- Therapy physical, definition, 1298
- speech, definition, 1298
- ultrasound 1313
- in amputations, 132
- Thermal injuries, 182 212. See also *Burns*
- Thermal necrosis of jaw 281
- Thermotherapy. See *Heat*
- Thiamine administration, in Meniere's disease 250
- preoperative 103
- Thigh, lymphangiography 1389
- Thiopental, 168
- Thioscobarbital 168
- Thomas splint in lower extremity fractures, 983
- Thoracentesis, in empyema, 392
- technique, 76
- Thoracic aorta, aneurysms, 462
- Thoracic cord, injuries, 1439
- Thoracic duct anatomy 1364
- Injuries, 1373
- Thoracoplasty in pulmonary tuberculosis, 402
- Thoracotomy exploratory in bronchogenic carcinoma, 420
- Thorax. See also *Chest*
- anomalies, congenital, 388
- cavity physiology 38
- chyle 1373
- foreign bodies in, 387
- fractures, 1048
- injuries, 384
- contaminated, 386
- infection in, 386
- lacerated, 386
- non-surgical, 387
- requiring immediate attention, 384
- types, 384
- lacerations, 386
- lower injuries, upper abdominal involvement in, 387
- lymphatic drainage, 1369
- operations, complications, 452
- surgical conditions, ambulation in, 1324
- antibiotic prophylaxis in, 59
- breathing exercises in, 13 4
- heat in, 1374
- physical medicine and rehabilitation in, 1323
- objectives, 13.3
- procedures, 1323
- preoperative orientation of patient, 1324
- therapeutic exercise in, 13 4
- work capacity in, 1324
- wall, 387-398
- actinomycosis, 391
- inflammations, 390
- involvement in bronchogenic carcinoma, 421
- tuberculosis, 390
- tumors, 396
- Thornwaldt's disease, 232
- Three-glass test in urinary tract disease, 858
- Throat, sore quinsy 234
- septic, 233
- Thromboangitis obliterans, 1353
- Thrombocyte count in radiation injuries, 211
- Thromboembolism, 1333
- incidence, 1335
- pulmonary following phlebotomy, 1338

- Surface lesions, evaluation, 1316
- Surgeons, consultant, recommendation, 1489
- fugitive 15
- qualifications, 1486-1490
- training of, history 13
- Surgery antiseptic history 4
- aseptic, history 4
- definition, 1
- goals in, conflicting, 1496
- history of, 1-21
- motivations in, 1492
- newness in, 1495
- rapport in, 1493
- Renaissance, 15
- routine in, 1493
- separation from medicine, 3
- stereotypy in, 1493
- Surgical care, technique in, basic principles, 63-78
- Surgical conditions, ambulation in, 1324
- manual arts therapy in, 1325
- occupational therapy in, 1325
- physical medicine and rehabilitation in, 1324
- objectives, 1324
- procedures, 1324
- speech therapy in, 1325
- therapeutic exercise in, 1324
- So glial judgment, 1491-1497
- fear and, 1494
- preoperative, 1492
- technical skill and, 1494
- Surgical techniques, teaching, 70
- Sutural, 168
- Sutura, 2, 1486, 1487
- Suture(s), coronal, premature closure, 1481
- cranial, premature closure, 1480
- delayed, in accidental wounds, 85
- multiple, technique in cavernous aneurysms of mouth, 265
- retention, 112
- scutellar, premature closure, 1481
- secondary in accidental wounds, 85
- skin, history 6
- Sweating pattern tests in peripheral nerve injuries, 1304
- Swelling in wound healing, 24
- Sylvester method of artificial respiration, 179
- Syme, James, 5 11 17
- Sympathectomy 1471
- in chronic obliterative arterial diseases, 1354
- in hyperhidrosis of nervous origin, 1467
- in lymphedema, 1379
- in tachycardia, 1468
- in vasomotor activity disorders, 1467
- prophylactic, in acute arterial occlusion, 1465
- regional, in increasing blood flow to extremity 1347
- response to, evaluation, tests for 1461
- Sympathicoblastoma, 839
- Sympathic, 1461
- Syncope in tetralogy of Fallot, 440
- Syndactylism, of hand, 1250
- of toes, 1271
- Syndrome, adrenogenital. See Adrenogenital syndrome.
- anemia intoxication, 794
- Syndrome, anterior scalene, 389
- 1478
- auriculotemporal, 275 1450
- brain stem, 1395
- Brown-Séquard in intraspinal tumors, 1443
- carotid sinus, 1466
- Cushing's. See Cushing syndrome
- dumping, following gastrectomy 626
- following peptic ulcer surgery 660
- following subtotal gastric resection, 639
- Felty's, 851
- Frohlich's 919
- Gradenigo's, 1432
- Klüfelter's, 919
- Lencho's, 1353
- low sodium, 133
- in convalescence, 149
- Maffucci's, 1152
- Meigs 968
- middle lobe, 406
- of superior longitudinal sinus in head injuries, 1402
- Pancoast, 1459
- in bronchogenic carcinoma, 420
- Plummer-Vinson, 600
- postphlebotic, prognosis, 1337
- treatment, 1341
- radiation, acute, 208
- scalene, 389 1458
- small stomach, following peptic ulcer surgery 660
- following subtotal gastric resection, 639
- Stem-Leventhal, vs sarcomatosis, 967
- superior sulcus, 1459
- in bronchogenic carcinoma, 420
- temporomandibular meniscus, 276
- Turner's, 919
- Synovial fluid, study in pyarthrosis, 1132
- Synovial membrane, 1138
- Synovium(s), 1172
- of abdominal wall, 483
- of hand, 1241
- origin, 1109
- Synovitis, villonodular, of hand, vs tuberculous synovitis, 1201
- Synovium, joint, chondromatous of 1171
- Syphilis, gastric, 629
- vs gastric carcinoma, 671
- in female, 974
- of jaw 283
- of mouth, 256
- pulmonary 414
- vs Hodgkin's disease, 1387
- Syphilitic aneurysms of aortic arch, 462
- Systemic diseases, debilitating, small intestine and, 688
- TACHYCARDIA, sympathectomy in, 1468
- Tailor's bottom, 1109
- Talc, granulomatous peritoneal lesions due to, 499
- Talipes cava, 1277
- Talipes equinovarus, congenital, 1266
- treatment, 1268
- Talus, body aseptic necrosis of, 1082
- dislocation, 1099
- fractures, 1080
- vertical, congenital, 1277
- Tamponade, balloon, in bleeding esophageal varices, 793
- cardiac, in pericardial effusion, 447
- Tapping, mallet, in fractures, 994
- Tarsometatarsal dislocation, 1099
- Tarsorrhaphy in exophthalmos, 221
- Tate, Lawson, 5
- Taylor back brace, in dorsal spine fractures, 1053
- in lumbodorsal spine fractures, 1054
- Tears, facial, 253
- Technique in surgical care, basic principles of 63-78
- Teeth, treatment in jaw fractures, 274
- Tela subcutanea of cervical fascia, 301
- Temperature, body 198
- effect on wound healing, 39
- in head injuries, 1398
- regulation in hypothermia, 430
- Temporal lobe, abscess, 1431
- herniation in head injuries, 1402
- tumors, 1474
- Temporomandibular joint disorders, 275
- Temporomandibular meniscus syndrome, 276
- Tenderness, abdominal, in appendicitis, 693
- in subphrenic abscess, 504
- Tendon(s), division in hand injuries, 1208
- excision, in wound cleansing, 84
- graft in reconstructive surgery of hand, 1253
- hand, injuries, 1214
- healing of, 39
- in cold injury 202
- in reconstructive surgery of hand, 1253
- of hand, extensor injuries, 1215
- extensor pollicis longus, rupture, 1216
- flexor injuries, 1215
- rupture, 1220
- repair in hand injuries, 1210
- sheaths, hand tumors arising from, 1238
- of hand, anatomy 1191
- infections, 1191
- xanthomatous tumor vs tuberculous synovitis, 1201
- snapping, 1228
- suture in reconstructive surgery of hand, 1253
- transfer, in reconstructive surgery of hand, 1254
- in rupture of extensor pollicis longus, 1217
- Tenosynovitis, of hand, acute, 1193
- diagnosis, 1194
- symptoms, 1194
- treatment, 1195
- gonorrheal, vs tuberculous synovitis, 1201
- nonspecific, vs tuberculous synovitis, 1201
- syphilitic, vs tuberculous synovitis, 1201
- traumatic, vs tuberculous synovitis, 1201

- Trauma, blunt, abdominal, 508
 effect on scar tissue 29
 esophageal, 598
 in infection, protection against, 50
 intra-abdominal, 507
 classification, 507
 nonpenetrating, 508
 pneumoperitoneum and, 511
 penetrating, 507
 surgical treatment, 508
 surgical treatment, objectives, 509
 moderate clinical management in, 143
 prototype, 139
 rectus sheath hematoma and, 481
 to biliary system, 764
 to liver 764
 to muscles, 1102
 wounds of antibiotic therapy in, 59
- Traumatic aneurysms of aortic arch, 462
- Trench foot, 199
- Trench mouth, 34
- Trendelenburg test in varicoities, 1330
- Trephinations, exploratory multiple in craniocerebral trauma, 1414
- Treponema pallidum infection, 44
- Triangle Hesselbach's, 535
- Inferior lumbar of Petri, anatomy 553
- superior lumbar of Grynfelt, anatomy 553
- Tribromethanol, 167
- Triceps surae, paralysis, 1,88
 short, 1774
- Trichinella spiralis infection of skeletal muscle 1103
- Trichlorethylene, 165
- Trichobezoar 627
 vs gastric carcinoma, 670
- Trichomonas vaginalis, 923
- Trichomoniasis, 923
 organisms in, 923
 trichomonads in, 974
- Trichterbrust, 388
- Trigeminal nerve, 1446
- Trigeminal neuralgia. See *Neuralgia, trigeminal*
- Trigoosis in female, 974
- Trilene, 165
- Trilicon in trichomoniasis, 924
- Trochanter fractures, 1068
 greater mallet tapping in fractures, 994
- Truncus arteriosus, 433
- Tum treatment of herpes, 523
- Trypsin, secretion by pancreas, 802
- Trypanogen, secretion by pancreas, 802
- TSH, 317
 increase in, 317
- Tubal reflux theory 963
- Tube, Ewald, passing, technique 76
- fallopian, cancer discharge in, 929
 lymph vessels, 1369
- Levin, in prevention of distention, 747
 passing, technique, 76
- Miller Abbott, in decompression of small intestine, 746
 passing, technique, 76
- stomach, passing, technique, 76
- uterus, lymph vessels, 1369
- Tube, Wangenstein, in decompression of small intestine 746
- Tubercle bacillus, in infection, 44
 types, 1138
- Tuberculin test, in tuberculosis of bones and joints, 1139
 in tuberculosis of spine 1141
- Tuberculosis, 405
- Tuberculosis, arthritis of foot in, 1294
 empyema secondary to, 395
 enterocolic, 702
 epididymitis in, 904
 fibrocystic, 404
 genital, 949
 intestinal, 688
 of ankle 1145
 of bones and joints, 1138
 clinical types, 1139
 diagnosis, 1139
 symptoms, 1139
 treatment, 1139
- of breast, 338
 of bone, 1147
 of chest wall, 390
 of colon, 702
 of elbow 1146
 of foot 1145
 of hip, 1143
 diagnosis, 1143
 symptoms, 1143
 treatment, 1144
 types, 1143
- of jaw 1142
- of joints vs. pyarthrosis, 1133
- of knee, 1144
 diagnosis, 1144
 treatment, 1145
- of mouth, 256
- of sacroiliac joint, 1143
- of shoulder 1145
- of spine, 1140
 diagnosis, 1141
 symptoms, 1140
 treatment, 1141
- of stomach, 629
- of urinary tract, 869
- of vulva, 975
- of wrist, 1146
- peritonitis with, 498
- prostatitis in, 910
- pulmonary 401
 chemotherapy in, 405
 pneumoperitoneum in, 402
 pneumothorax in, artificial, 402
 pulmonary resection in, 403
 reinfection, 404
 thoracoplasty in, 402
- splenomegaly in, 851
- Tuberculum sellae, tumors, 1425
- Tubocurarine 169
- Tuffier 12
- Tularemia, hand involvement in, 1203
- Tumor(s), Brenner 964
 Ewing's, of hand, 1246
 fatty of biliary tract, 784
 giant cell, extraskeletal, 1163
 of bone, 1162
 of hand, 146
 gliomas, of peripheral nerves, 1459
 granulosa cell, 967
 intracranial. See *Intracranial tumors*
 intraspinal. See *Spinal cord tumors*
 Krukenberg, 972
- Tumor(s) of abdominal wall. See *Abdomen wall tumors*
- of adrenal cortex. See *Adrenal cortex tumors*
- of adrenal medulla 838
 of appendix, 698
 of biliary tract, 784
 of bladder. See *Bladder tumors*
 of blood vessels, 1420
 of bones. See *Bones tumors*
 of breast, benign, 344
 biopsy in, exchional, 360
 common 345
 malignant, 346
 of breast, 1109 1171
 of carotid body. See *Carotid body tumors*
 of cerebellopontine angle, 1425
 of cerebellum, 1474
 of chest wall, 396
 of colon, benign, 706
 classification, 706
 malignant, 708
 of duodenum. See *Duodenum tumors*
 of epididymides, 904
 of esophagus, benign, 601
 malignant, 601
 of extremity soft parts, 1148, 1173
 of foot, 1793
 of hand. See *Hand tumors*
 of hepatic system, 1148 1163
 of heart, 466
 malignant, 466
 of inguinal lymph nodes, metastatic, 908
 of inner ear 251
 of islets of Langerhans. See *Islets of Langerhans, tumors*
 of jaw 1143
 giant cell, 284
 of joints, 1148 1171
 of kidney 880
 of larynx, benign, 312
 malignant, 313
 of lips, benign, 263
 malignant, 767
 of liver 795
 benign, 795
 malignant, 796
 of lung, 414
 of lymphatic system, 1384
 of marrow 1148 1163
 of mediastinum, 376
 of middle ear 251
 of mouth, benign, 263
 malignant, 67
 of mouth, tongue and lips, mixed, 267
 of nasopharynx, 237
 benign, 240
 malignant, 240
 of neck, primary 298
 of nose 37
 benign, 237
 malignant, 238
 of orbit, removal, 220
 of ovary. See *Ovary tumors*
 of pancreas, aberrant, 665
 benign, 811
 ulcerogenic, in peptic ulcer 614
 of paranasal sinuses, 237
 benign, 237
 malignant, 238
 electrocure in, 240
 of parathyroid gland, removal, 325

- Thromboembolism, treatment, 1342
 Thrombopem in radiation injuries, 211
 Thrombophlebitis, 1332
 acute, inflammatory active treatment, 1340
 as cause of postoperative fever, 122
 clinical picture, 1336
 deep vein, active treatment, 1340
 etiology, 1334
 in varicose veins, 1331
 incidence, 1335
 intestinal obstruction in, 754
 migratory, 1334
 of lower extremities, prognosis, 1337
 pathogenesis, 1334
 phlebothrombosis and, distinction, 1334
 septic, puerperal, 958
 suppurative, 1334
 treatment, 1339
 Thrombosis, arterial, acute, 1352
 bland. *See Phlebothrombosis*
 coronary, 460
 hepatic vein, in portal thrombosis, 791
 vs. mitral block, 791
 of lateral venous sinus vs. intracranial abscess, 1432
 of lower extremities, sites of, 1335
 of upper extremities, 1342
 silent. *See Phlebothrombosis*
 venous, iliofemoral, with lymphedema, lymphangiography in, 1390
 prophylaxis, 1339
 Thrush, 928
 Thumb, fracture-dislocation, Bennett's, 1000
 loss of, compensation for operations, 1255
 metacarpal bone, fracture, 1000
 metacarpophalangeal joint, dislocation, 999
 polydactyly, 1251
 snapping, 1228
 Thymoma, 378
 Thymus, lymph drainage, 1371
 Thyroglossal cysts. *See Cysts thyroglossal*
 Thyroglossal fistulae. *See Cysts thyroglossal*
 Thyroglossal sinuses. *See Cysts thyroglossal*
 Thyroid, desiccated, uses, 323
 Thyroid glands, 317-325
 adenocarcinoma, 321-322
 treatment, 322
 adenoma, 321-326
 carcinoma. *See Carcinomas of thyroid*
 in catabolism, 140
 nodules. *See Goiter nodular*
 sarcoma, 321
 treatment, 322
 structure and function, relationship, 317
 tissue, ectopic, vs. thyroglossal cysts, 296
 Thyroid hormone, 317
 in male infertility, 918
 Thyroid struma, 318
 desiccated thyroid fa, 323
 Thyroid-stimulating hormone, 317
 increase in, 317
 Thyroidectomy in Graves disease, 319
 in hyperthyroidism, 319
 Thyroiditis, 320
 granulomatous, 320
 Hashimoto's, 320
 subacute, 320
 types, 320
 Tibia, anterior spine and tubercle, fractures, 1073
 fractures, 1073
 plateau, fractures, 1073
 shaft, fractures, 1073
 Tibial nerve injuries, 1456
 Tibialis anterior, paralysis, foot deformities in, 1285-1287
 Tibialis posterior, paralysis, foot deformities in, 1287
 Tic douloureux. *See Neuralgia trigeminal*
 Tilt test in gastroduodenal hemorrhage, 651
 Time, venous filling, in vasospastic disorders, 1345
 Tinel's sign, in peripheral nerve injuries, 1457
 in tardy median nerve palsy, 1458
 Timpanus in Meniere's disease, 249
 Tissue(s) devitalized, wound healing and, 32
 fluids, biochemical status in fracture healing, 993
 granulation. *See Granulation tissue*
 heat loss in cold injury, 198
 necrosis, amount, in fracture healing, 992
 wound healing and, 32
 nervous, healing of, 40
 pH, in wound healing, 24
 resistance, local, in peptic ulcer, 614
 response, alterations by antibiotics, 45
 scar formation, 27
 soft, injuries, from mechanical forces, 79-86
 in fractures, 991
 of mouth, 52
 of extremities, tumors, 1148
 classification, 1175
 malignant, 1173
 of mouth, infection, 255
 supercooling of, 200
 Tobacco, lung cancer and, relationship, 415
 restriction in peptic ulcer, 622
 Tobey Ayer test of jugular compression, 1432
 Toe(s) affections, 1278
 extensor muscles, paralysis, 1285
 hammer, 1281
 Morton's, 1282
 paronychia, 1293
 superficial, 1271
 transfer in loss of thumb, 1455
 webbed, 1271
 Toenails, growing, 1283
 Tonge, Crutchfield, in cervical vertebral fractures, 1051
 Tongue, 270
 carcinoma, 271
 treatment, 272
 leucation, 254
 tumors, benign, 270
 mixed, 267
 Tonid(s), abscess, 234
 cerebellar herniation, in head injuries, 1403
 facial, 232
 anatomy, 233
 lingual, 232
 anatomy, 233
 palatine, 232
 anatomy, 233
 pharyngeal, anatomy, 232, 233
 streptococcal infection, acute, 233
 Tonsillectomy complications, 236
 indications, 235
 postoperative, 236
 technique, 236
 Tonsillitis, acute, 233
 Tonsillopharyngitis, diphtheria, 234
 Tonsillectomy, 1436
 Topical anesthesia, 170
 Tork procedure in esophageal carcinoma, 603
 Torkildsen operation in intracranial tumors, 1428
 Torsion of omentum, 515
 Torus mandibularis, 783
 Torus palatinus, 283
 Tourquet, history, 9
 in hand injuries, 1204
 in wounds of large arteries, 1348
 test in primary thrombocytopenic purpura, 847
 Toxemia theory in intestinal obstruction, 738
 Toxins, bacterial, in peritonitis, 449
 in wound healing, 33
 escape, in intestinal obstruction, 738
 Trachea, 310-316
 anatomy, 308
 bullet wounds, 308
 carcinoma, squamous cell, 314
 compression from aortic arch anomalies, 435
 constriction by anomalous vessels in mediastinum, 574
 diseases, 311
 foreign body in, 315
 fractures, 314
 infection, 314
 injuries, 313
 tumors, 314
 Tracheoesophageal fistula, 314-317
 Tracheostomy elective, in mediastinotomy, 380, 381
 postoperative, 317
 Tracheostomy, 314
 in burns of face, 196
 technique, 314
 Traction, 1314
 cervical, 1314
 in lower extremity fractures, 983
 in lower motor neuron disorders, 1320
 lumbar-pelvic, 1315
 Russell, in acromioclavicular fractures, 1058
 in intertrochanteric fractures, 1068
 Traction fixation of fractures, 985-986
 Tractotomy aponeurotic, 1469
 Transillumination of breasts, 335
 Trauma. *See also Injuries and Wounds*
 abdominal wall, 479
 as cause of hypostrenia, 150

- Uterus, devolution, in dysmenor-
rhea 93
lymph vessels, 1369
mobility testing, 954
palpation, 954
prolapse 951
etiologic factors, 951
symptoms, 95
treatment 952
ventral hernia and, anatomic
similarity 952
- Vaurea, examination in urinary tract
disease, 858
hernia, posterior. See *Enterocoele*
lymph vessels, 1369
relaxation, anterior. See *Cystocele*
posterior. See *Rectocele*
strawberry 93
symptoms of ovarian failure in,
948
- Vaginitis, endocrine 977
mycotic 928
septic, 927 948
- Vagotomy gastric acid secretion
and, 610
in duodenal ulcer results, 635
in peptic ulcer 638 641
in stress ulcer 636
technical considerations, 642
with antrectomy in peptic ulcer
643
with gastrojejunostomy in peptic
ulcer 642
with pyloroplasty in peptic ulcer
642
- Vagus nerve, in gastric acid secre-
tion, 610
sensory fibers, disorders, 1449
- Vakaria Cordus, 11
- Valisava, aneurysms, congeni-
tal, 462
surgical repair 459
- Valve aortic anatomy 454
- Valvular pulmonary stenosis, 437
- Valvulus, aortic, rheumatic, 455
- Valvuloplasty in mitral stenosis, low
sodium syndrome following, 133
- Vancomycin, toxic reactions, 56
- Vasishing lung phenomenon, 409
- Varices, complications, 1331
diagnosis, 1330
esophageal, 601
bleeding, balloon tamponade in,
793
in portal hypertension, 79
etiology 1329
in chronic venous insufficiency
treatment, 1341
incidence, 1329
intracranial, 1435
pathology 1330
rupture 1331
secondary 1338
spider web, 1330
symptoms, 1330
treatment, 1332
- Variocele, 907
vs. femoral hernia, 539
vs. indirect inguinal hernia, 530
- Varioloides. See *Varicella*.
- Vas deferens, anomalies, 906
infection, 907
tumors, 907
- Vascular disease degenerative, rectus
sheath hematoma and, 481
occlusive 1464
of foot, 1 95
peripheral, diagnostic tech-
niques, 1322
physical medicine and rehabil-
itation in, 1322
objectives, 1322
procedures, 1322
treatment, release of neuro-
genic vasoconstriction in,
1464
techniques, 1323
vasospasm in, 1463
- Vascular gangrene amputations for
1179
- Vascular injuries of neck, 308
- Vascular lesions of brain, 1433
- Vascular occlusion, intestinal ob-
struction in, 754
- Vascular rings, 435
symptoms, 436
treatment, 437
- Vascular system, 1327-1391
blood, in cold injury 200
- Vasectomy in chronic epididymitis,
904
- Vasitis, 907
- Vasoconstriction, neurogenic re-
lease, in peripheral vascular dis-
ease therapy 1464
- Vasodilator material, and vaso-
constrictor material, balance be-
tween, effect on circulation, 90
elaboration by liver 763
- Vasodilators in Meniere's disease
250
- Vasodilator material and vaso-
constrictor material, balance between,
effect on circulation, 90
- Vasopressor drugs in shock treat-
ment, 94
- Vasospasm in peripheral vascular
disease, 1463
- Vasospastic disorders, 1349
symptoms, 1344
- Vasostomostomy in male infertility
918
- Vater ampulla of, carcinoma of, 786,
811
- VDM elaboration by liver 763
- VDM VEM and, balance between,
effect on circulation, 90
- Vena(s) 1327-1344
anatomy 1327
bronchial, anatomy 400
coronary great, ligation in coro-
nary artery disease, 461
draining anorectum, 716
femoral, 1328
for venipuncture, choice of, 67
hepatic, thrombosis, in portal hy-
pertension, 791
vs. intrahepatic block, 791
hypogastric, 1328
iliac, 1328
of abdominal wall, 476
of extremities, 1328
physiology 1327
popliteal, 1328
pulmonary anatomy 400
saphenous, 1328
tibial, 1328
varicose. See *Varices*.
- Vespeau, 16
- VEM and VDM balance between,
effect on circulation, 90
- Venipuncture 67
technique 68
- Venous complications, postoperative
early ambulation and, 120
- Venous filling time in vasospastic dis-
orders, 1345
- Venous insufficiency chronic, lym-
phangiography in, 1390
prognosis, 1337
treatment, 1341
- Venous occlusion, massive, treat-
ment, 1340
- Venous sinus, lateral, thrombosis, vs.
intracranial abscess, 1432
- Ventilation, function, 400
- Ventricle, aneurysm, 462
surgical correction, 462
septal defects, 445
diagnostic features, 445
surgical repair 445
types, 445
third, tumors, 1425
- Ventricular fibrillation, correction of
109
in cardiopulmonary by-pass, 429
management, 468
- Ventriculography in craniocerebral
injuries, 1414
in intracranial tumors, 1428
- Verruca vulgaris, 1230
- Vertebrae cervical, dislocations,
1051
fractures, 1051
lumbar transverse processes, frac-
tures, 1054
- Vertigo, surreal, in Meniere's disease
49
in head injuries, 1400
in Meniere's disease, 1449
- Vetulus, 4 15
- Vesicles, seminal. See *Seminal es-
icles*.
- Vesiculectomy seminal, in chronic
seminal vesiculitis, 916
- Vesiculitis, seminal, 916
- Vesicle, anomalous, in mediastinum,
esophageal constriction by 574
cardiac, great, transposition, sur-
gical correction, 446
symptoms, 446
- Vesiphatic, anatomy 1364
- Ophthalmocentric, anomalies, 479
- umbilical, aberrations, 479
- urachal, anomalies, 479
- Vincent-Plant angina, 34
- da Vinci, Leonardo, 4 15
- Vincula longa et brevia, 1191
- Vinethene, 163
- Vinyl ether 163
- Violet, gentian, in thrush, 928
- Virchow's nodes in gastric carci-
noma, 671
- Varicella. See *Adenoviral syn-
drome*.
- Virus, infection, 46
of lymphogranuloma venereum in
rectal stricture 725
- Viscera, abdominal, lymph vessels,
1368
nonpenetrating wounds, 508
penetrating wounds, 507
deep pain conduction from, 1471
exposed, during operation, protec-
tion of, 106

- Tumor(s)** of parotid gland, benign, removal, 267-287
malignant, 288-289
of penis, 895
of peripheral nerves, 1458
of peritoneum, 512
of pharynx, 235
of pituitary, 1425
of pleura, 396
of rectum, 706. *See also* Colon tumors
of renal pelvis. *See* *Kalner pelvis tumors*
of salivary glands, benign, 286
malignant, 288
of scalp, benign, 214
malignant, 215
of scrotum, 897
of sellar region, 1425
of seminal vesicles, 916
of skeletal muscle, 1104
of skull, benign, 216
malignant, 216
metastatic, 217
secondary, 217
of small intestine. *See* *Intestine small, tumors*
of spermatic cord, 907
of spinal cord. *See* *Spinal cord tumors*
of spleen, 850
of stomach. *See* *Stomach, tumors*
of testis. *See* *Testis, tumors*
of third ventricle, 1425
of tongue, benign, 270
of trachea, 314
benign, 314
of ureter. *See* *Ureter tumors*
of urethra. *See* *Urethra, tumors*
of urinary tract, 877
of vas deferens, 907
osteoma, 1156
Pancoast's, 397
puffy, 1413
retroperitoneal, 416
submaxillary malignant, 288-289
thecal cell, 968
turbin, 214
vascular of hand, 1241
Wilm's, 880
- Tumor albus**, 1139
- Tumores vaginalis testis**, 905
anatomy, 905
anomalies, 905
physiology, 905
trauma to, 905
- Tunnel**, carpal, median nerve compression in, 1458
- Turban** tumors, 214
- Turner's syndrome**, 919
- Tympanoplasty** in chronic otitis media, 46
- Typhoid fever**, 687
- Ulcer**, Curling, 635
in burns, 186
duodenal. *See* *Duodenum, ulcer*
esophageal. *See* *Esophagus, ulcer*
gastric. *See* *Stomach, ulcer*
gastroduodenal, incidence, 606
gastrojejunal. *See* *Gastrojejunal ulcer*
of mouth, biopsy, 271
peptic. *See* *Peptic ulcer*
- Ulcer** recurrent, 653
incidence, 660
rodent, 267
stomal. *See* *Stomach ulcer*
stress, 635
acute, resection in, 636
vagotomy in, 636
varicose, 1331
- Ulceration**, chronic following thrombophlebitis, 1338
cortical, 1413
in chronic venous insufficiency treatment, 1341
- Ulna**, absence, congenital, 1249
fractures, 1016
comminuted, 1019
in children, 1017
oblique, 1019
transverse, in adults, 1019
in children, 1018
shaft, fractures, 1022
with radial head dislocation, 1022
- Ulnar nerve**, injuries, 1454
median nerve and combined injuries, 1455
palsy, tardy, 1457
- Ultrasound therapy**, 1313
in amputations, 1322
- Ultraviolet radiation**, 1312
- Umbilical hernia**. *See* *Hernia umbilical*
- Umbilical region**, abnormalities, 477
anatomy, 547
embryology, 472
Umbilical sinuses, 478
Umbilical vessels, aberrations, 479
Umbilicus, endometriosis, 479
infections, 479
pain around, in appendicitis, 693
Unconsciousness in head injuries, 1396
Unctus, herniation, in head injuries, 1402
Undecylenic acid in dermatophytosis, 1295
- United States**, surgery in, history, 18
- Unna's paste boot** in chronic venous insufficiency, 1341
- Urachal vessels**, anomalies, 479
- Urachus**, anomalies, 478
cysts, 478
pneum, acquired, 478
congenital, 478
sinuses, umbilical, 478
- Urea**, in intracranial operations, 1429
invert sugar and, in craniocerebral injuries, 1418
- Uremia**, effect on wound healing, 39
- Ureter(s)** anomalies, 869
double, 869-870
injuries, 871
in pelvic ring fractures, 1046
lymph vessels, 1369
obstruction, causes, 864
pain, in urinary tract disease, 855
retrocaval, 870
tumors, 880
chemical signs, 880
pathologic changes, 880
treatment, 880
- Ureterocele**, 870
- Ureterolithotomy**, 876
- Ureteronephrectomy** in tumors of ureter and renal pelvis, 880
- Urethra**, 891
carcinoma, squamous cell, 878
treatment, 878
exploration, in urinary tract disease, 862
injuries in pelvic ring fractures, 1056
lymph vessels, 1369
male, carcinoma, 895
obstruction, causes, 864
resistance, in neurogenic bladder, 883
decrease, measures for, 887
tumors, 877
clinical course, 878
diagnosis, 878
pathologic changes, 878
treatment, 878
- Urethritis**, chronic, nonspecific, vs. chronic prostatitis, 910
gonorrheal, in female, 974
- Urethrocele**, symptoms, 952
- Urethrovaginal obstruction**, causes, 864
- Urgency urinary** in urinary tract disease, 856
- Urinolysis** in preoperative routine, 97
- Urinary bladder** exstrophy, 479
- Urinary frequency** in urinary tract disease, 856
- Urinary stasis**, calculus and, 873
- Urinary system**, 854-889
- Urinary tract**, care in spinal injuries, 1440
complications as cause of postoperative fever, 121
disease, diagnosis, presumptive, 854-861
confirmation, 862
diagnostic steps in, 854
history in diagnosis, 854
in hyperparathyroidism, 324
pain in, 855
physical examination in, 857
symptoms, general, 855
in burns, pathologic changes in, 187
infection, 866
acute vs. acute prostatitis, 910
lymph vessels, 1369
obstruction, 862
causes, 864
diagnosis, 865
treatment, 866
stones, vs. appendicitis, 694
tuberculosis, 869
tumors, 877
upper studies, 862
- Urination**, phases, 881
physiology, 881
reflex arc in, 881
- Urine**, cloudy in urinary tract disease, 857
examination in urinary tract disease, 858
in peritoneal cavity, 499
osmolality postoperative, 126
output, postoperative diuresis in, 126
residual, PSP test and, 860
- Urography** intravenous, in neurogenic bladder, 883
in urinary tract disease, 861
- Ureteric tube**, lymph vessels, 1369
- Uterus**, adenomyosis, 963
body carcinoma. *See* *Carcinoma of corpus uteri*

- Wound(s) healing, keloid formation and 79
 lag phase 3, 27
 lymphatic obstruction and, 35
 metabolic diseases and, 39
 necrotic tissue and, 3
 nutrition and, 37
 of special tissues, 39
 pill of tissues in, 4
 phases, 23
 physiology 22-41
 portal clirrhosis and 39
 postoperative 111
 primary 31
 rest and, 39
 scab formation in, 30
 scar contraction phase 23
 secondary 3
 sensation and 31
 suppurative versus healing by first intention, history 2
 swelling in, 24
 temperature and, 39
 tissue pill in, 4
 uremia and, 39
 vitamins and, 38
 hematomas, 112
 incised, of neck, 307
- Wound(s) infection in open fractures, prophylaxis and treatment, 986
 incisional hernia and, 554
 postoperative 111
 prevention, history 2
 open, 80
 treatment 8
 types, 80
 penetrating, 81
 repair history 2
 technique history 6
 scalp, 214
 seroma, 112
 severe cure 67
 shock 87
 simple care of 65
 technique, faulty incisional hernia and, 554
 traumatic, antibiotic therapy in, 59
 treatment in open fractures, 991
 types, 22
- Wrisberg, accessory lobe of 409
 nerve of 1448
- Wrist, dislocation, perilunar 1008
 fracture first aid, 982
 joint, fractures, 1003
 sprain, 1108
- Wrist sprain, treatment, 1004
 tuberculosis, 1146
- XANTHOMAS, giant cell, of synovium, 1171
 of hand, 1240
 of tendon sheath vs. tuberculous synovitis, 1201
- X ray See also *Radiation Radium and Roentgenotherapy*
 burns of vulva, 973
 radiation as factor in carcinoma of hand, 1232
 studies in preoperative routine, 97
- Z-PLASTY in constrictions of hand, digits or arm, 1248
- Zona fasciculata, 871
 hyperplasia, 830
- Zona glomerulosa, 871
 hyperplasia, 830
- Zona reticularis, 821
 hyperplasia, 830
- Zygoma, fracture-dislocation, 278
 reduction, 279
- Zygomatic arch fractures, 277

- Viscera, pelvic lymph vessels, 1368
 pain in, 929
 perforated, in pneumoperitoneum, 510
 sensory innervation, 1473
 Visceral space of neck, infections, 306
 injuries, 308
 Visceromotor activity disorders, sympathectomy in, 1467
 Viscus. See *Viscera*.
 Vision, blurring, in intracranial tumors, 1421-1423
 disturbances in head injuries, 1401
 Visual acuity falling, in intracranial tumors, 1421
 Vital capacity timed, 401
 Vitamin(s) deficiencies, effect on mouth, 256
 following gastrectomy 626
 in burns, 191
 metabolism, liver and, 762
 therapy preoperative, 103
 wound healing and, 38
 Vitamin A, deficiency effect on mouth, 256
 effect on wound healing, 39
 Vitamin B, complex, effect on wound healing, 39
 deficiency effect on mouth, 256
 small intestine and, 688
 Vitamin B₁₂, in Meniere's disease, 250
 preoperative administration, 103
 Vitamin C deficiency effect on mouth, 257
 management, preoperative, 104
 effect on wound healing, 38
 Vitamin D effect on wound healing, 39
 Vitamin K, deficiency management, preoperative, 104
 effect on wound healing, 39
 in obstructive suppurative operations, 782
 Vitelline cysts, 478
 Vitelline duct, 472
 anomalies, 477
 persistent, 583 See also *Driver inculum Macek's*.
 Volkmann's contracture, 1221
 in supracondylar humeral fractures, 1034
 treatment, 1222
 vs. Ledderhose's disease, 1293
 Volvulus, 701
 age incidence, 701
 in sigmoid, 741
 intestinal obstruction in, 757
 pathology, 701
 sex incidence, 701
 symptoms, 701
 treatment, 701
 Vomiting, in congenital hypertrophic pyloric stenosis, 575
 in head injuries, 1400
 in intestinal obstruction, 736, 739
 in intracranial tumors, 1421
 in peptic ulcer 613
 in peritonitis, 497
 in stoma ulcer 617
 Von Bergmann, 5
 Von Bruns, 5
 Von Ebersberg exclusion operation, 644
 Von Ebersberg gastrotomy
 639 640
 Von Ebersberg operation in gastric cancer 671
 Von Eschmarch, 9
 Von Graefe, 18
 Von Haberer gastroduodenostomy 639 640
 Von Langenbeck, Bernhard, 18
 Von Recklinghausen's abdominal wall neurofibromas, 482
 Von Recklinghausen's disease, 1459
 in hand, 1246
 Von Recklinghausen's osteitis fibrosa cystica generalisata, 324
 V-phlegmon, 1193
 Vulva, 972
 carcinoma, 975
 chancroidal infection, 974
 Durey infections, 974
 folliculitis, 972, 973
 furunculosis, 972, 973
 gonorrheal infections, 974
 herpes infection, 973
 hidradenitis, 972, 973
 intertrigo, 972
 kraurosis, 975
 leukoplakia, 975
 carcinoma of vulva and, relation, 976
 lymphatics, 1369
 staphylococcal infections, 972
 treatment, 973
 syphilitic infection, 974
 x ray burns, 973
 Vulvectomy in leukoplakia of vulva, 976
 radical, in carcinoma of vulva, 975
 Vulvitis, atrophic, chronic, 976
 tuberculous, 975
 Waldmeyer, 960
 Waldmeyer's ring, 232
 Walking, 1264
 Wangersteen sleeve resection in peptic ulcer 643 644
 Wangersteen tube in decompression of small intestine, 746
 Wangersteen tubular gastric resection, 643 644
 Ward, recovery 107
 Warren, John Collins, 11
 Warts, of hand, 1230
 plantar 1282
 subungual, of hand, 1231
 venereal, 694
 Water clearance postoperative, 126
 cold injury and, 198
 intolerance, postoperative, 124
 intoxication, 133
 loss in intestinal obstruction, 735
 Weather in carcinoma of hand, 1232
 Weber hearing test, 247
 Wedge resection, in congenital talipes equinovarus, 1 69
 in pes cavus, 1278
 Weight, bearing, in fractures, 995
 body in assessing hydration, 127
 in post traumatic metabolism, 142
 change, following gastrectomy 626
 gain in late convalescence, 146
 loss, amount, importance in preoperative management, 99
 in stoma ulcer 617
 preoperative, in obesity 102
 Welch, William H., 19
 Wells, Horace 11
 Wens of scalp 214
 Werlhof's disease, 846, 847
 Wheel chair, 1311 1319
 Whipple operation, in pancreatic carcinoma, 813
 operative mortality rate, 814
 Whitlow melanotic, of hand, 1236
 Whitman frame in Pott's disease, 1141
 Willis, circle of, aneurysms, 1433
 Wilms tumor 800
 Winslow foramen of, 466
 Wire fixation, in tibial spine fracture, 1073
 of patellar fractures, 1072
 Wire mesh repair of incisional hernia, 560
 Wire-pin fixation in jaw fractures, 775
 Wither operation in peptic ulcer 642
 "Worms, finger 1193
 Wound(s) See also *Injuries and Trauma*.
 accidental, cleaning, 83
 closure 84
 delayed primary 85
 anabolism, 144
 care, history 2
 catabolism, 141
 closed, 80
 treatment, 82
 closure in hand injuries, 1211
 complications, postoperative, avoidance, 111
 coostimulation, care, 112
 definition, 22
 dehiscence, 113
 disruption, 112
 causes, 113
 treatment, 114
 during late convalescence, 146
 edges, protection during operation, 11
 eversion, 112
 causes, 113
 shock in, 113
 treatment, 114
 excision, 83. See also *Debridement*.
 in hand injuries, 1209
 extensive, in convalescence, endocrine and metabolic features, 152
 healing, agammaglobulinemia and, 38
 bleeding in, excessive, 4
 blood flow in, increased, 24
 blood supply and, 37
 local, 35
 by first intention, 31
 by second intention, 26
 by third intention, 32
 classification, 31
 clotting mechanism in, 24
 contraction of scar phase, 3
 crusting in, 30
 definition, 23
 devitalized tissue and, 32
 diabetes and, 39
 edema and, 35
 factors influencing, general, 37
 fibroplasia stage, 23
 foreign bodies and, 34
 granulation tissue in, 25
 hemorrhage and, 37
 impingement, local factors in, 32
 infection and, 33
 initial phase, 27

- Wound(s) healing, keloid formation and 29
 lag phase 23-77
 lymphatic obstruction and, 35
 metabolic diseases and, 39
 necrotic tissue and, 3
 nutrition and, 37
 of special tissues, 39
 pH of tissues in, 4
 phases, 23
 physiology 22-41
 portal cirrhosis and, 39
 postoperative 111
 primary 31
 rest and, 39
 scab formation in, 30
 scar contraction phase 23
 secondary 32
 sensation and, 31
 suppurative, versus healing by
 first intention, history 2
 swelling in, 24
 temperature and, 39
 tissue pH in, 24
 uremia and, 39
 vitamins and 38
 hematoma, 11
 incised, of neck, 307
- Wound(s) infection in open frac-
 tures, prophylaxis and treat-
 ment 986
 incisional hernia and 554
 postoperative, 111
 prevention, history 2
 open, 80
 treatment, 8
 types 80
 penetrating, 81
 repair history 2
 technique history 6
 scalp 214
 seroma 112
 severe care 67
 shock 87
 simple care of 65
 technique faulty incisional hernia
 and, 554
 traumatic antibiotic therapy in, 59
 treatment in open fractures, 991
 types, 22
- Wrisberg, accessory lobe of, 409
 nerve of 1448
- Wrist, dislocation, perilunar 1008
 fracture, first aid, 982
 joint, fractures, 1003
 sprain, 1108
- Wrist sprain treatment, 1004
 tuberculosis, 1146
- XANTHIOMAS, giant cell of synovium,
 1171
 of hand 1240
 of tendon sheath vs. tuberculous
 synovitis, 1201
- X-ray See also *Radiation Radium*
 and *Röntgenotherapy*
 burns of vulva, 973
 radiation as factor in carcinoma of
 hand, 1232
 studies in preoperative routine, 97
- Z-PLASTY in constrictions of hand,
 digits or arm, 1248
- Zona fasciculata, 821
 hyperplasia, 830
- Zona glomerulosa, 821
 hyperplasia, 830
- Zona reticularis, 821
 hyperplasia, 830
- Zygoma, fracture-dislocation, 278
 reduction, 279
- Zygomatic arch fractures, 277